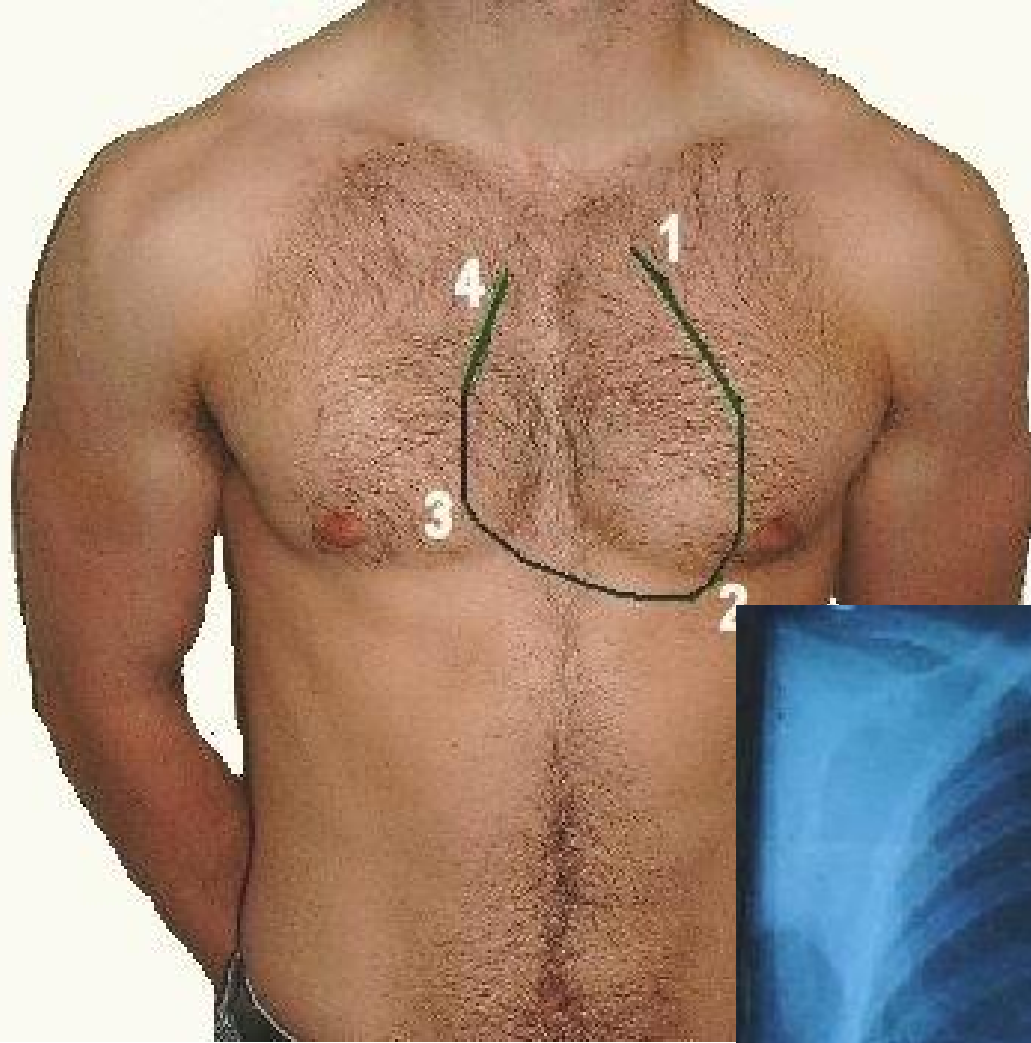
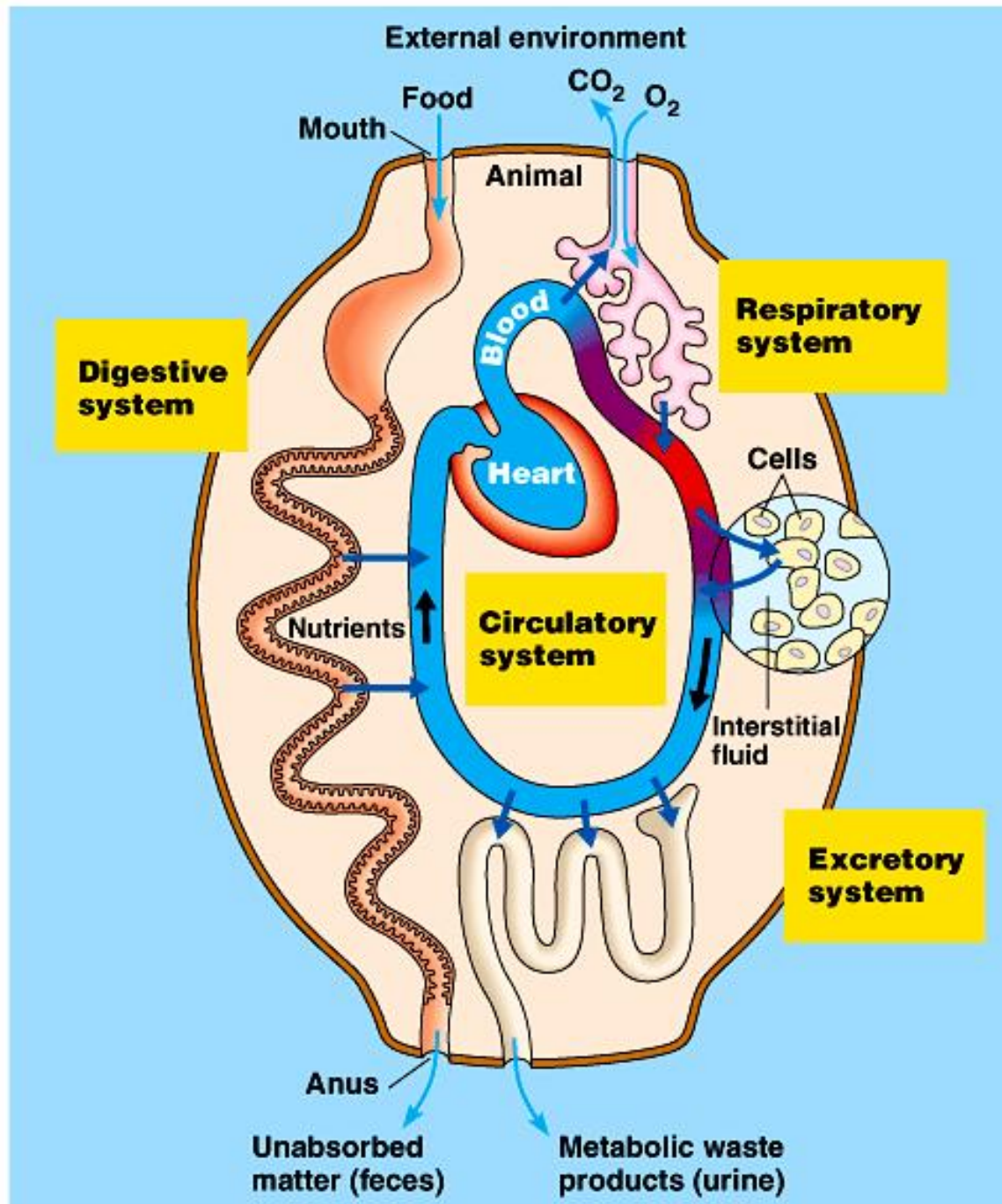


# Cardiovascular System







# The Heart: A Two Sided Pump

Handles two types of blood

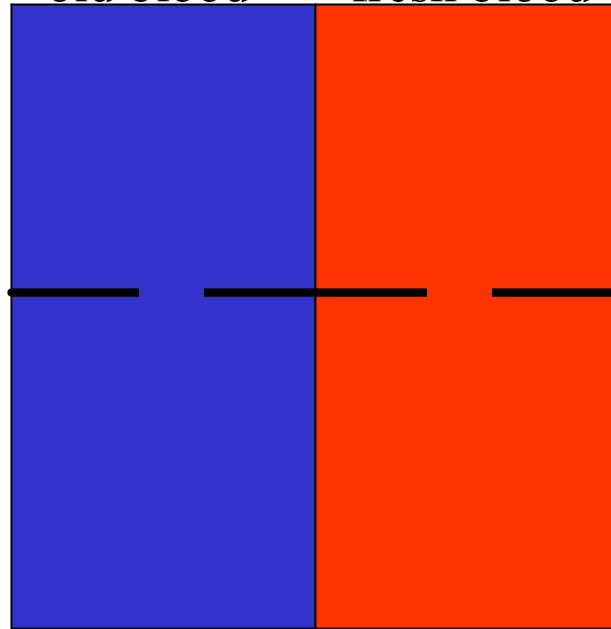
Deoxygenated- blue

Oxygenated- red

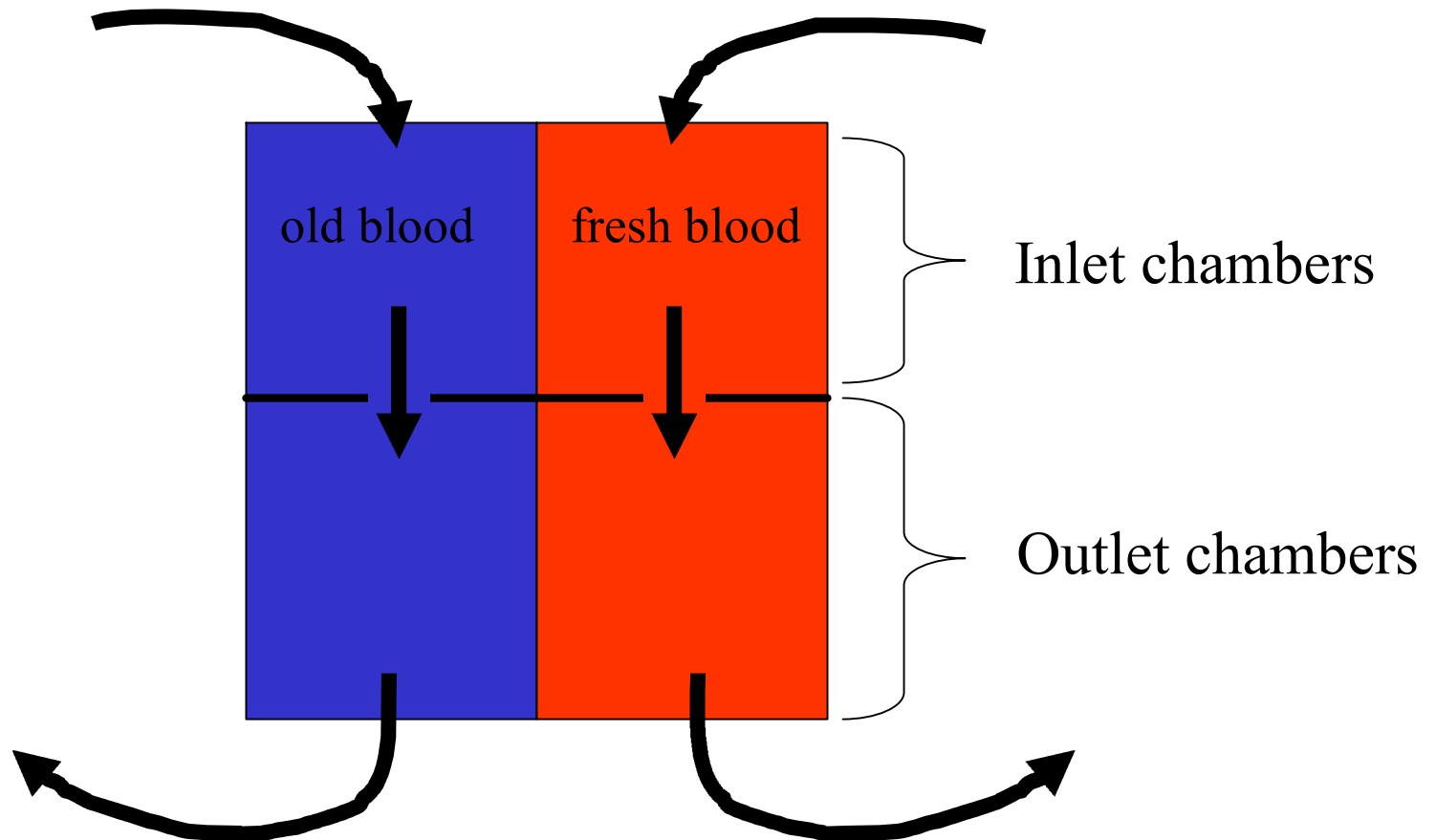
Right side    Left side

old blood

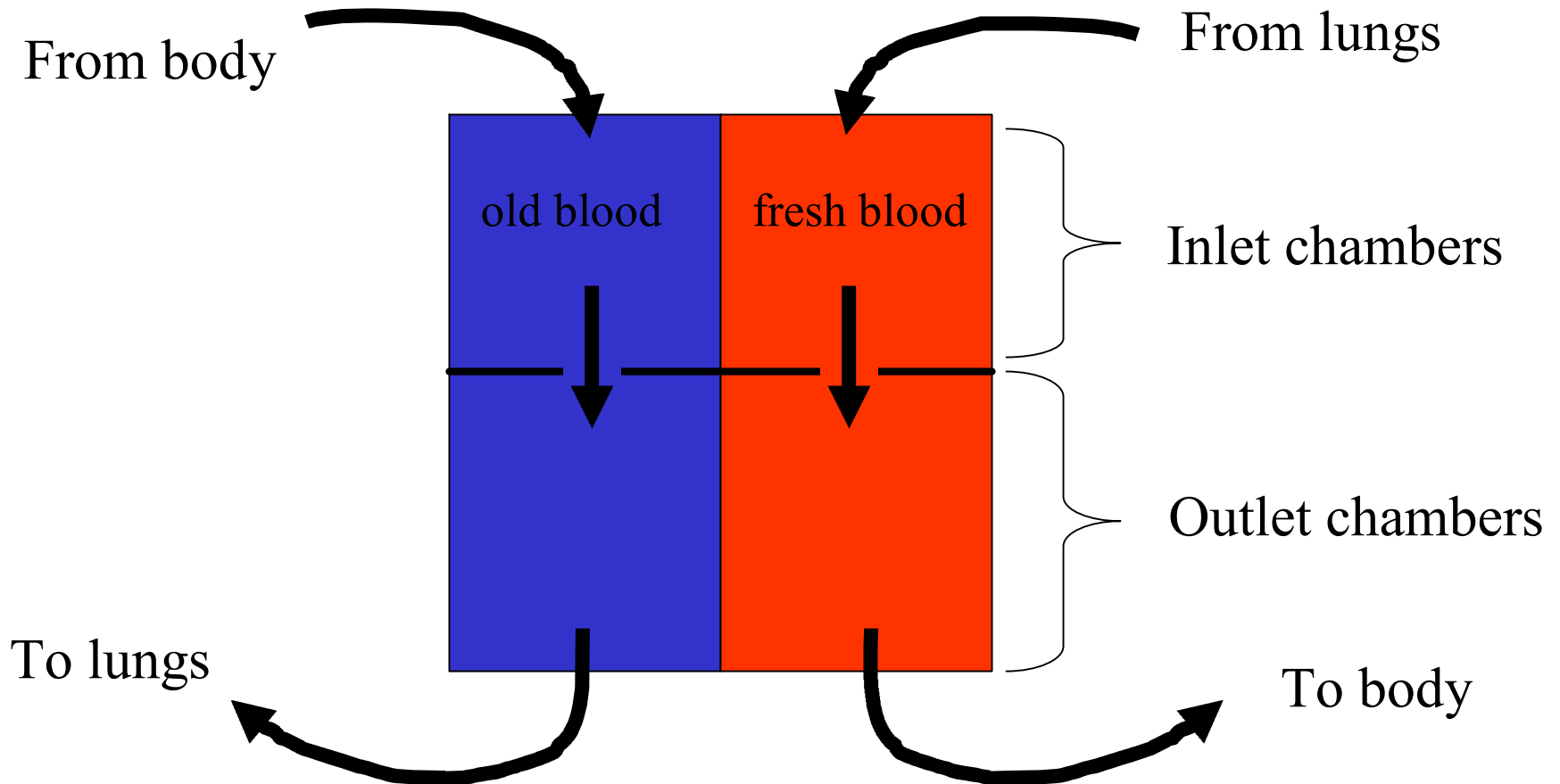
fresh blood



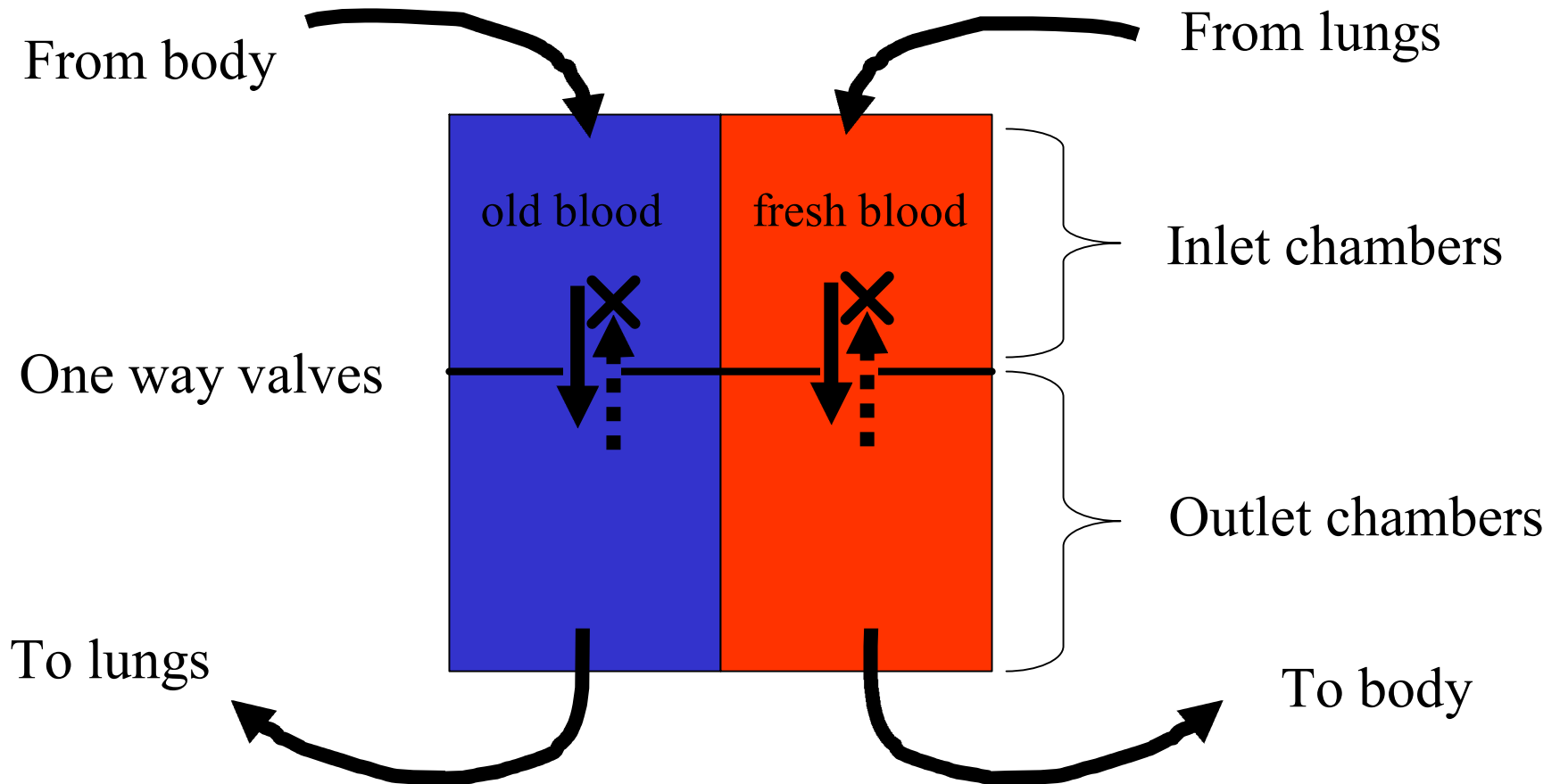
# The Heart: A Two Sided Pump



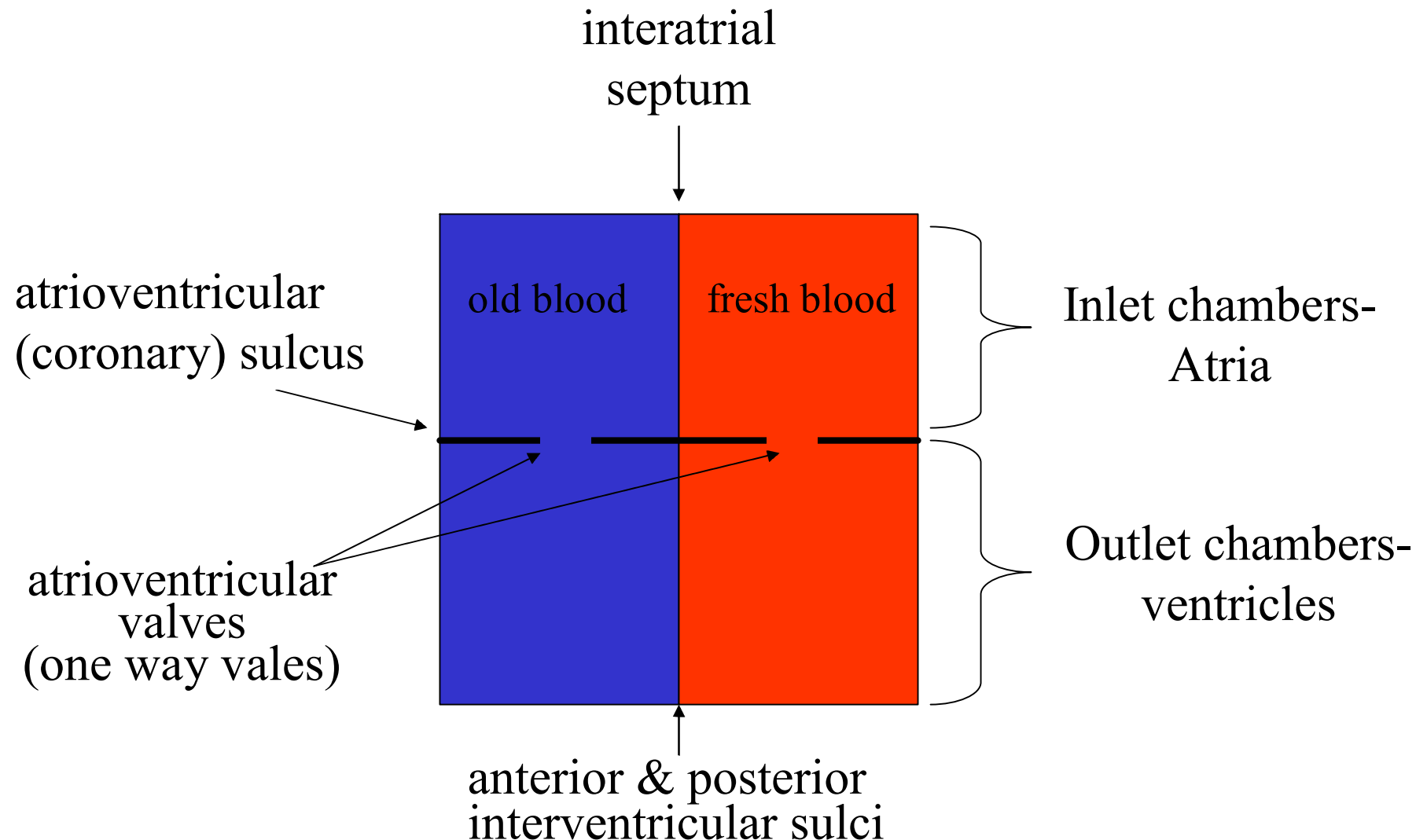
# The Heart: A Two Sided Pump



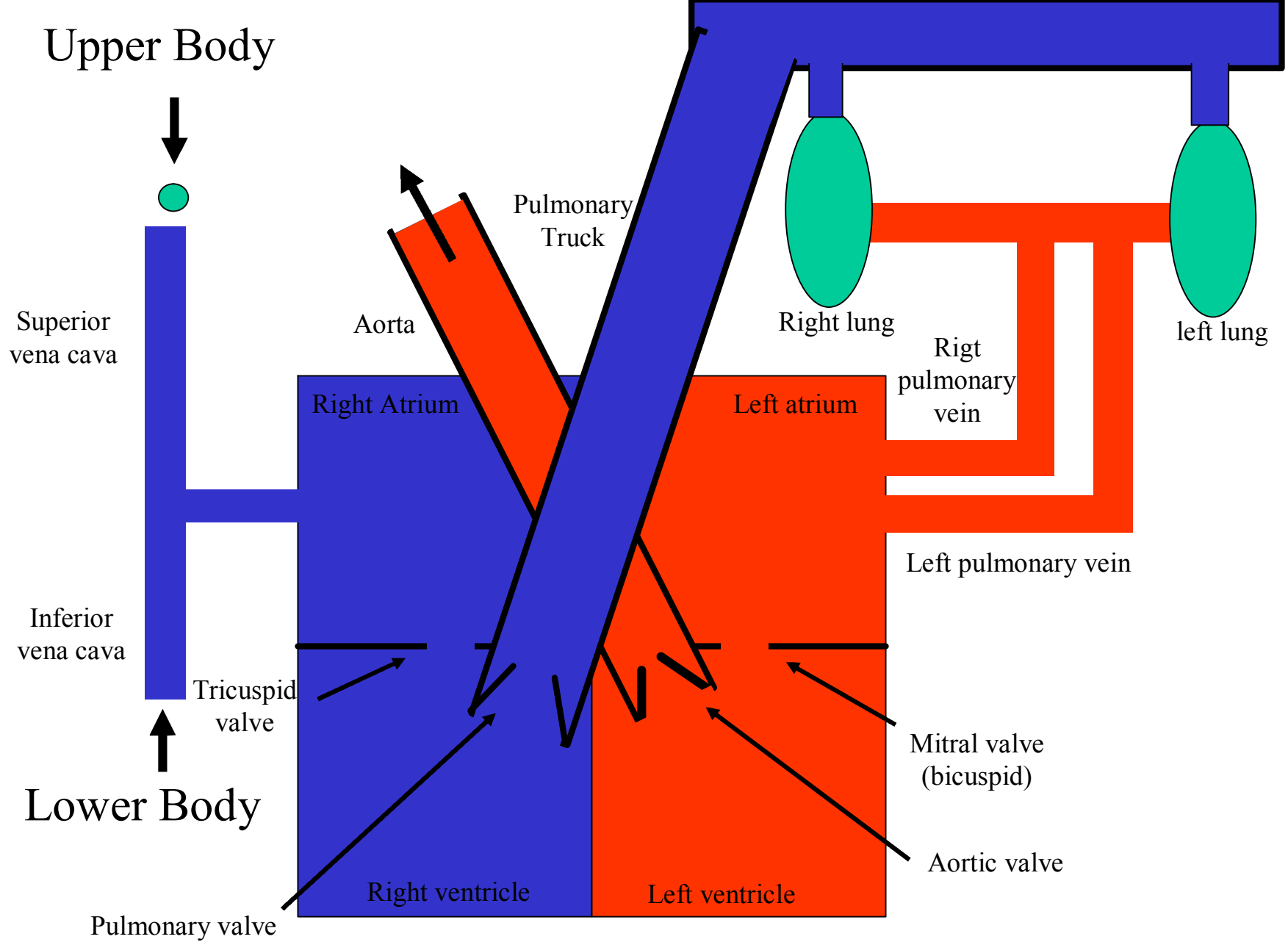
# The Heart: A Two Sided Pump

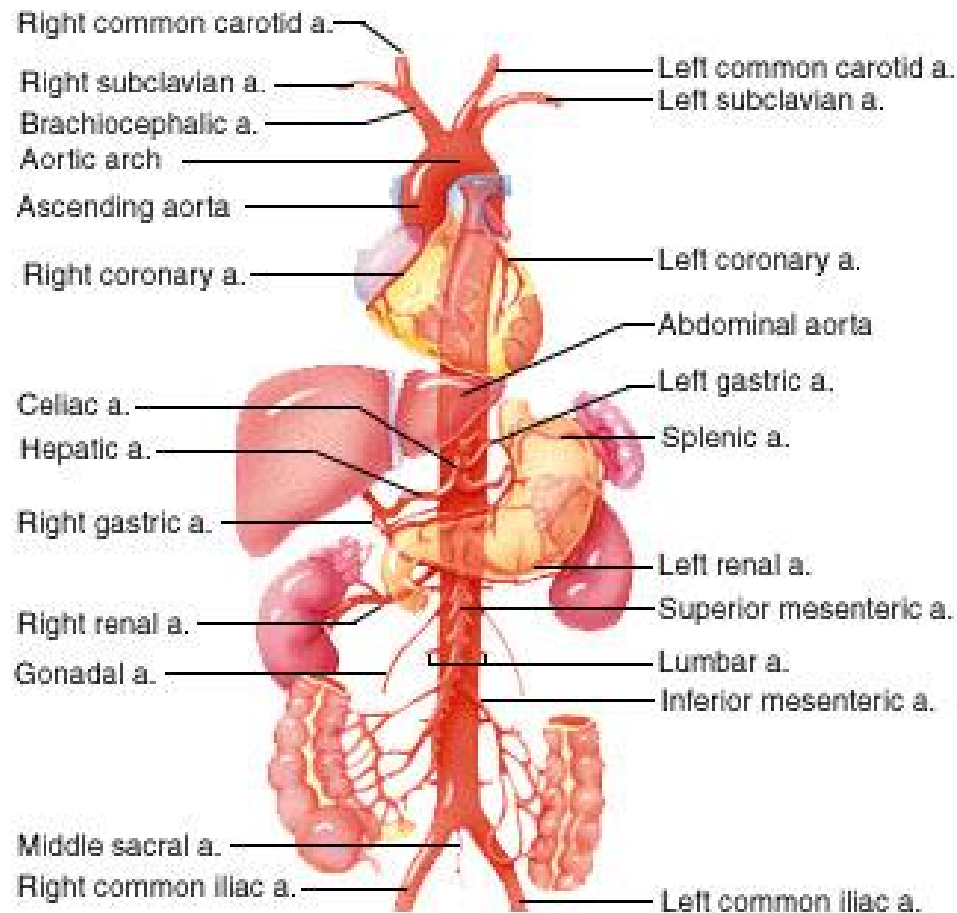
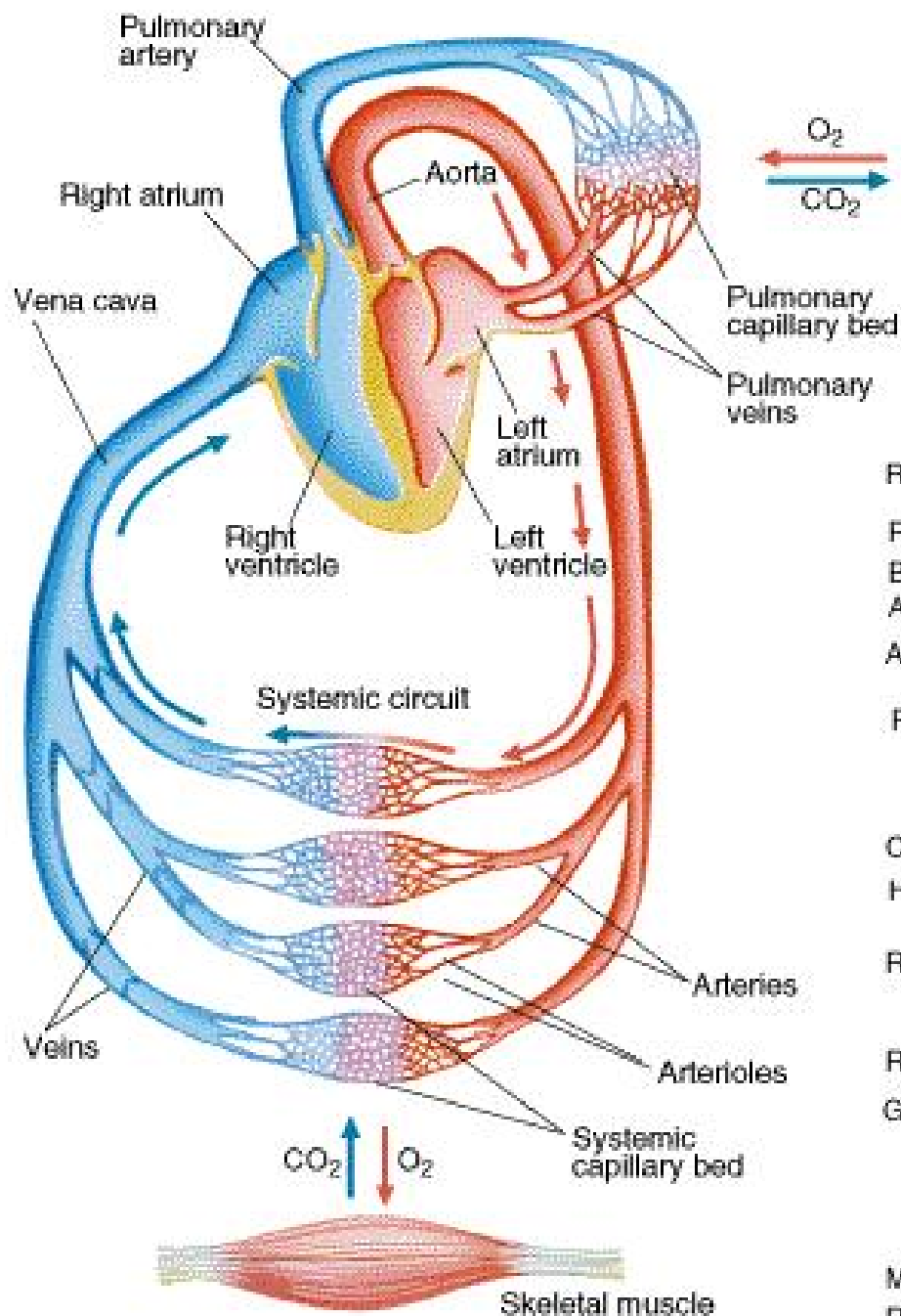


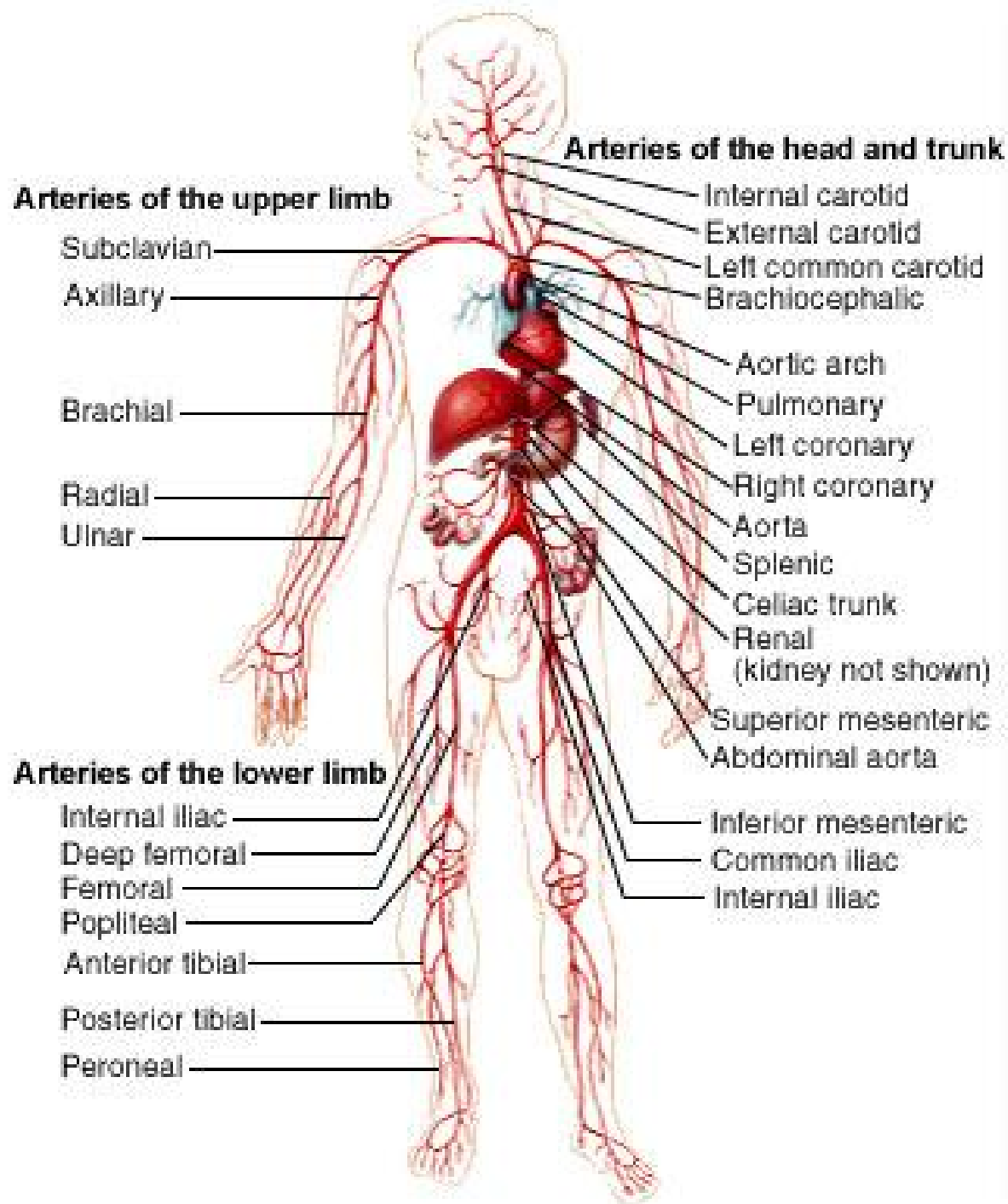
# The Heart: A Two Sided Pump

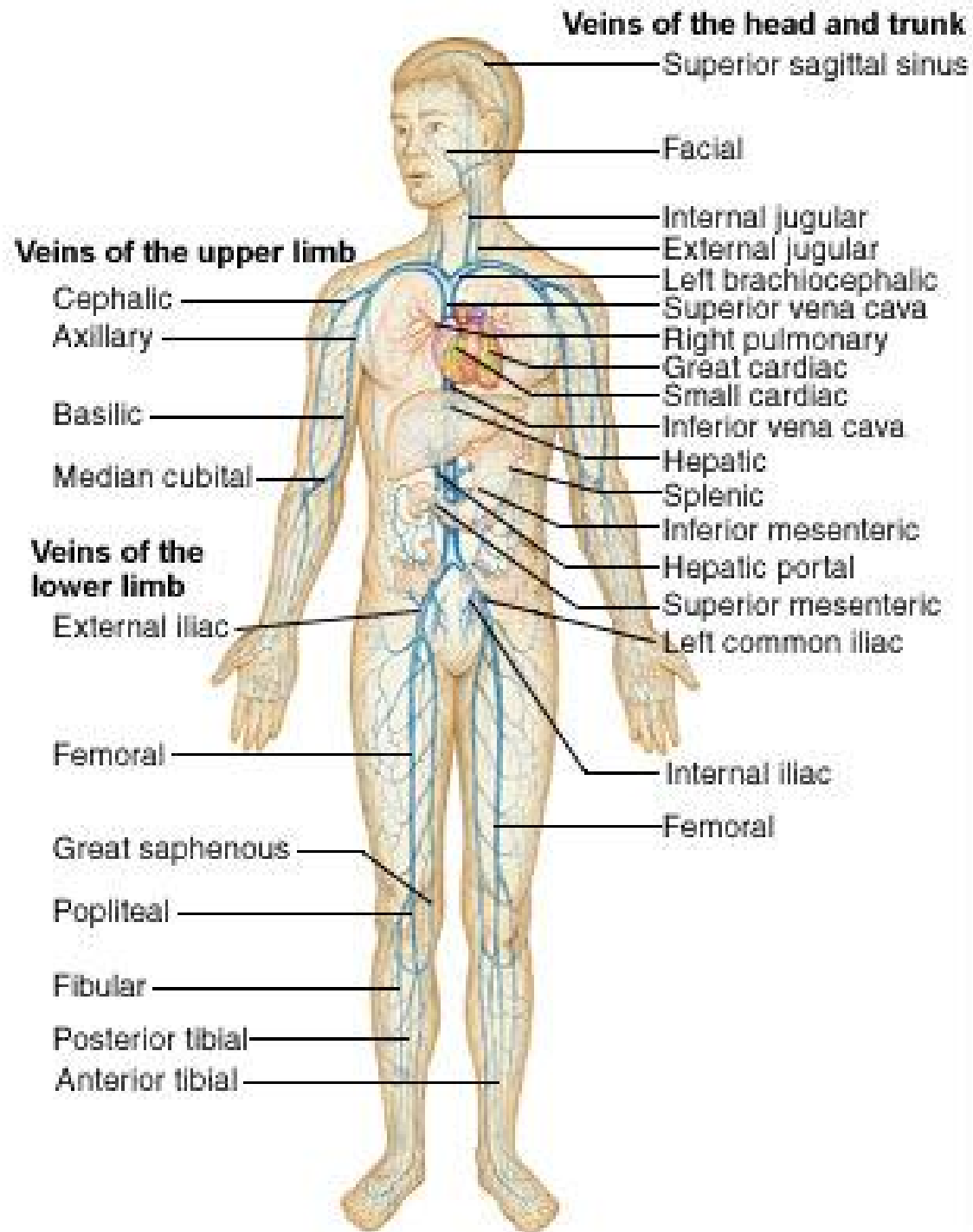


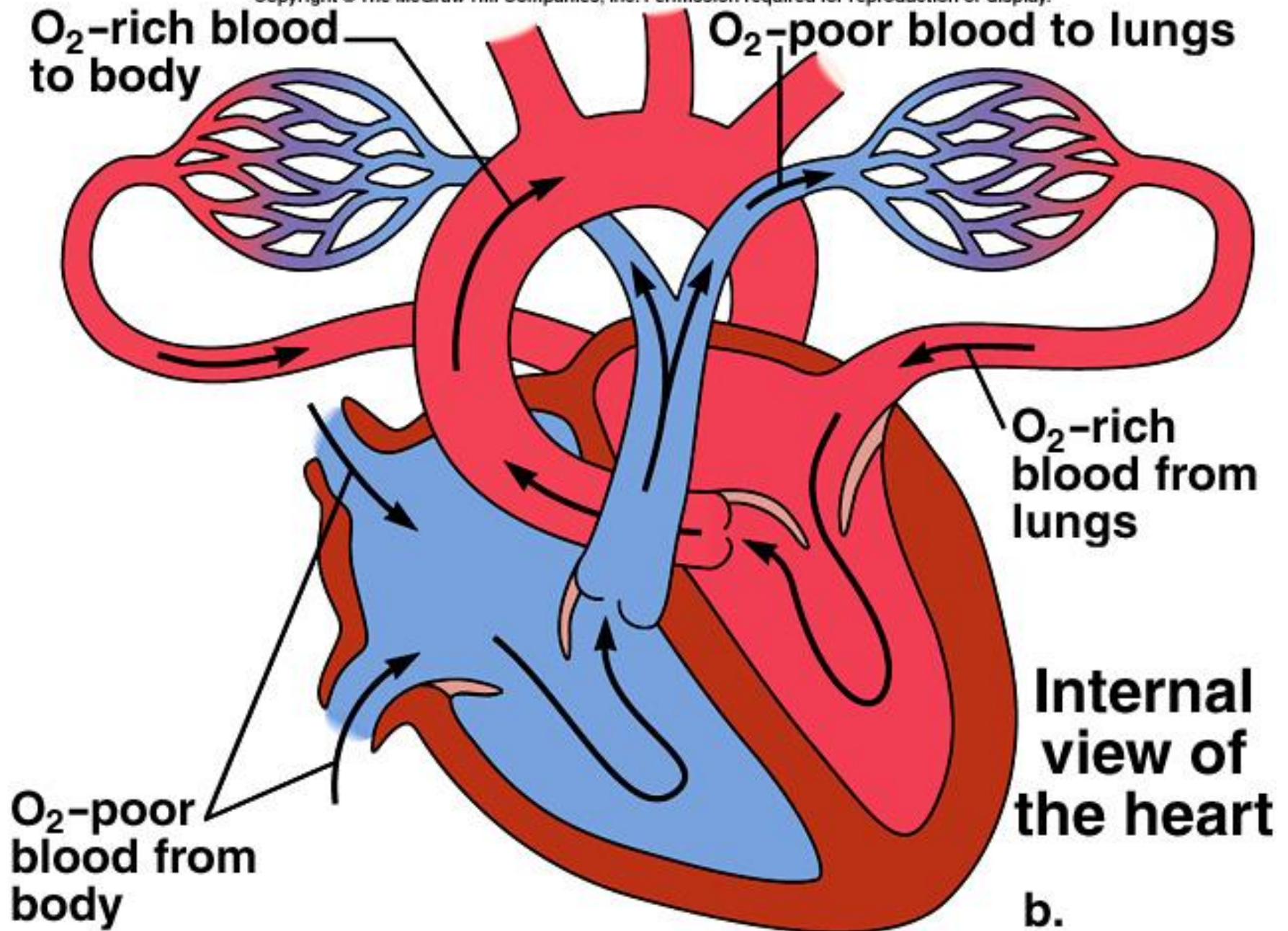












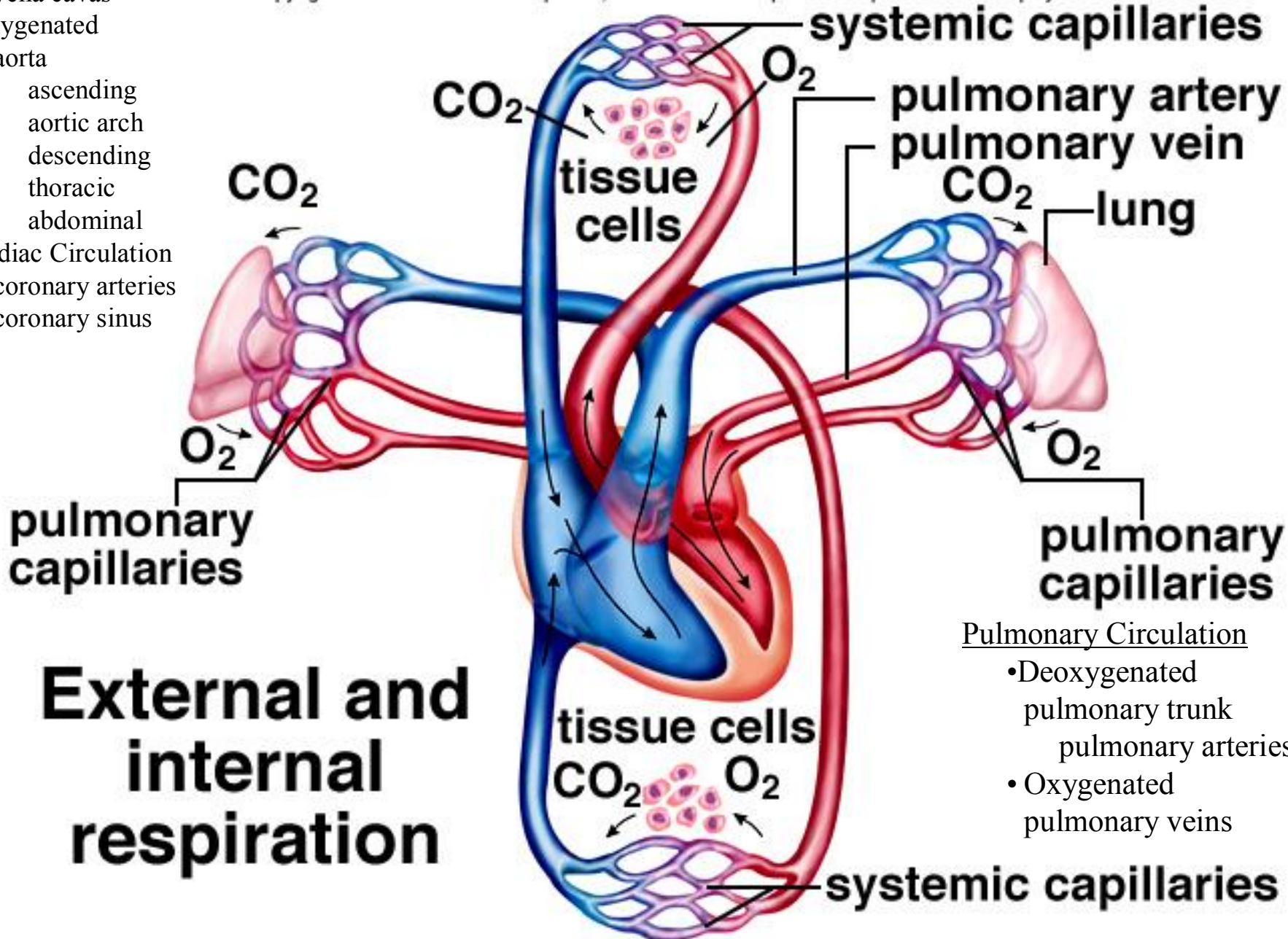


Systemic Circulation

- Deoxygenated  
vena cava
- Oxygenated  
aorta

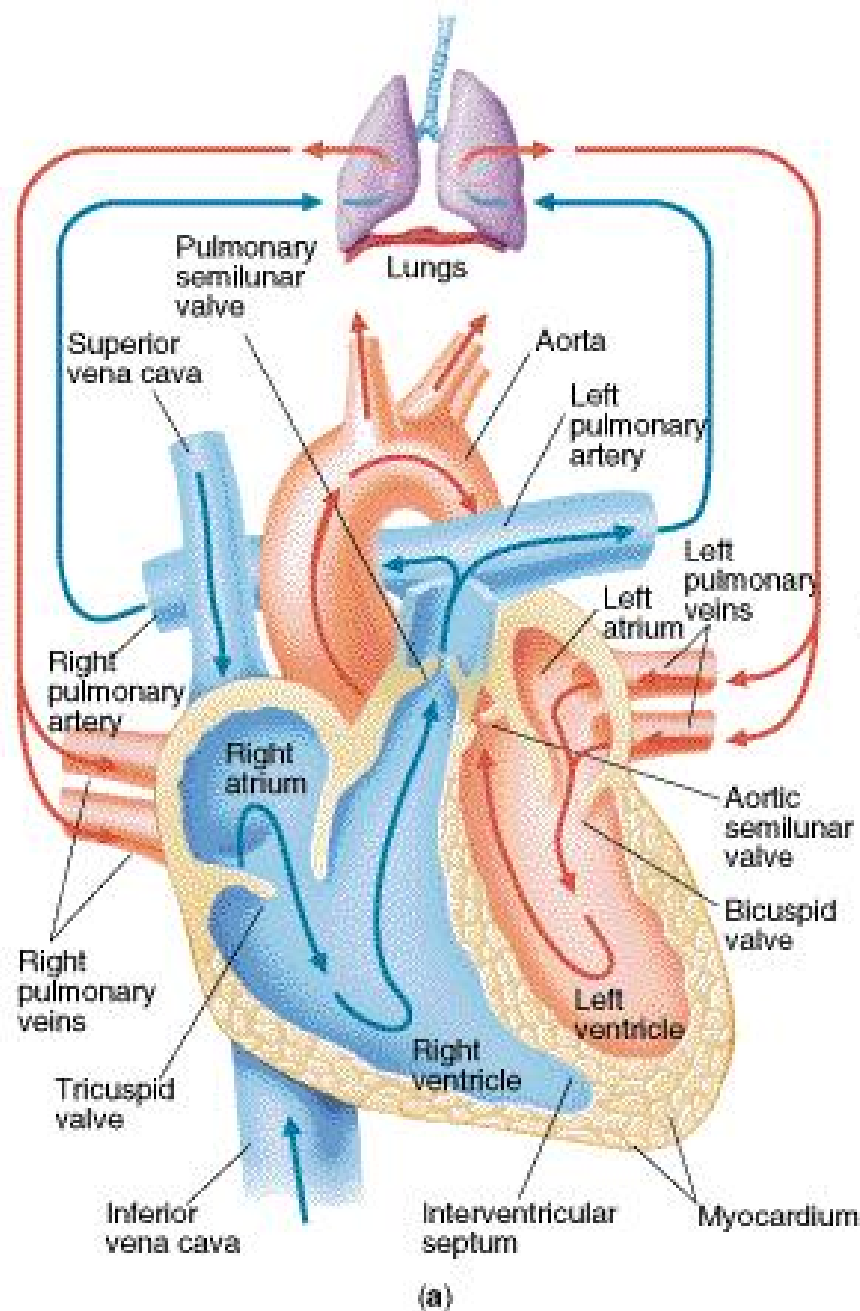
- ascending
  - aortic arch
  - descending
  - thoracic
  - abdominal
- Cardiac Circulation
- coronary arteries
  - coronary sinus

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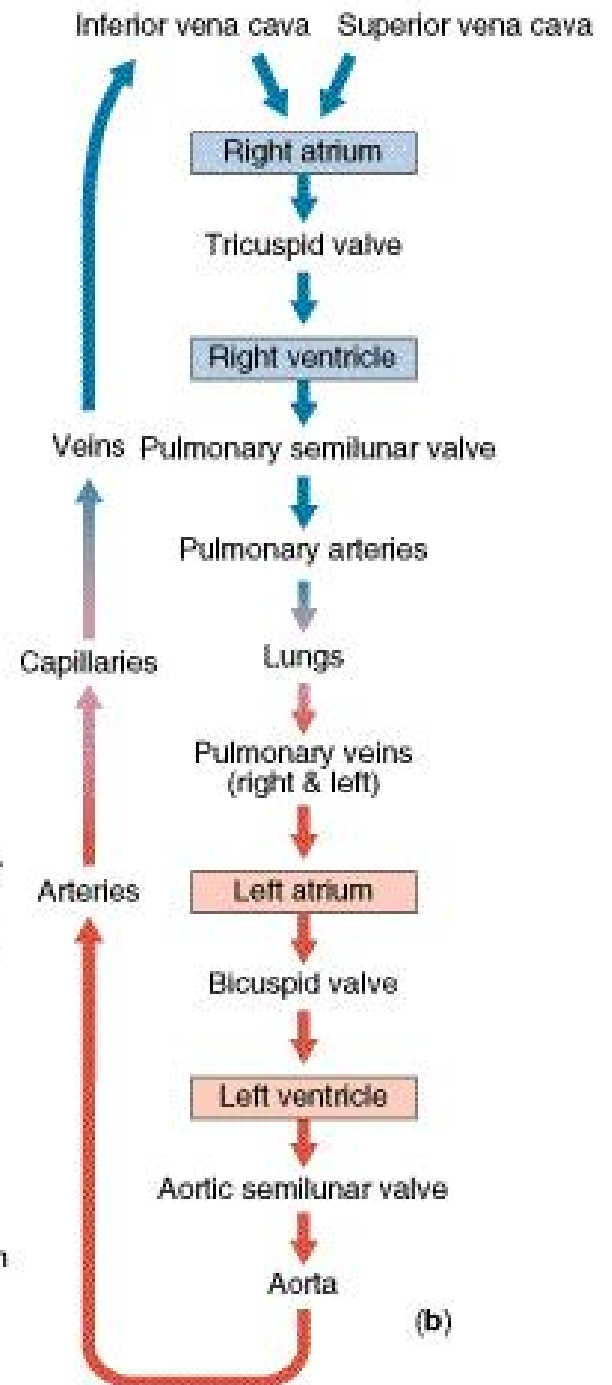


Pulmonary Circulation

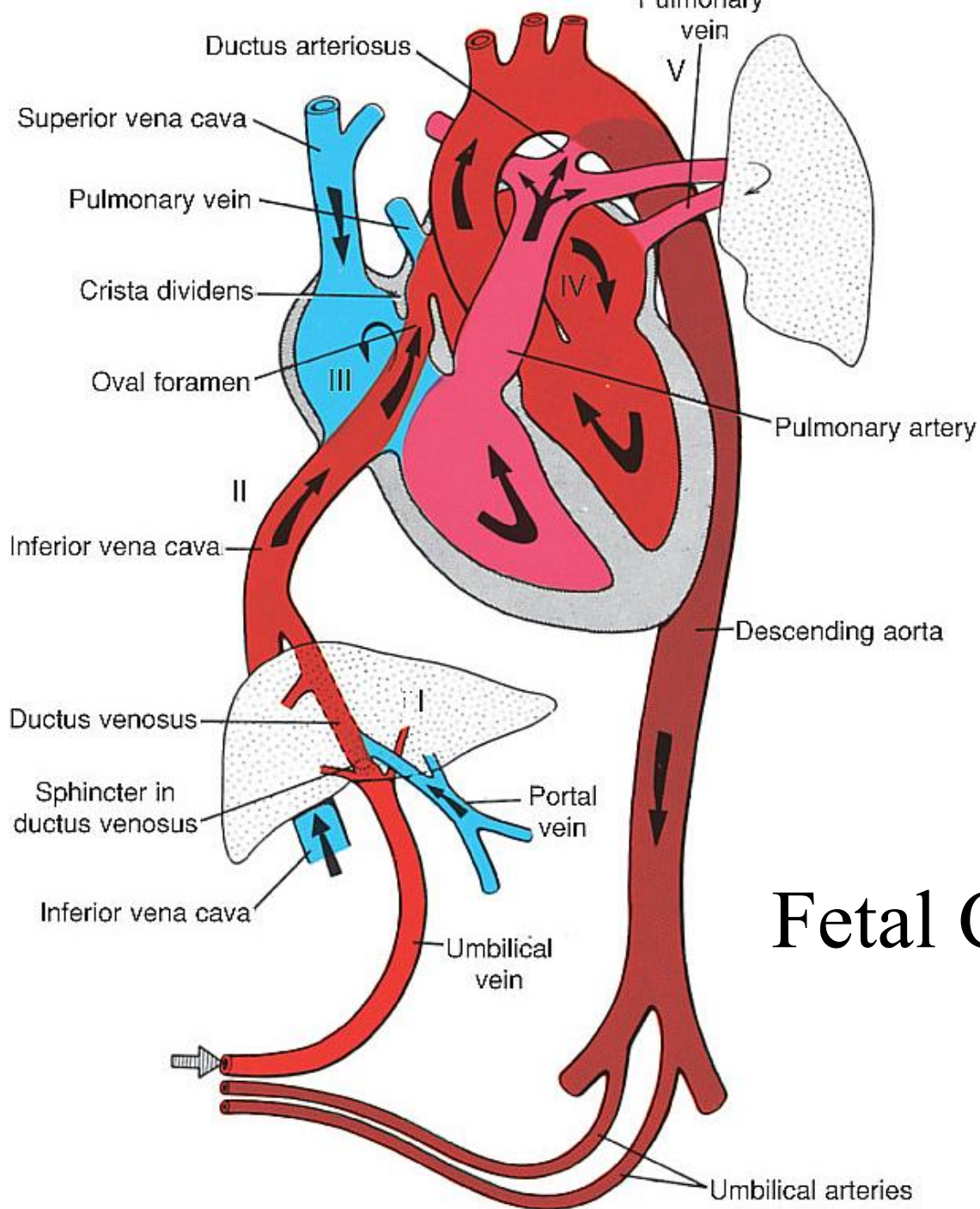
- Deoxygenated  
pulmonary trunk  
pulmonary arteries
- Oxygenated  
pulmonary veins



(a)



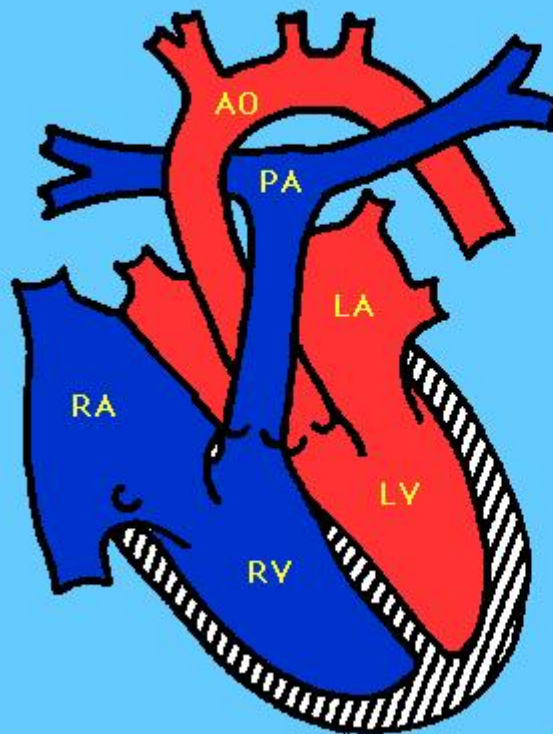
(b)



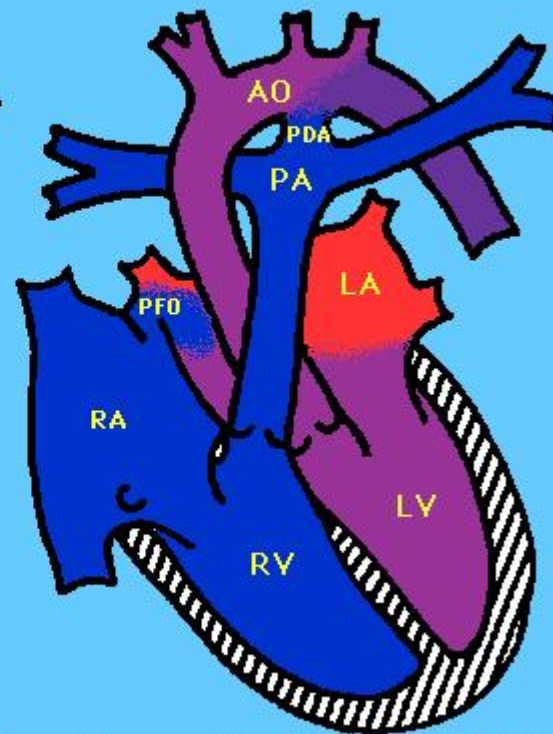
# Fetal Circulation



### Persistence of the Fetal Circulation

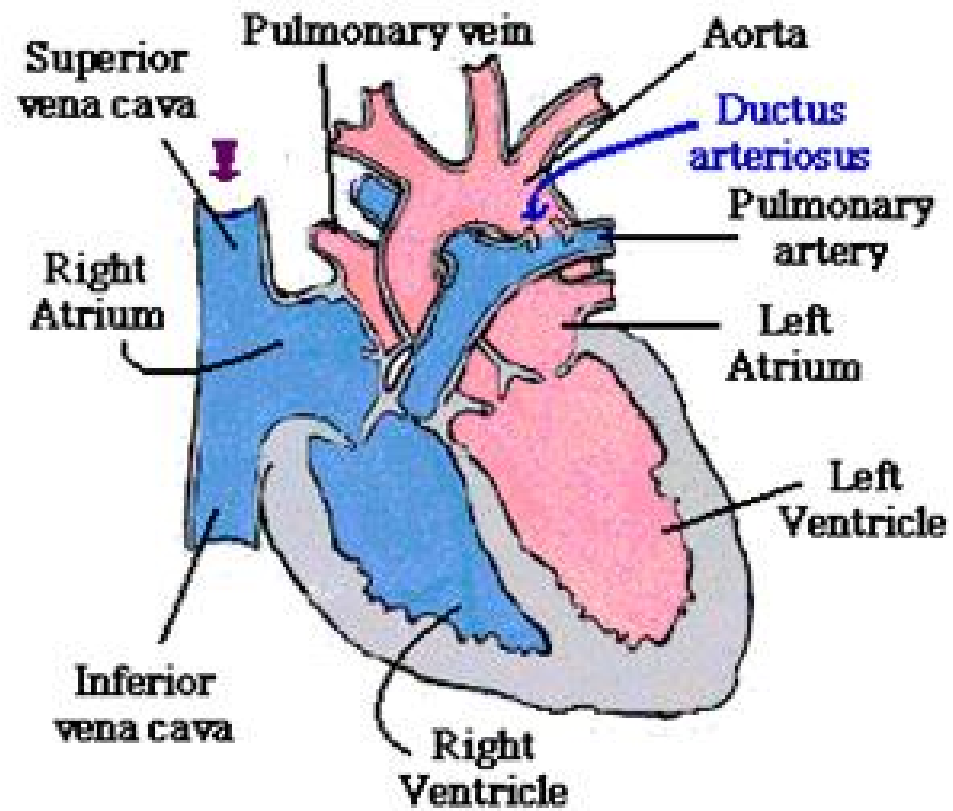


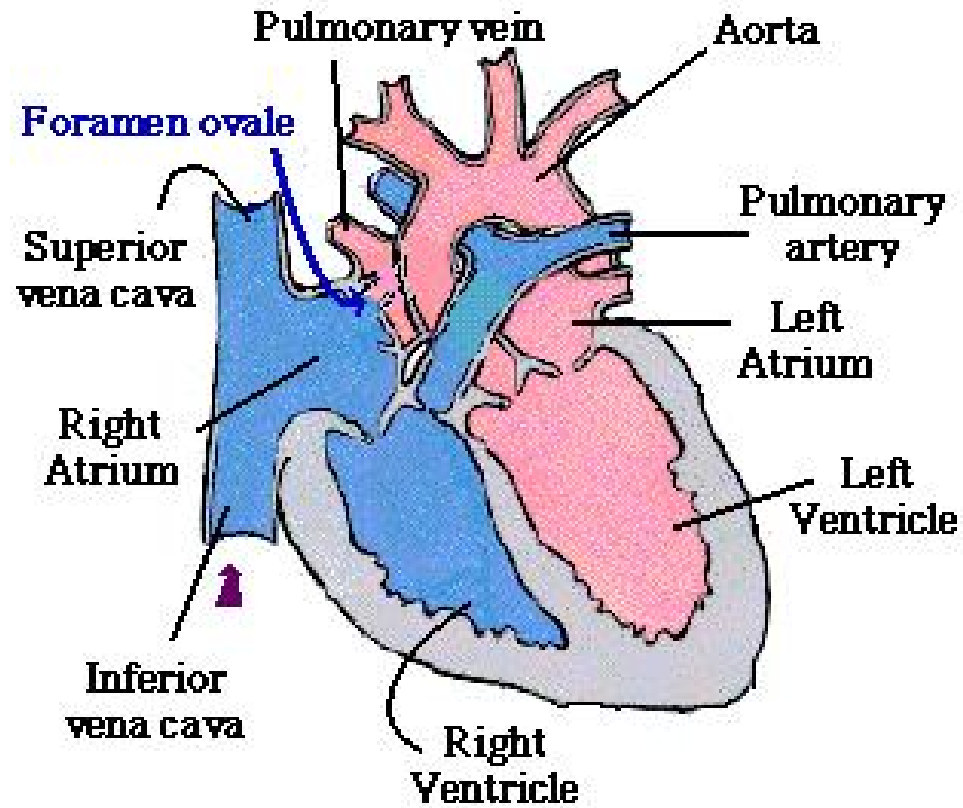
Normal



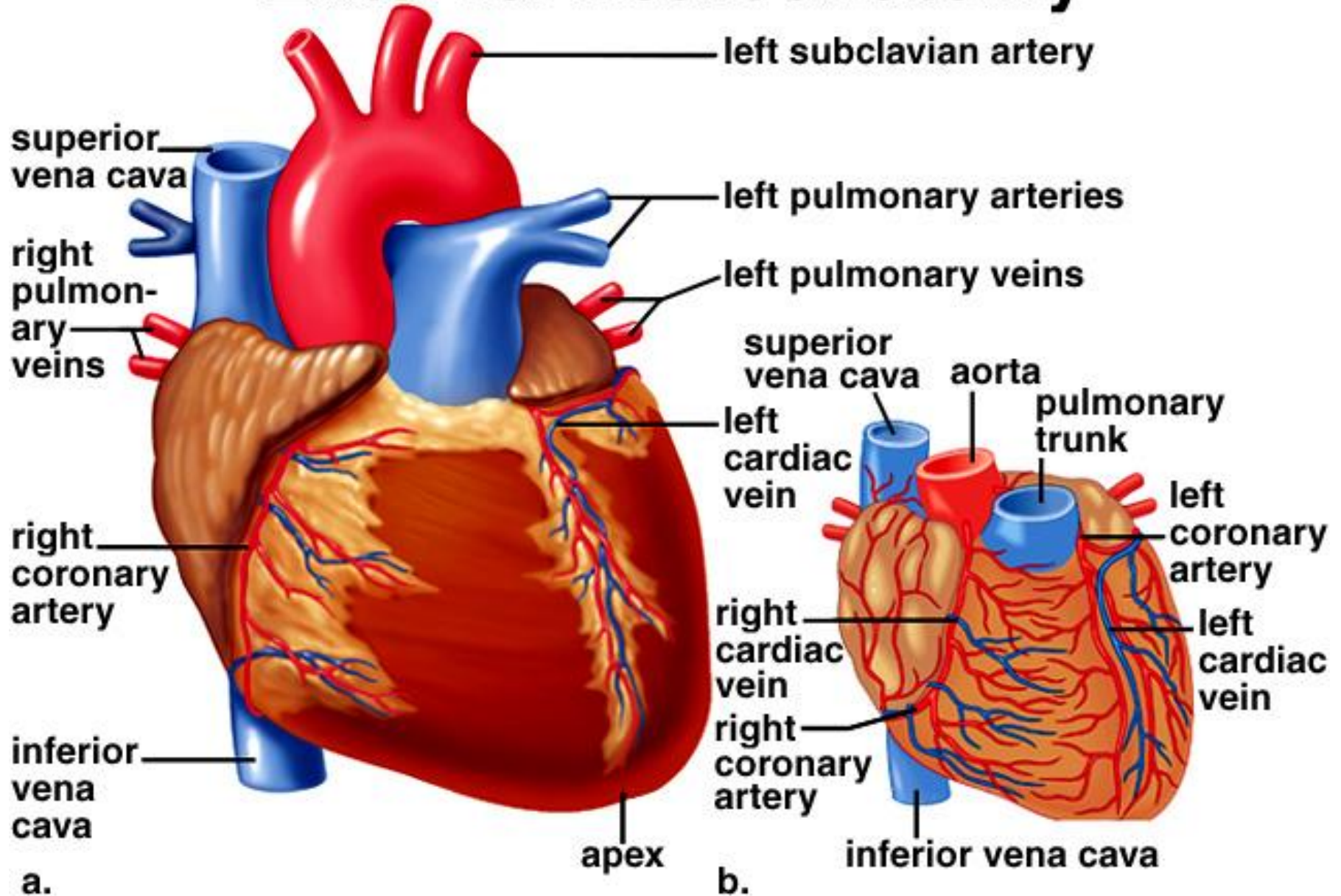
### Persistence of the Fetal Circulation



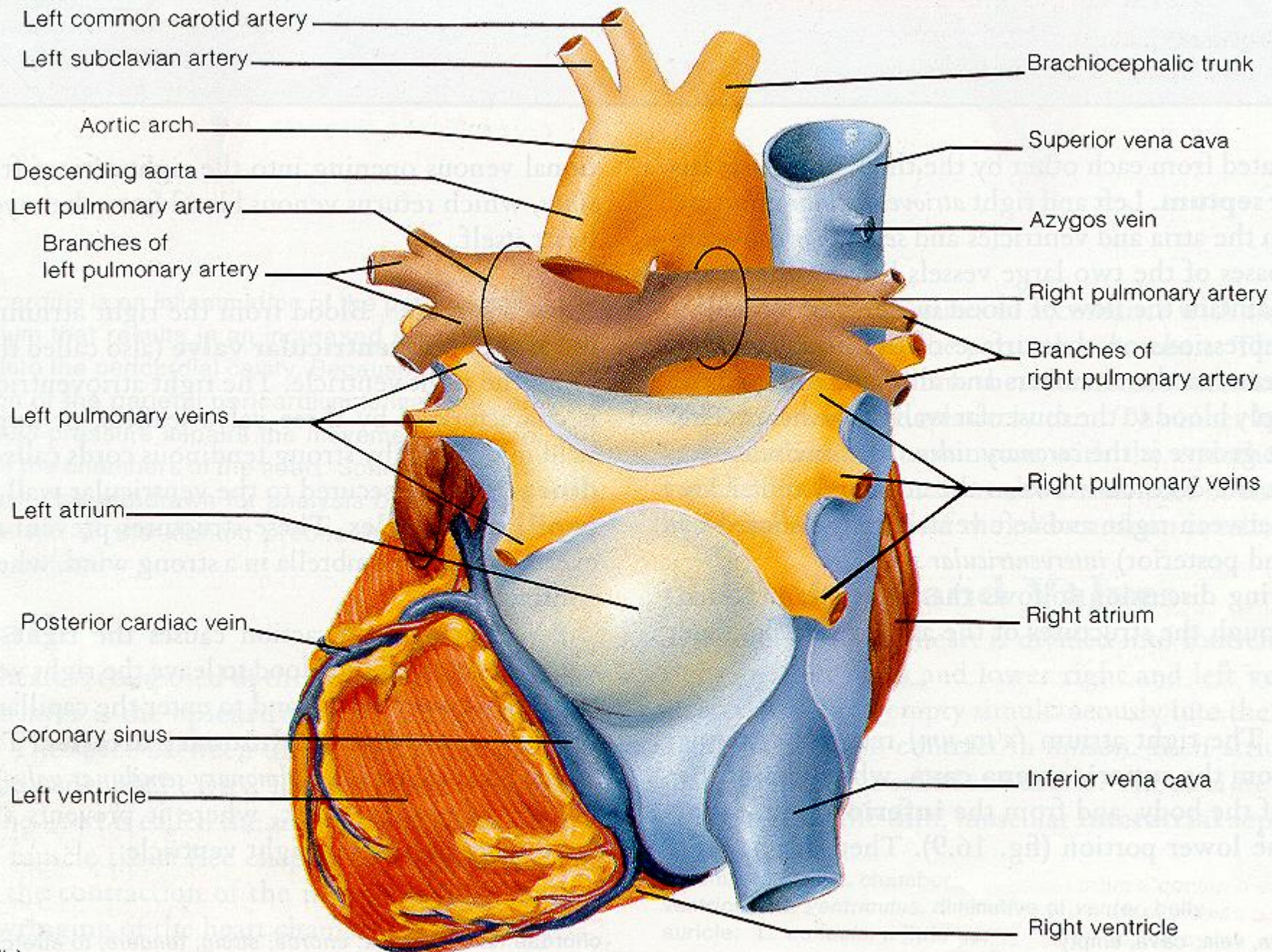




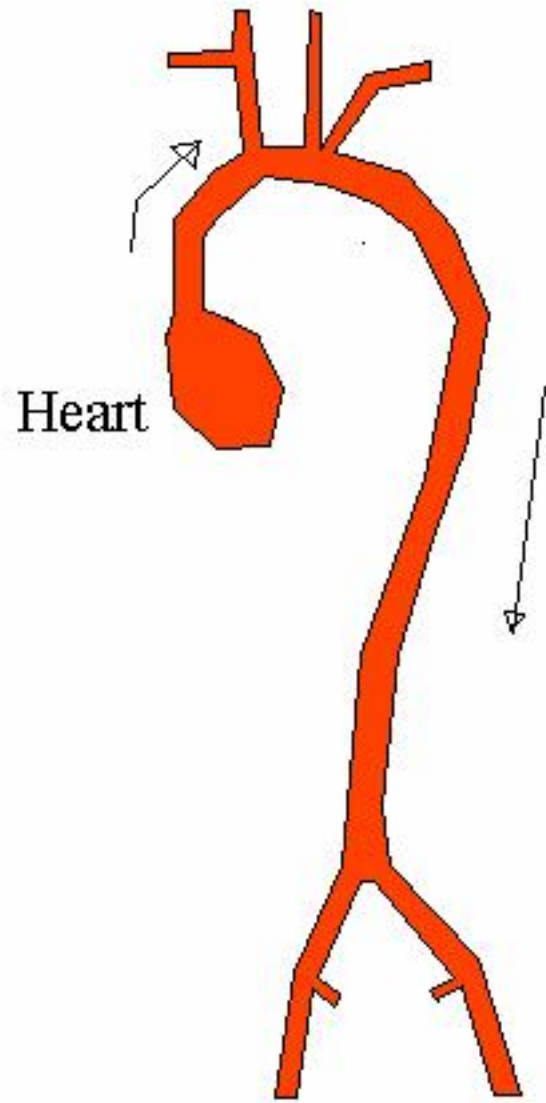
# External heart anatomy







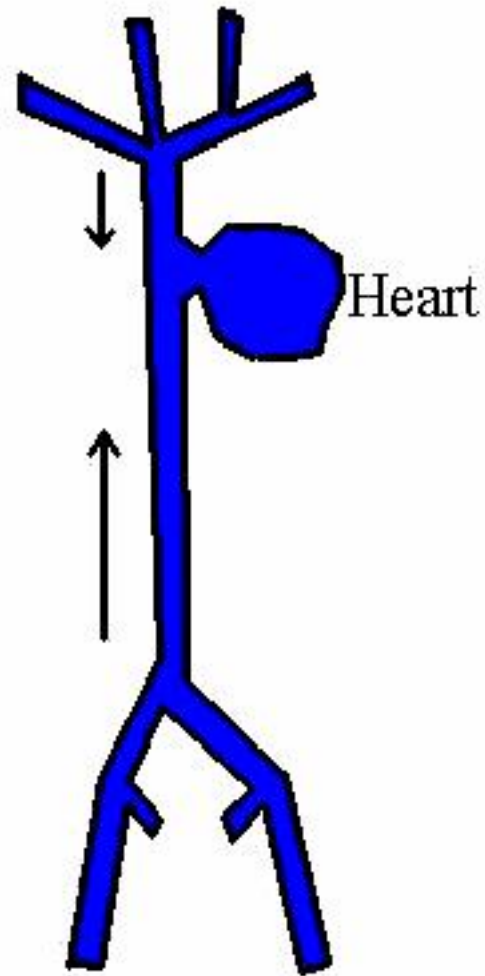
Arms and head



Heart

Arteries

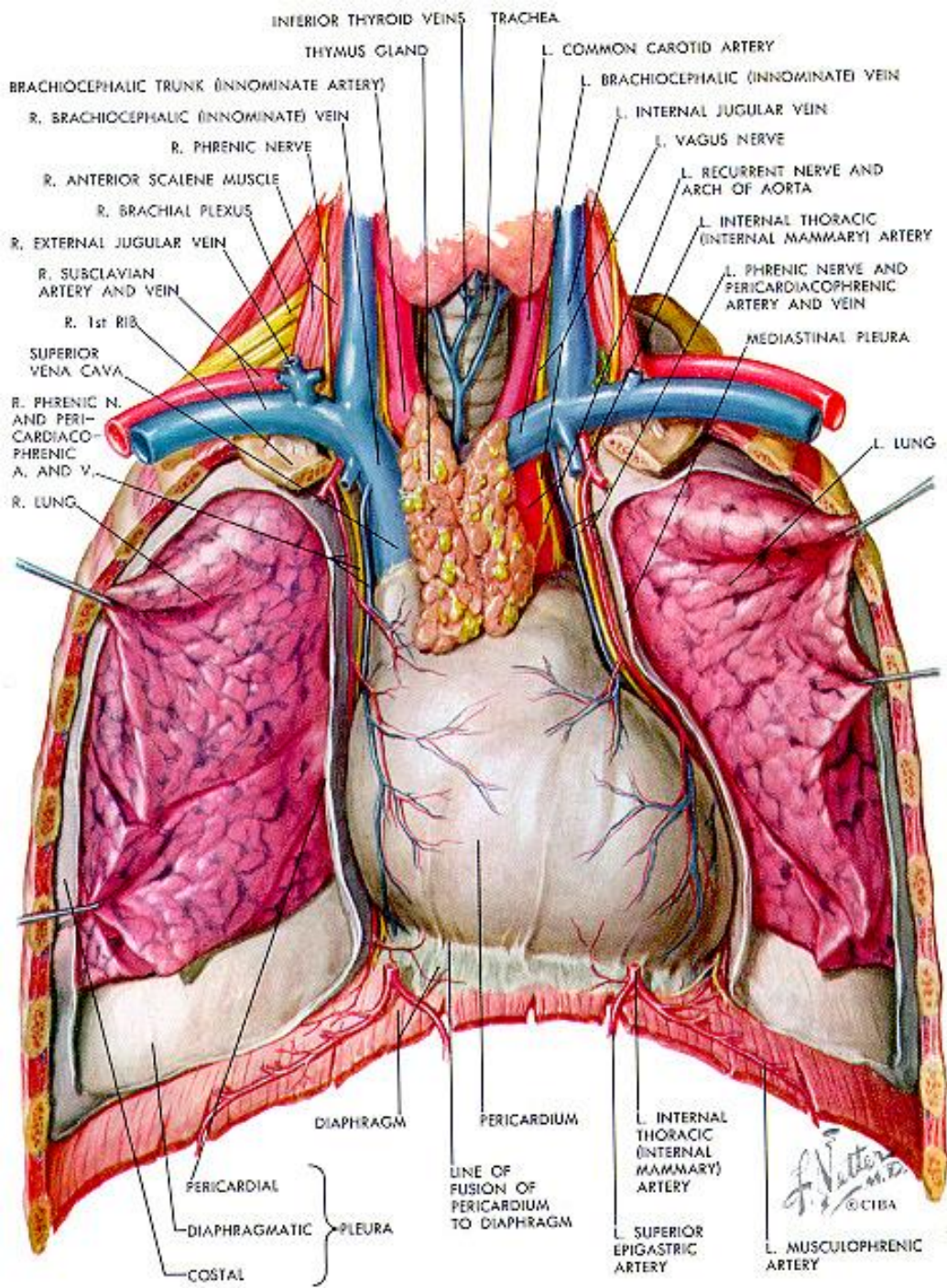
Arms and head



Heart

Veins





## Pericardium

- Fibrous- dense connective tissue that prevents outstretching of the heart and anchors the heart to the mediastinum
- Serous- thin double layered serous membrane around the heart
  - parietal- directly beneath the fibrous pericardium.
  - visceral (epicardium)- attached directly to the heart
- Pericardial fluid- a thin film of serous fluid that is contained within the pericardial sac (pericardial cavity) and serves to prevent friction between the membranes of the heart.
- Cardiac tamponade

# Pericarditis (Cow's Heart)

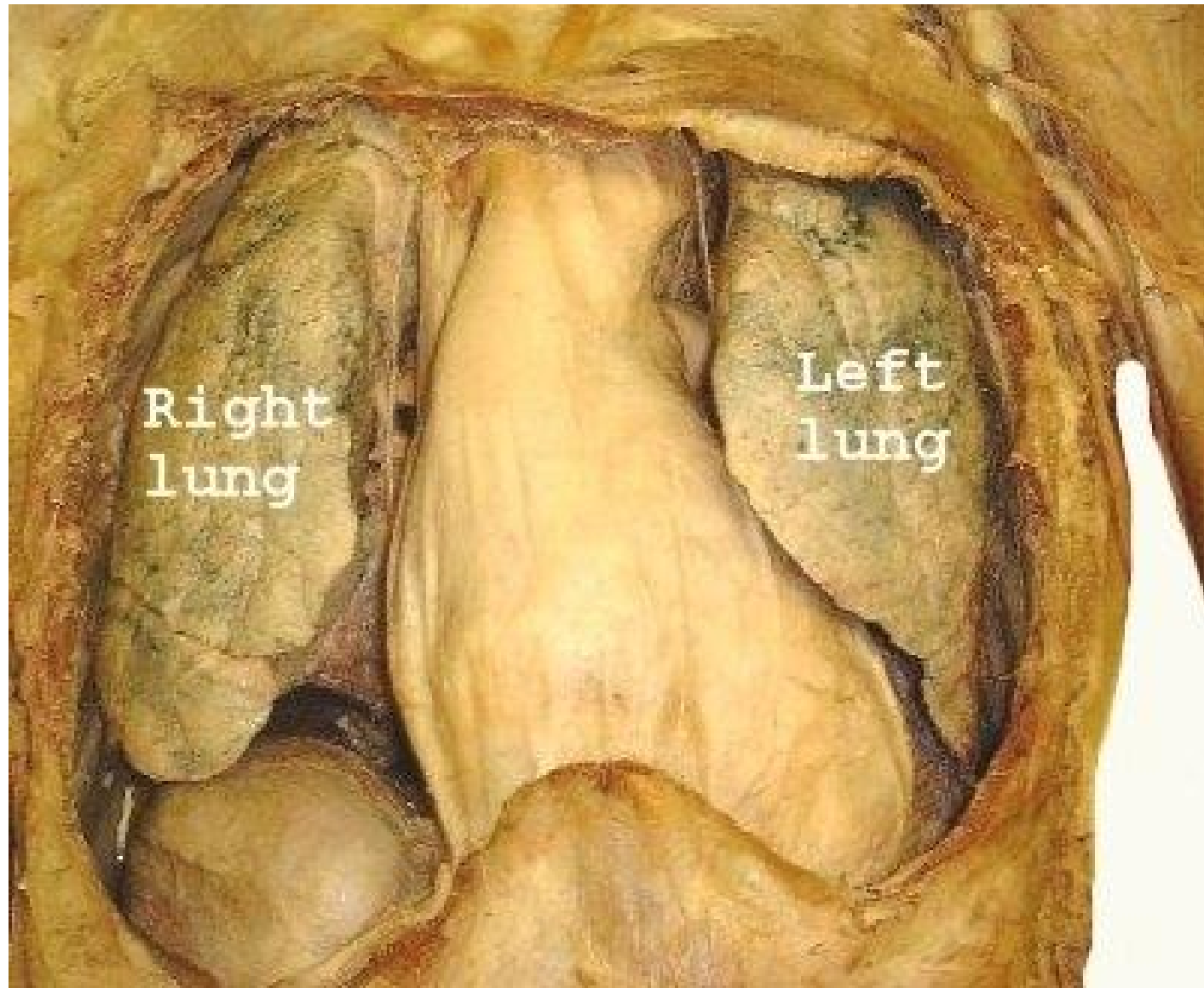


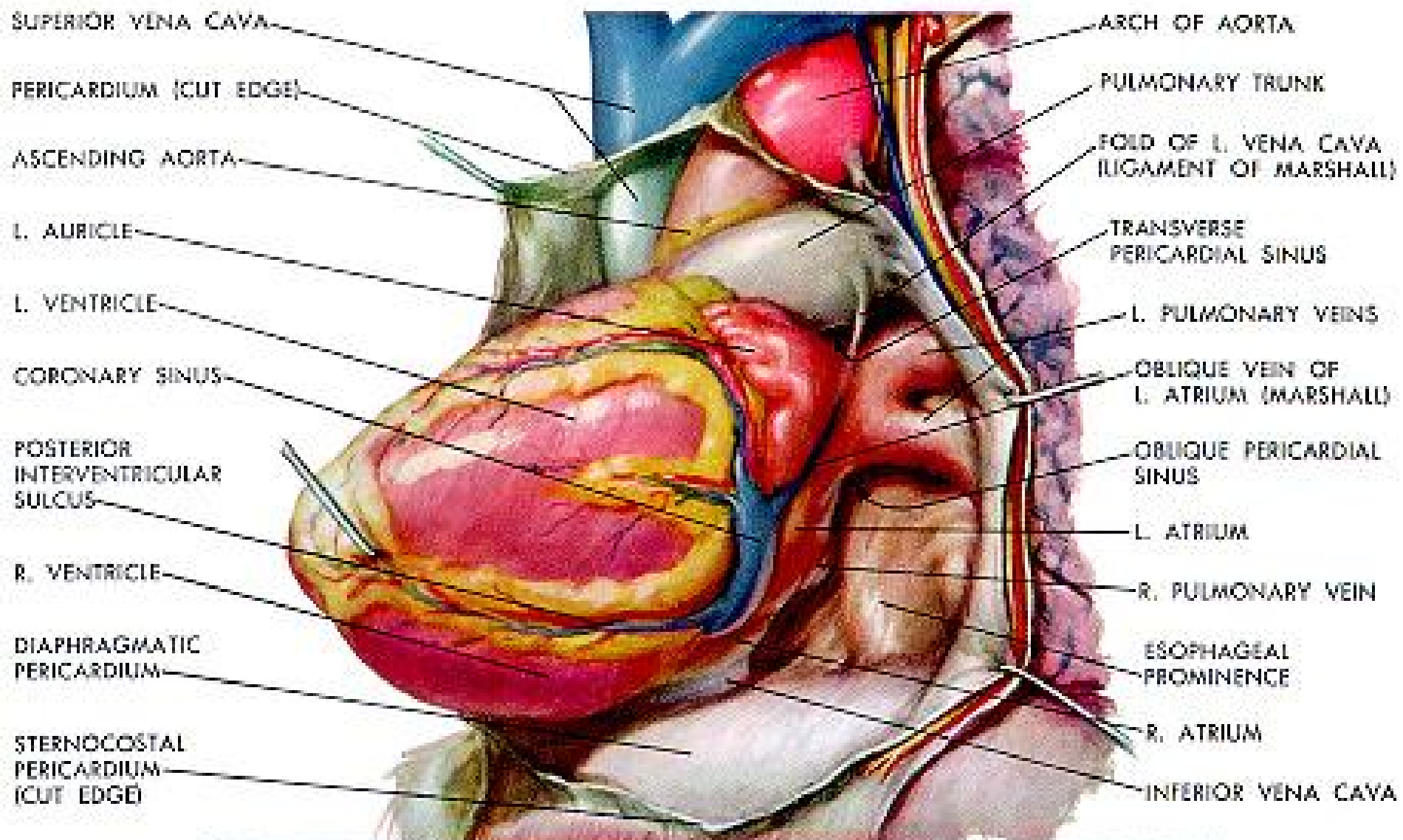
the heart sac has been opened and you can see that the heart is surrounded by fibrous material. This material is due to infection within the heart sac. This can be referred to as a "shaggy heart".



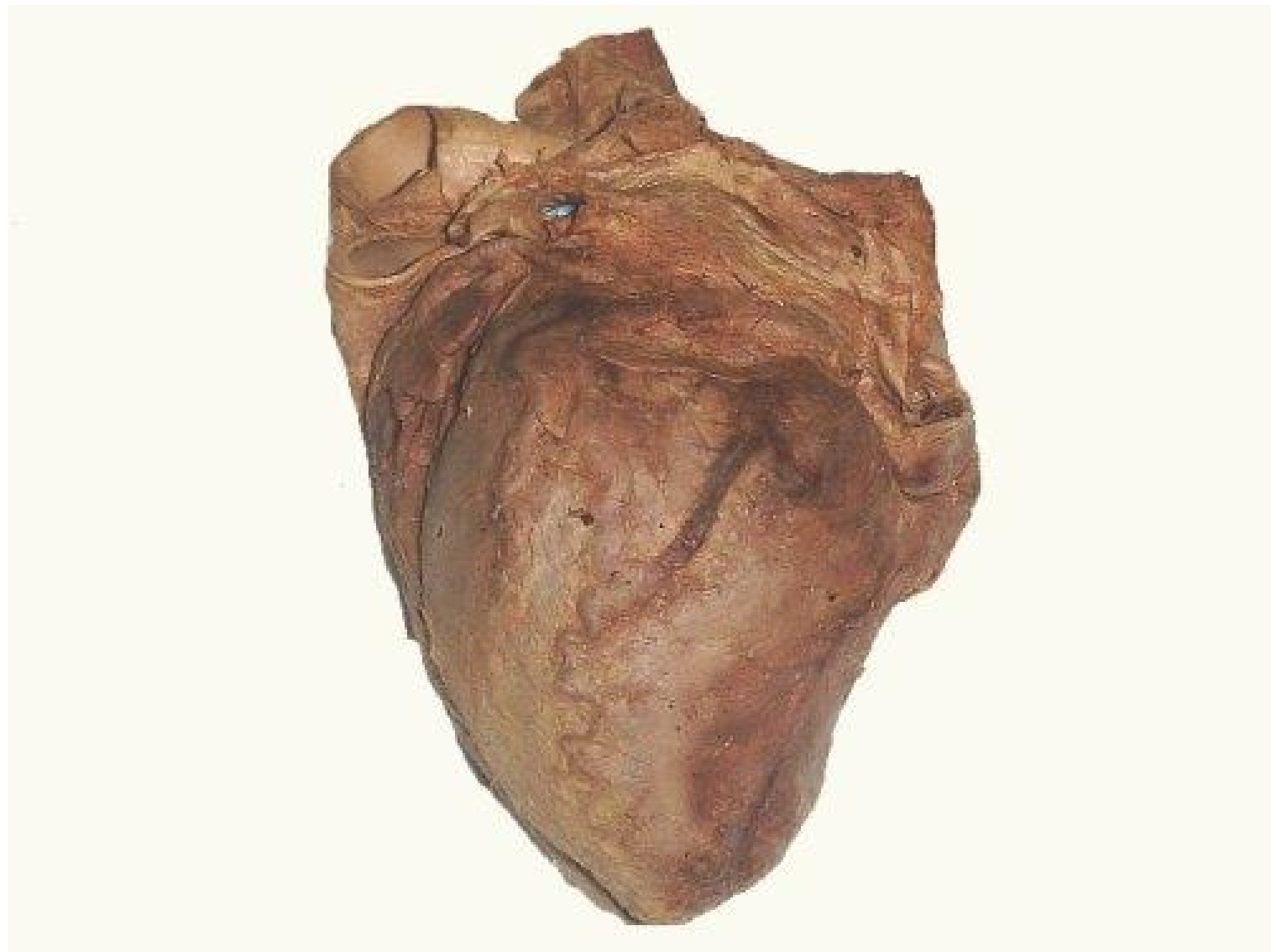
This is a heart after the fibrous material has been removed.





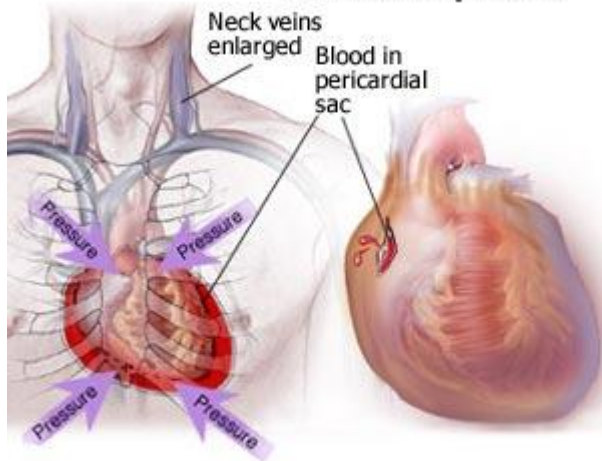


HEART DRAWN OUT OF PERICARDIAL SAC (OPENED AND VIEWED 3/4 FROM LEFT)



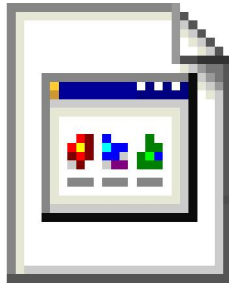


## Cardiac Tamponade

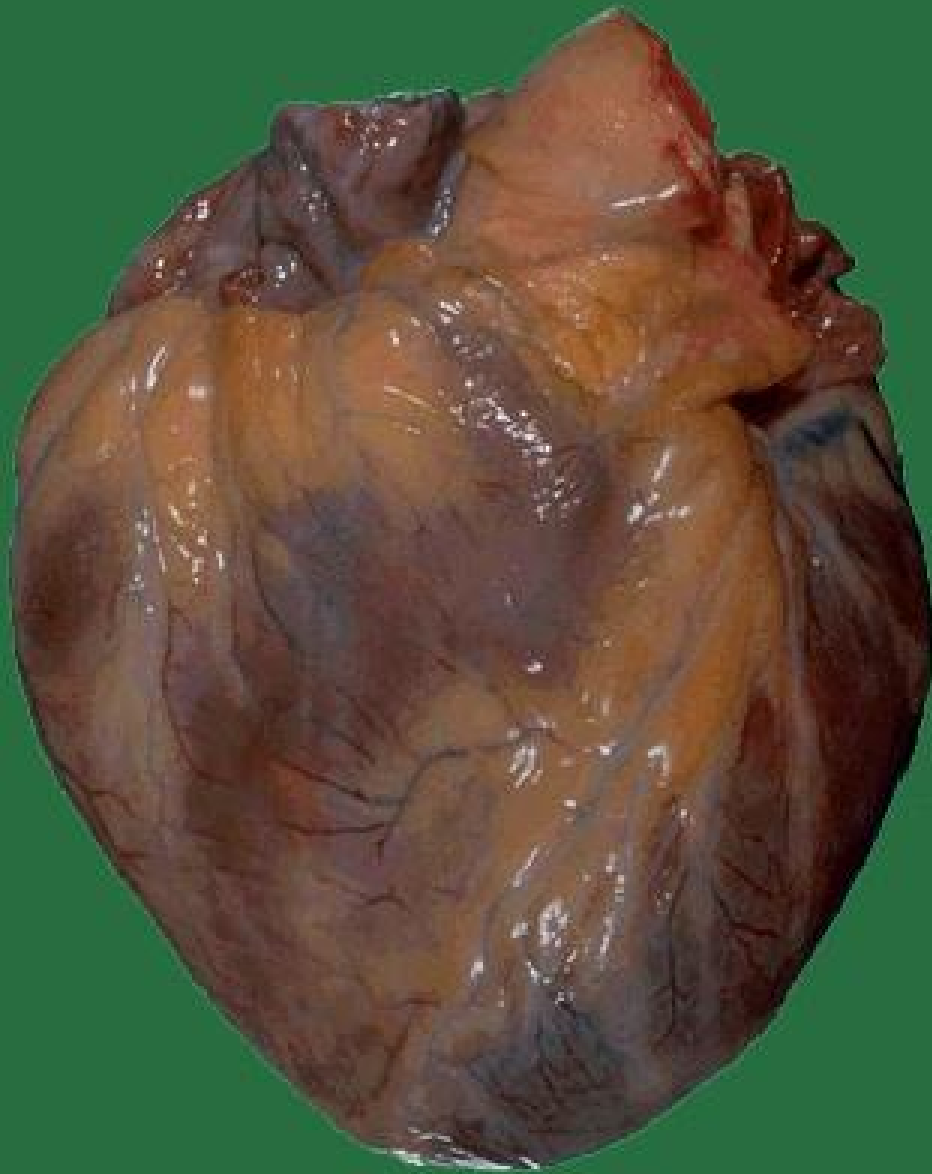


Under pressure, blood or other fluid in the pericardial sac compresses the heart, which interferes with heart function.

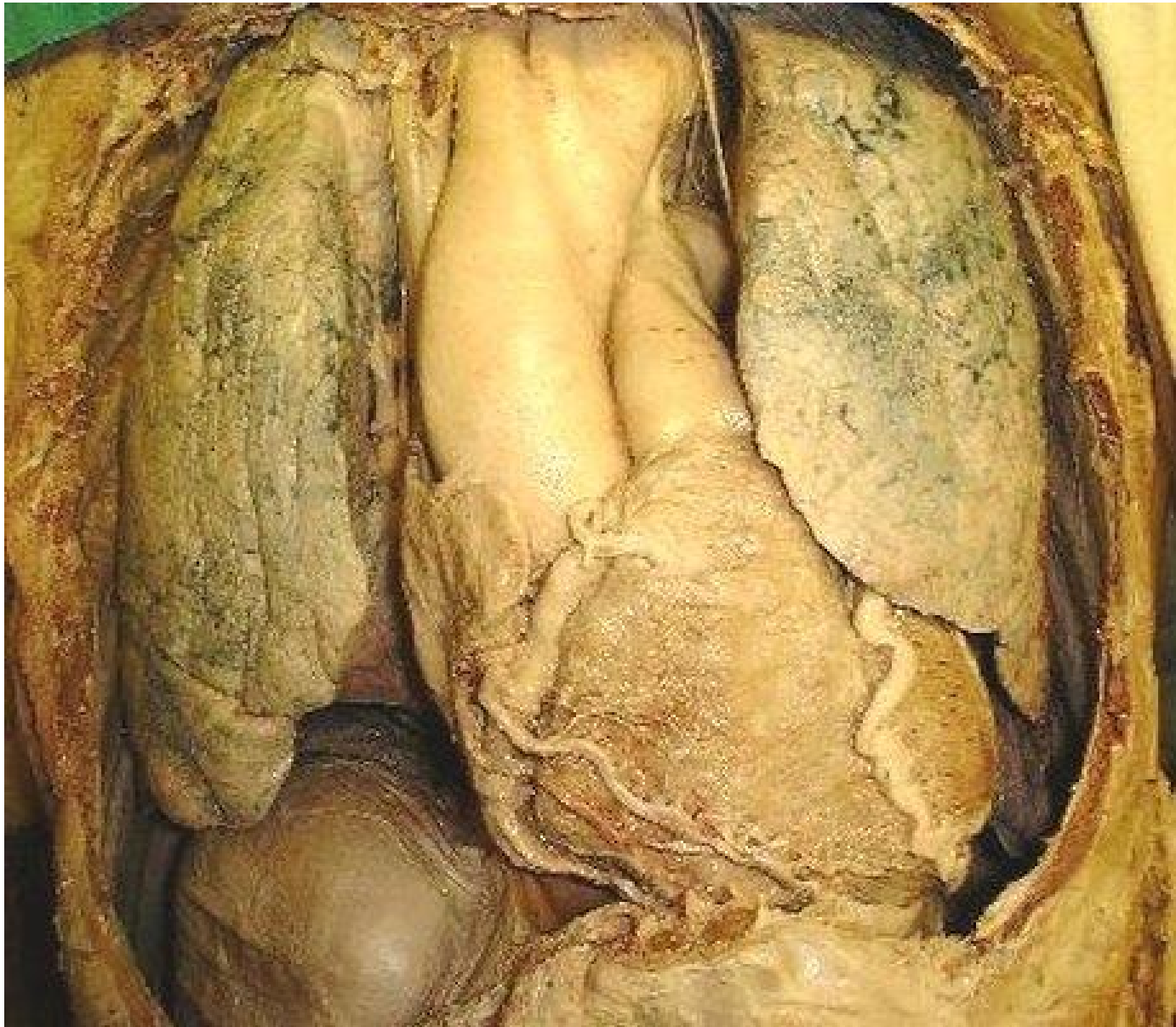




Perecardiocentesis, Movies.htm



This is the external appearance of a **normal heart**. The epicardial surface is smooth and glistening. The amount of epicardial fat is usual. The left anterior descending coronary artery extends down from the aortic root to the apex.



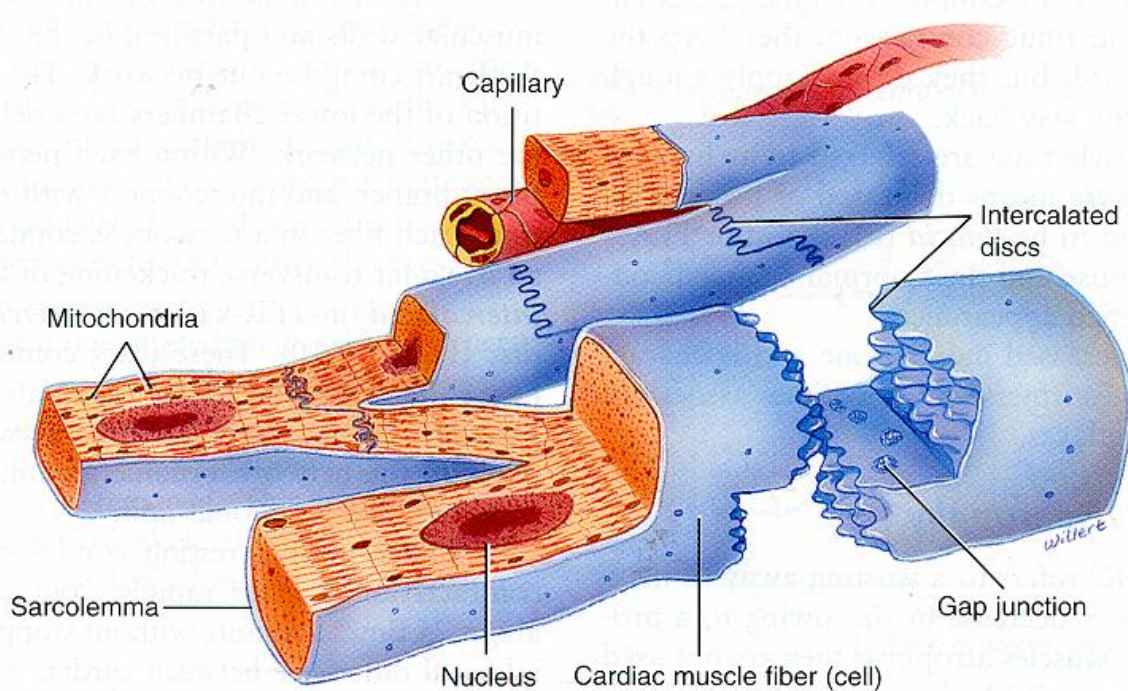
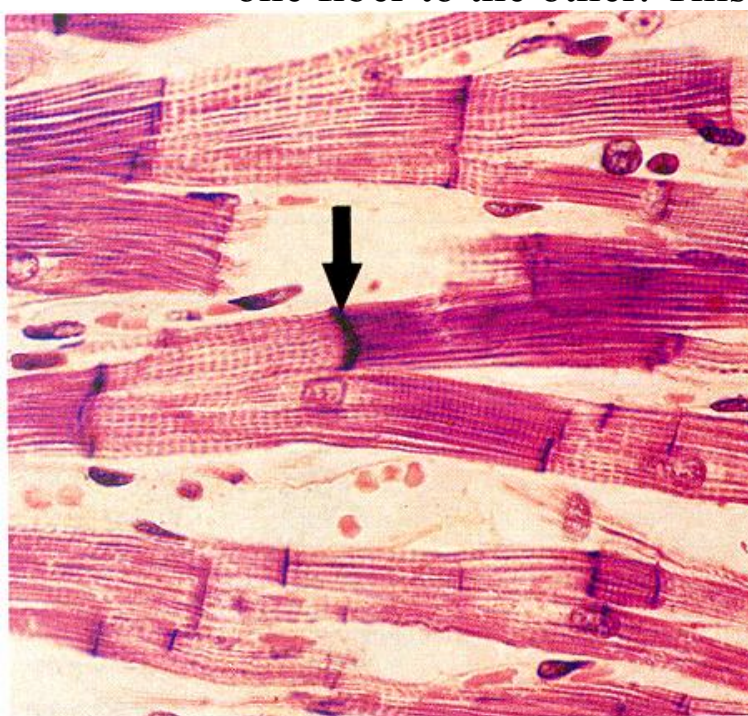


# Cardiac Muscle

- involuntary
- striated
- have only a single centrally located nuclei
- exhibit branching

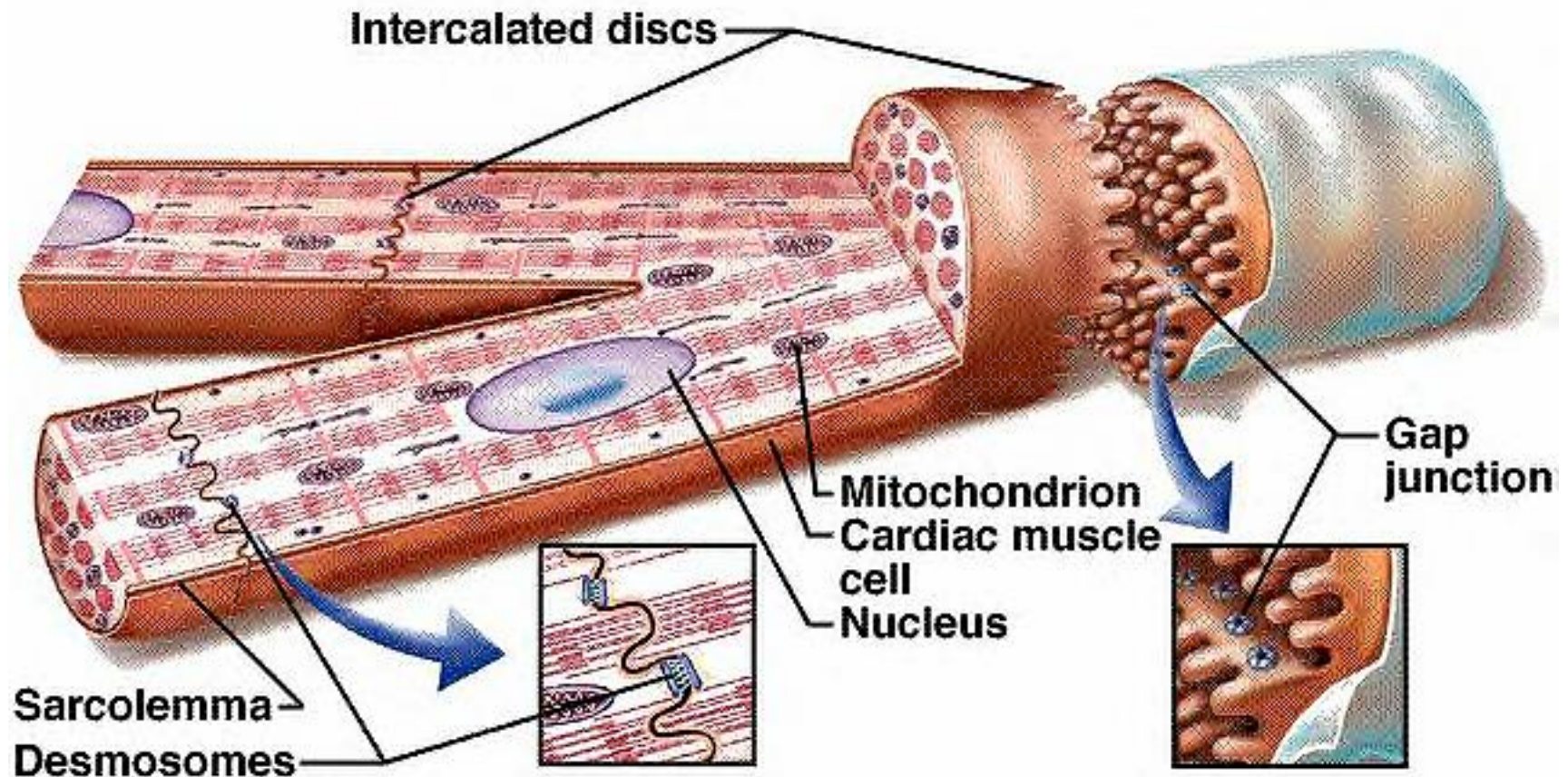
Consist of two separate networks:atria and ventricles

- fibers in each network are connected to each other via **intercalated disks** (irregular transverse thickening of the sarcolemma).
- the discs contain **gap junctions**, which conduct muscle action potentials from one fiber to the other. This enables each network to conduct as a unit.

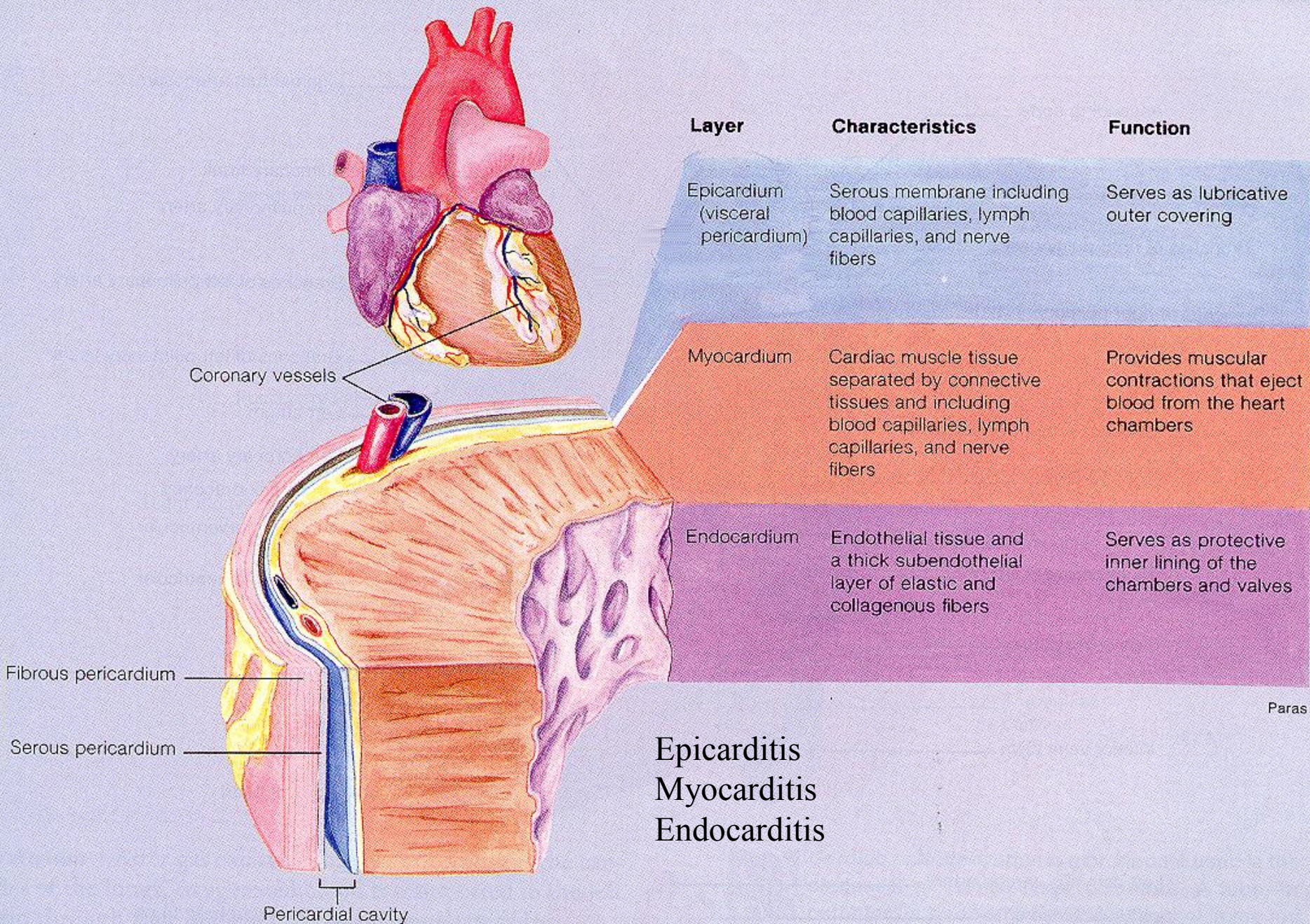




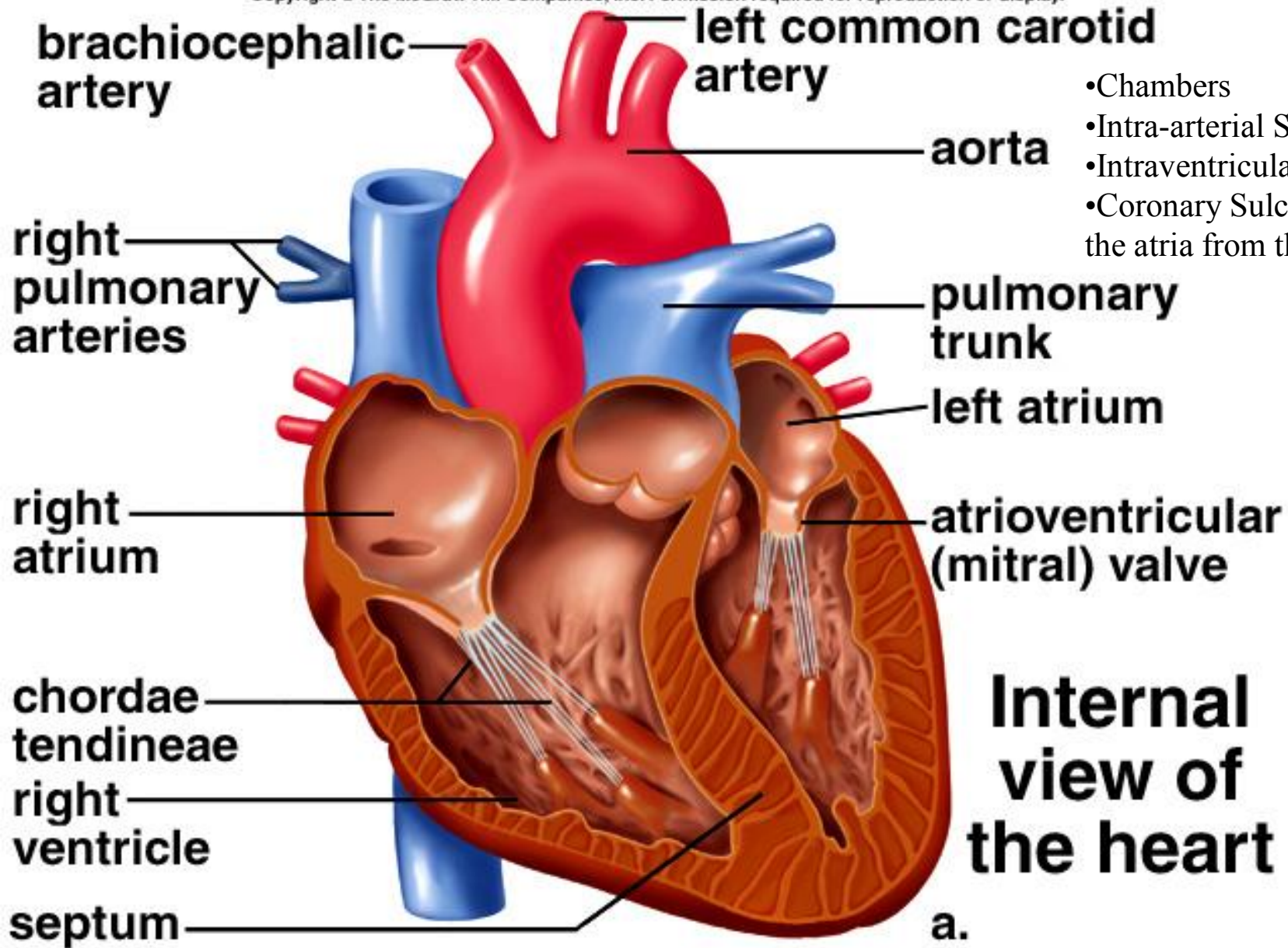
# Cardiac Muscle Structure and Intercalated Discs

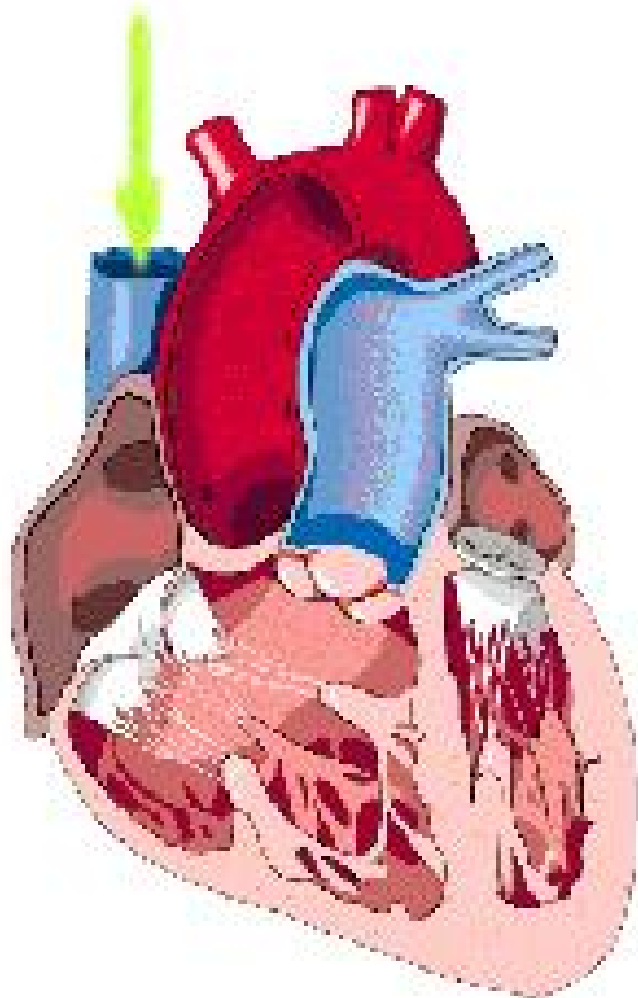












- **Right Atrium**
- **Tricuspid Valve**
- **Right Ventricle**
- **Pulmonic Valve**
- **Pulmonary Arteries**
- **Pulmonic Veins**
- **Left Atrium**
- **Mitral Valve**
- **Left Ventricle**
- **Aortic Valve**
- **Aorta**

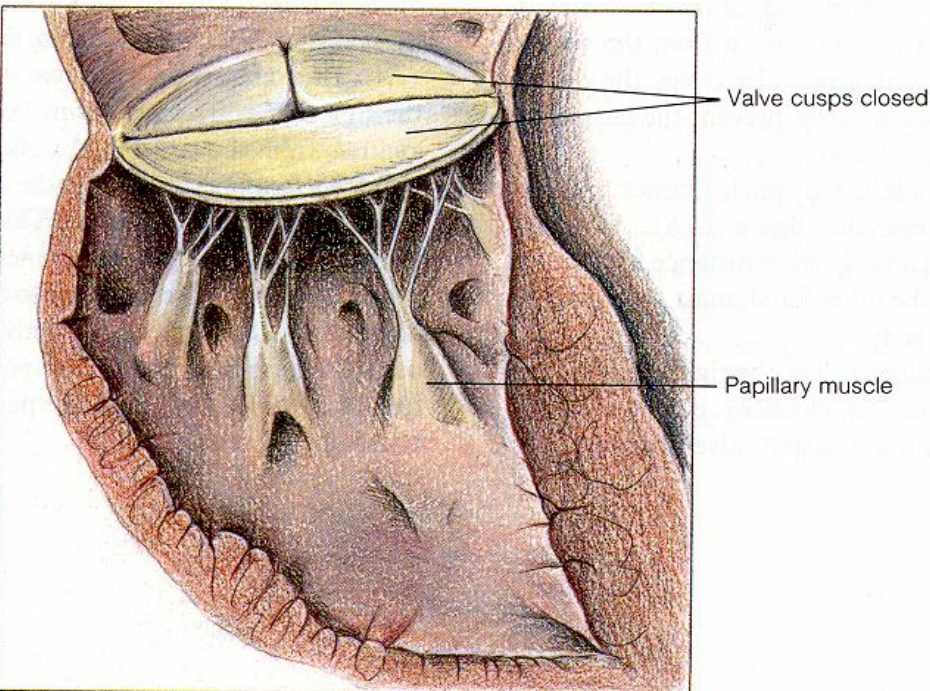
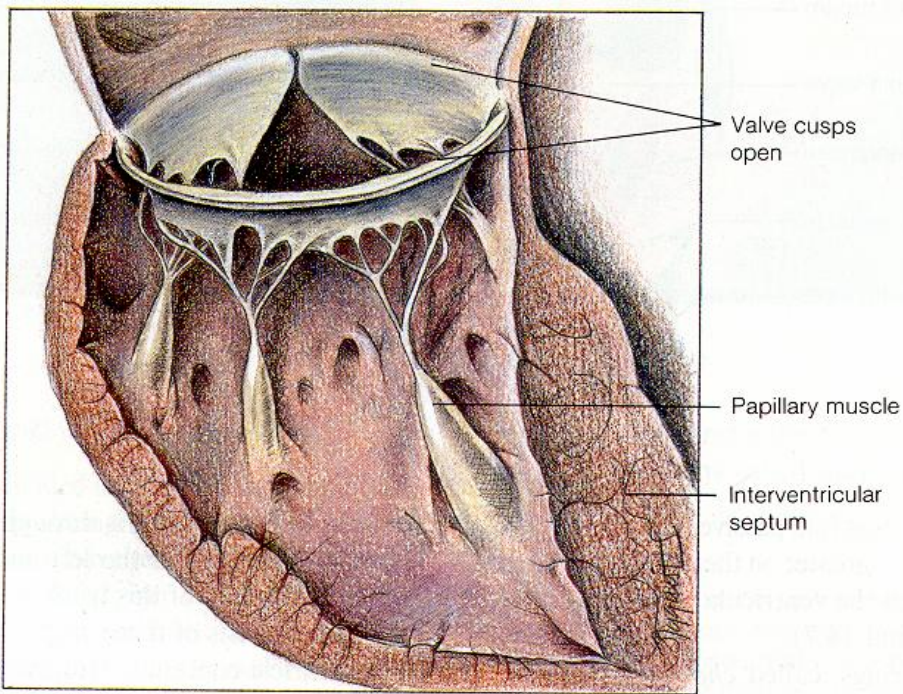
Atrioventricular Valves- prevent  
backflow into the atria

tricuspid

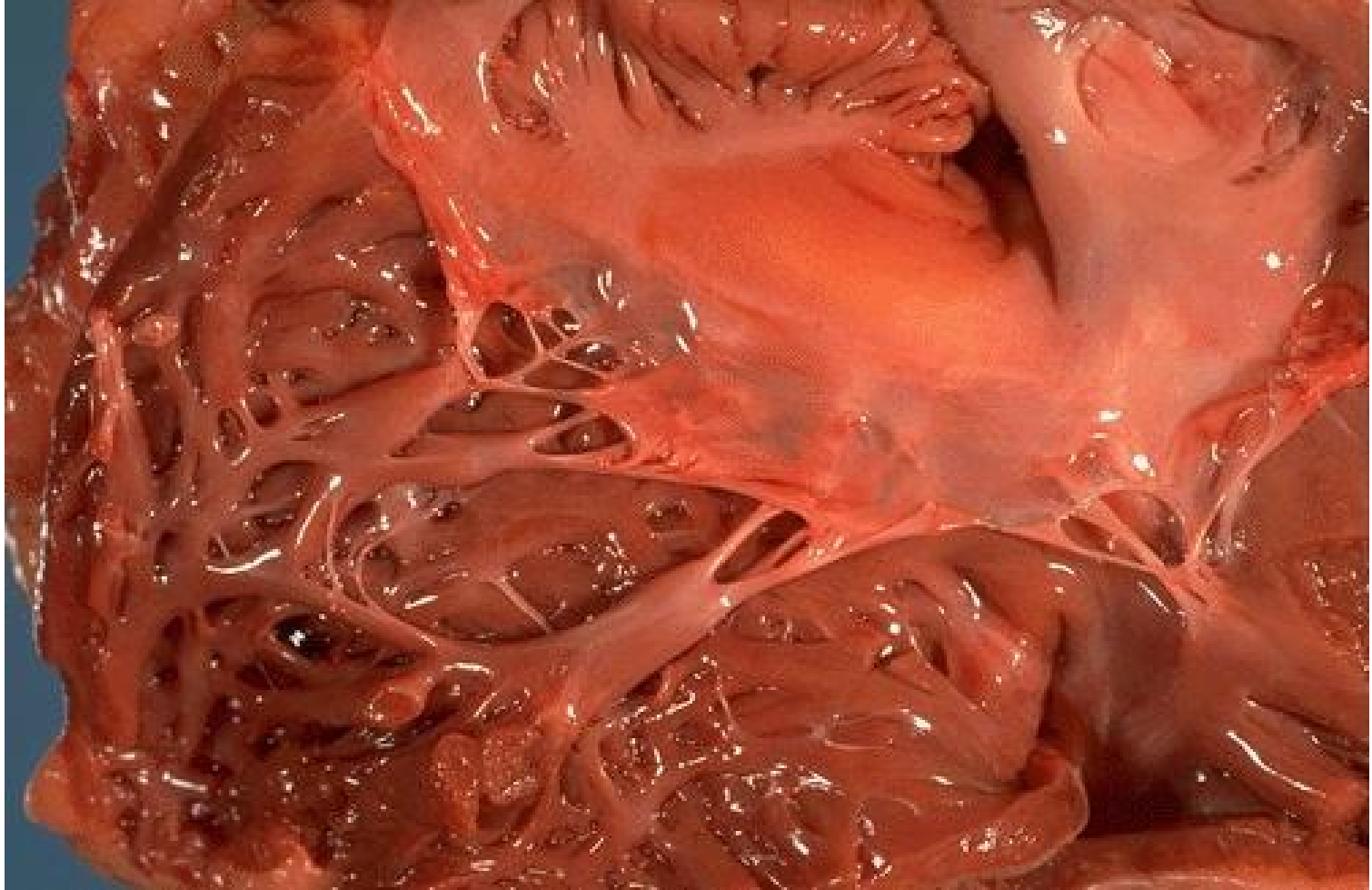
bicuspid (mitral)

papillary muscles

chordae tendineae





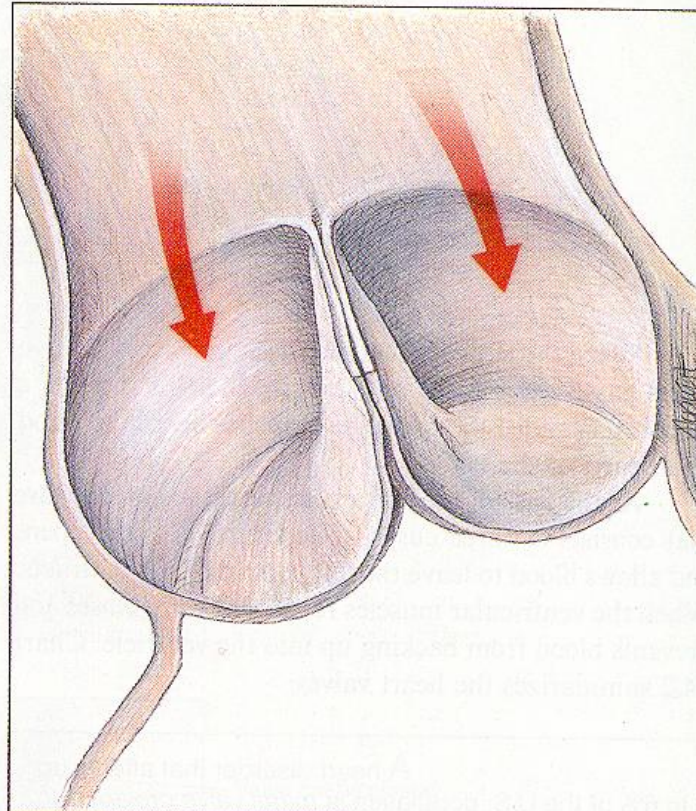
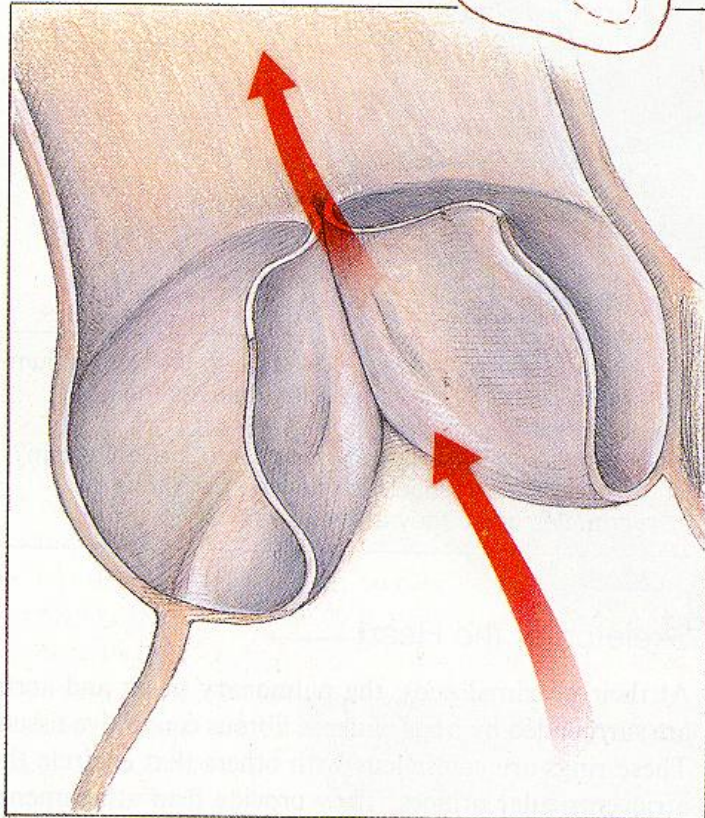
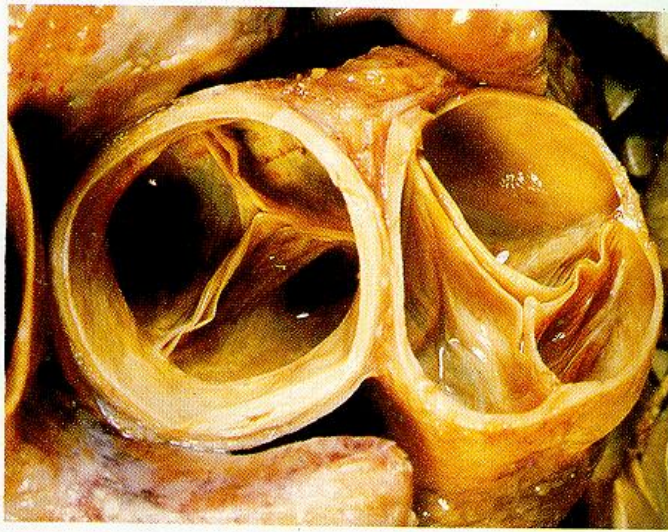
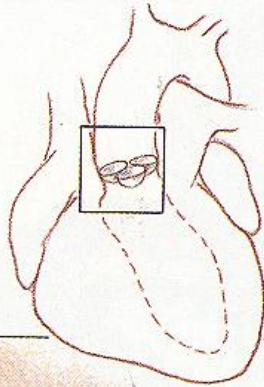


- This is the tricuspid valve. The leaflets are thin and delicate. Just like the mitral valve, the leaflets have thin chordae tendineae that attach the leaflet margins to the papillary muscles of the ventricular wall below.

Semilunar Valves- prevent backflow  
into the ventricles

Aorta

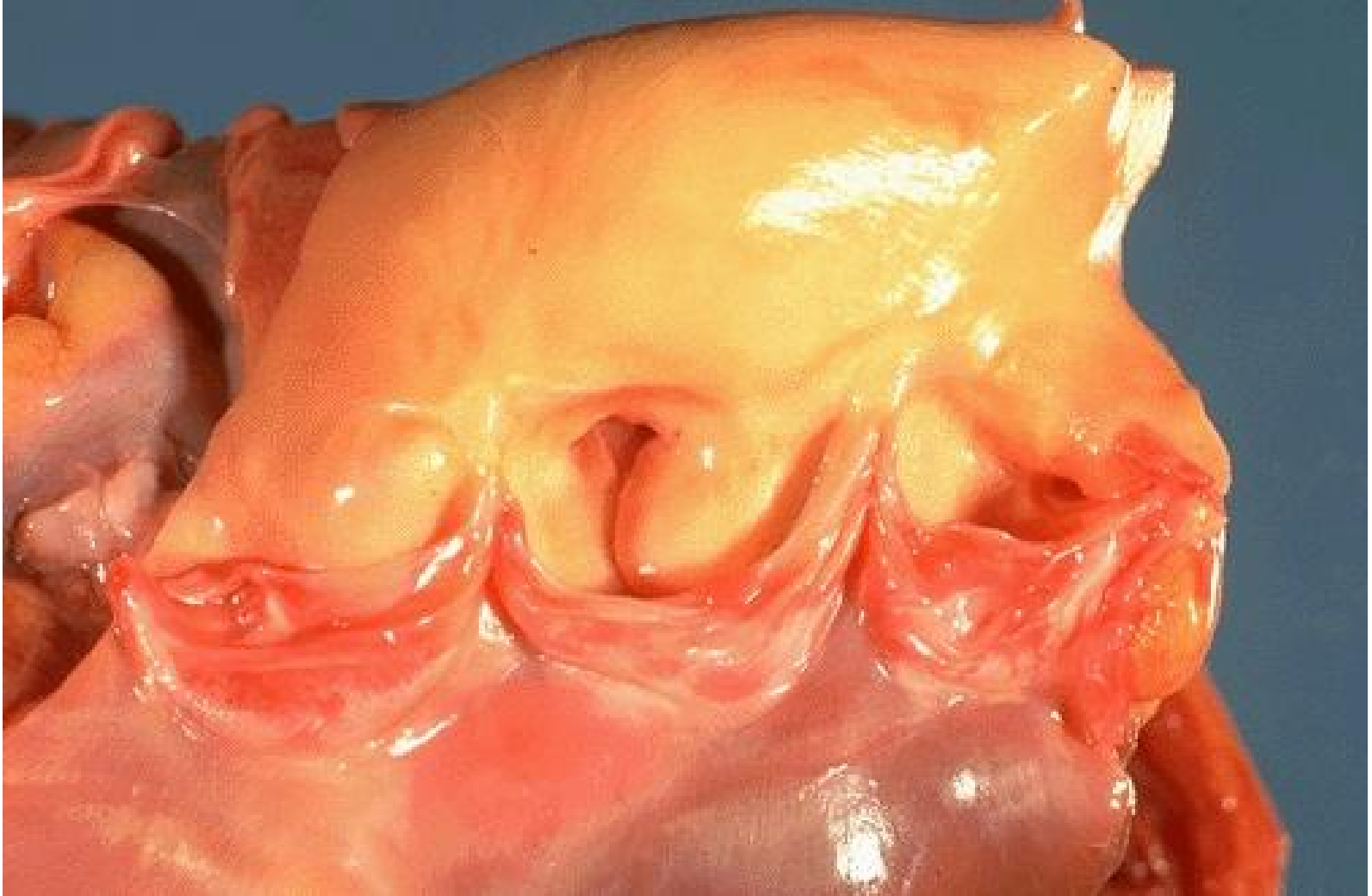
Pulmonary



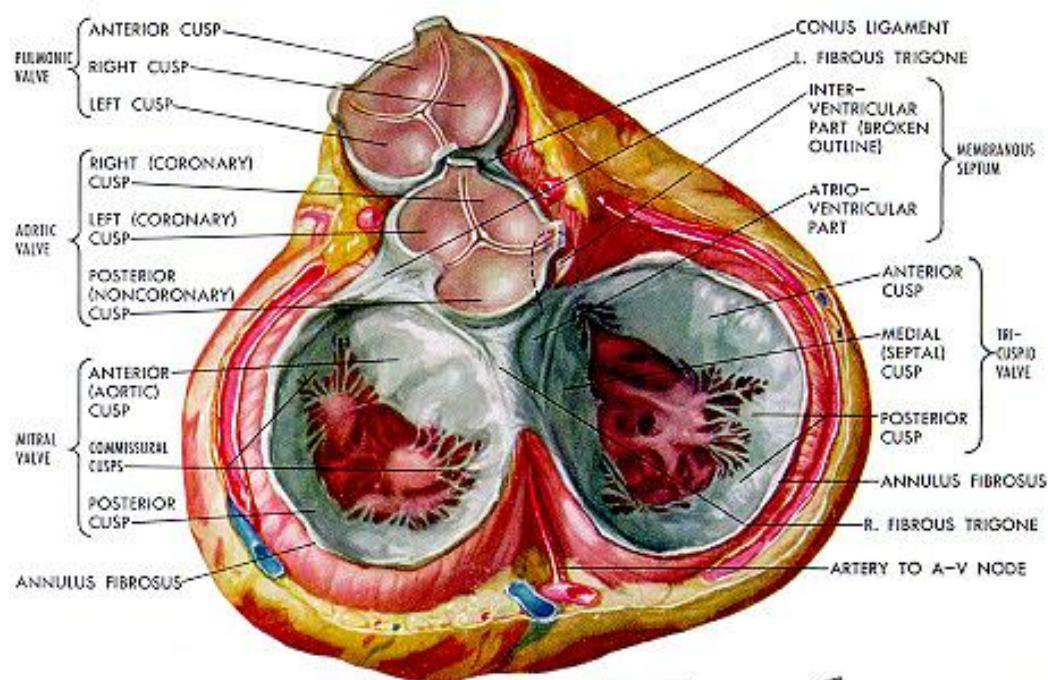
(b)

Nyquist



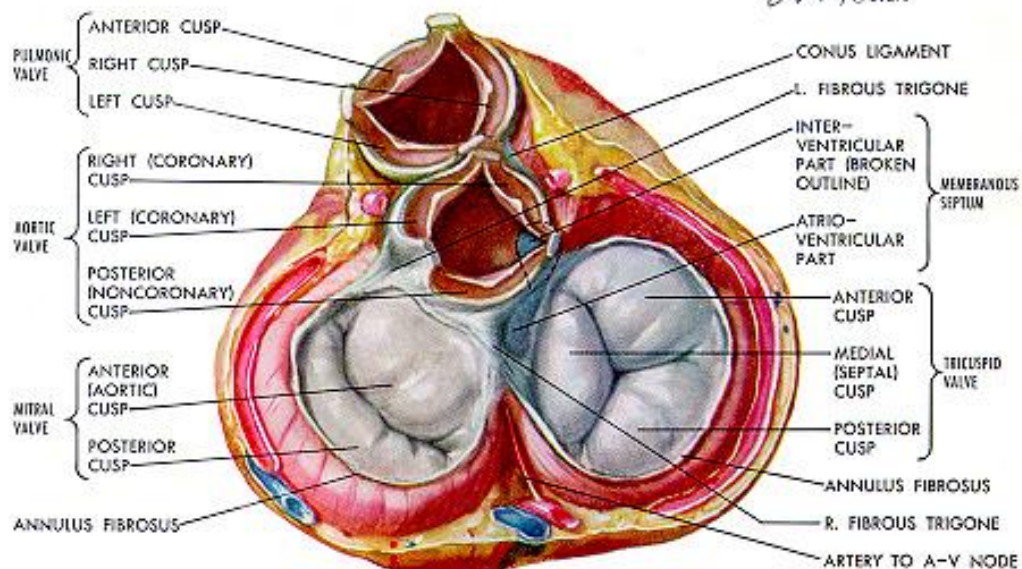


- The aortic valve shows three thin and delicate cusps. The coronary artery orifices can be seen just above. The endocardium is smooth, beneath which can be seen a red-brown myocardium. The aorta above the valve displays a smooth intima with no atherosclerosis.



THE HEART IN DIASTOLE: VIEWED FROM BASE WITH ATRIA REMOVED

*J. Netter M.D.*  
© CIBA



THE HEART IN SYSTOLE: VIEWED FROM BASE WITH ATRIA REMOVED



Opening  
Pericardium



Viewing and lifting the  
heart



Heart Valve Movie

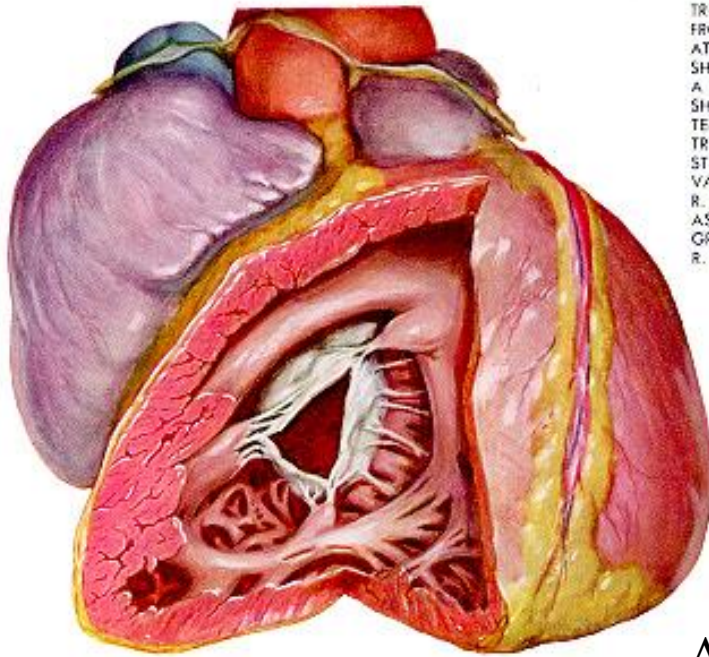


Pumping of  
heat in chest  
cavity



Heart removal  
from the chest



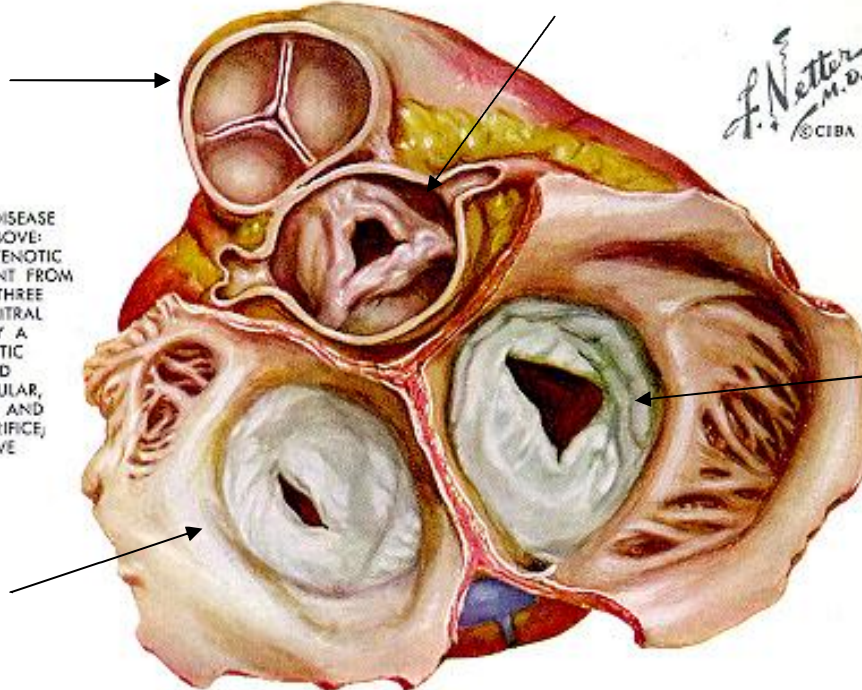


TRICUSPID VALVE VIEWED FROM BELOW: SOME FUSION AT EACH COMMISSURE, SHORTENING OF CUSPS, AND A LITTLE THICKENING AND SHORTENING OF CHORDAE TENDINEAE, LEAVING A TRIANGULAR ORIFICE OF A STENOTIC, INSUFFICIENT VALVE; HYPERTROPHY OF R. VENTRICLE DUE TO ASSOCIATED MITRAL DISEASE; GREAT ENLARGEMENT OF R. ATRIUM

Valvular Heart Disease  
Valvular incompetence  
Stenosis  
Regurgitation

Aorta

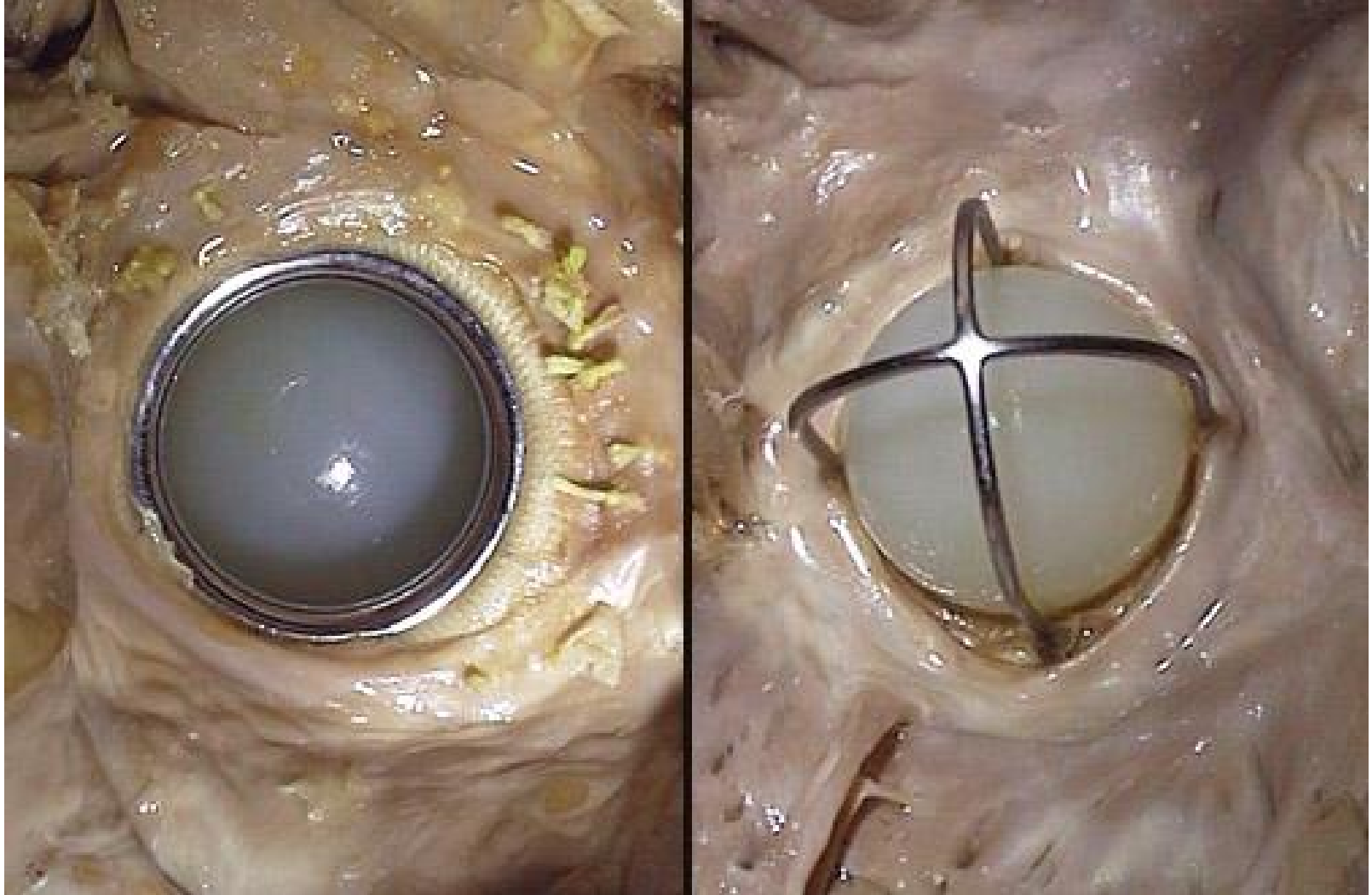
Pulmonary



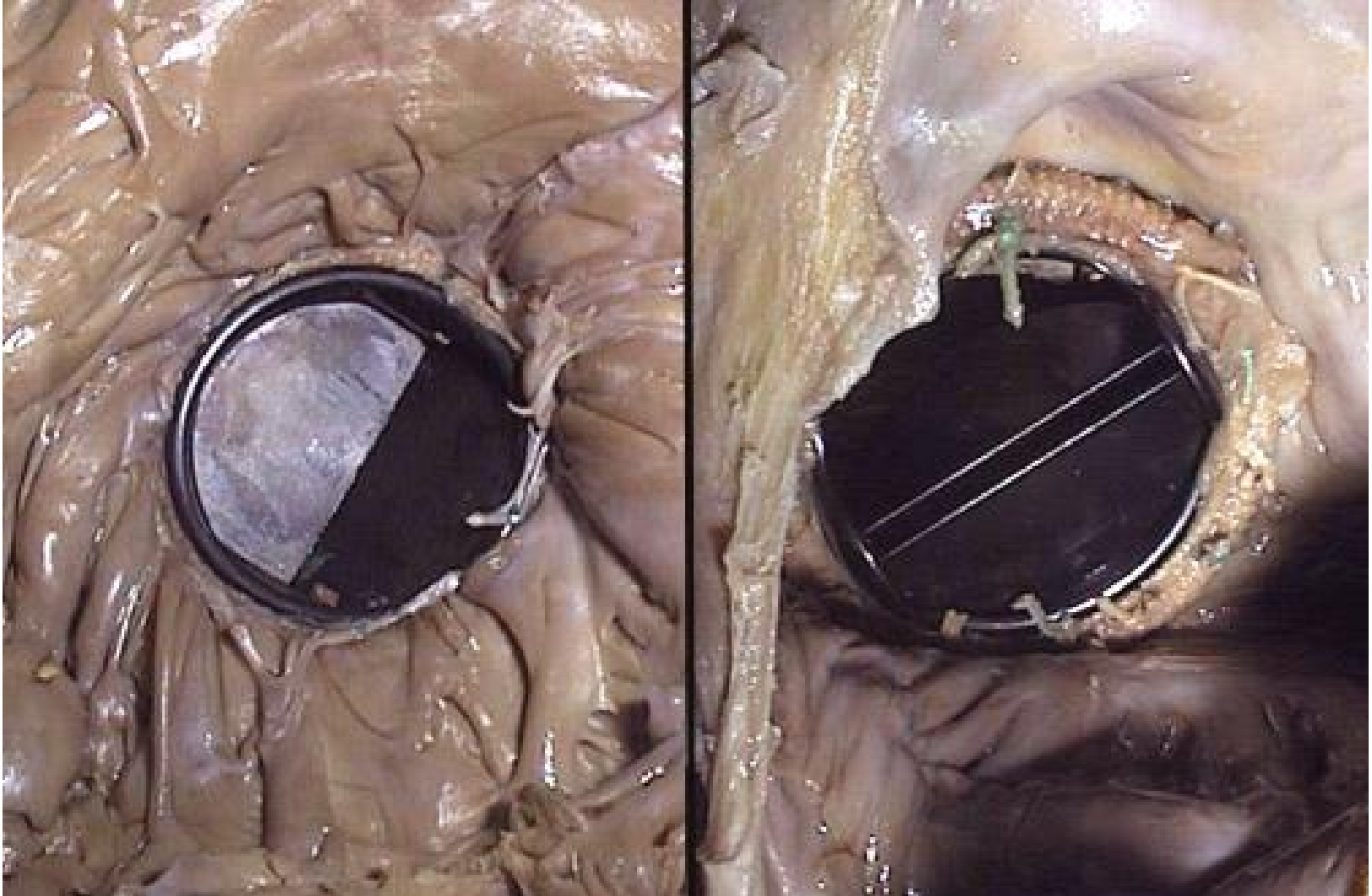
MULTIVALVULAR DISEASE VIEWED FROM ABOVE: AORTIC VALVE STENOTIC AND INCOMPETENT FROM FUSION OF ALL THREE COMMISSURES; MITRAL VALVE HAS ONLY A "SILT-LIKE" STENOTIC ORIFICE, TRICUSPID VALVE A TRIANGULAR, FIXED, STENOTIC, AND INCOMPETENT ORIFICE; PULMONARY VALVE NORMAL

Mitral

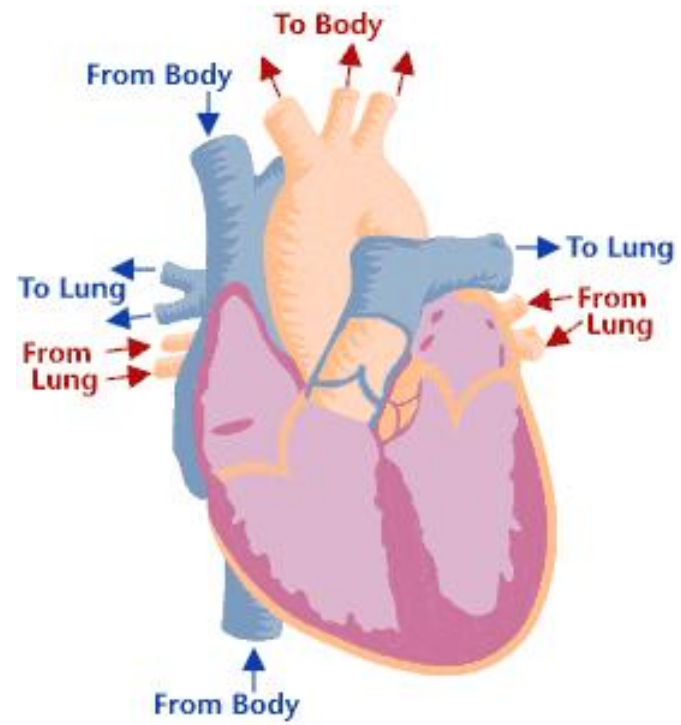
Tricuspid



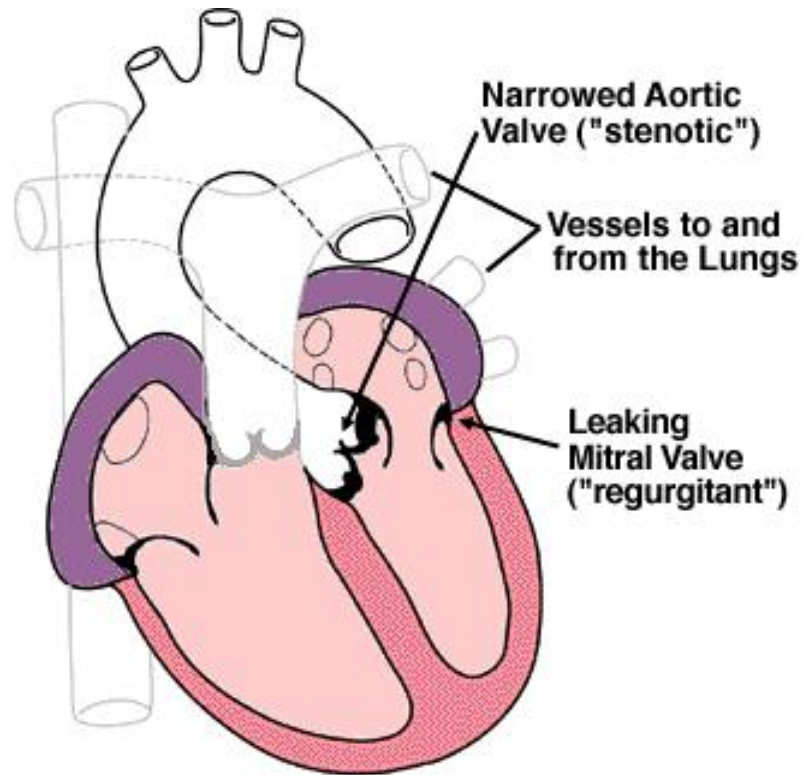
- This is a mechanical valve prosthesis of the older ball and cage variety. Such mechanical prostheses will last indefinitely from a structural standpoint, but the patient requires continuing anticoagulation because of the exposed non-biologic surfaces. The superior aspect (here the left atrium) is seen at the left, while the outflow is at the right into the left ventricle in this mitral valve prosthesis.



- This is a mechanical valve prosthesis of the more modern tilting disk variety. Such mechanical prostheses will last indefinitely from a structural standpoint, but the patient requires continuing anticoagulation because of the exposed non-biologic surfaces. The superior aspect (here the left atrium) is seen at the left, while the outflow, with the two leaflets tilted outward toward the left ventricle, is at the right in this mitral valve prosthesis.



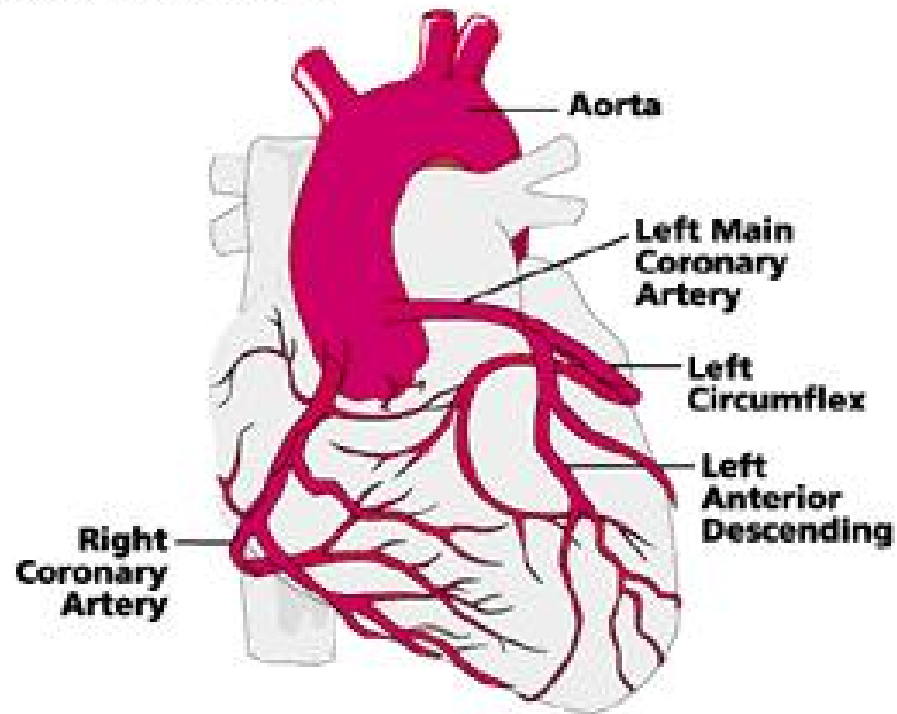
# Valvular Regurgitation and Stenosis





# Coronary Circulation

© 1997 HeartPoint



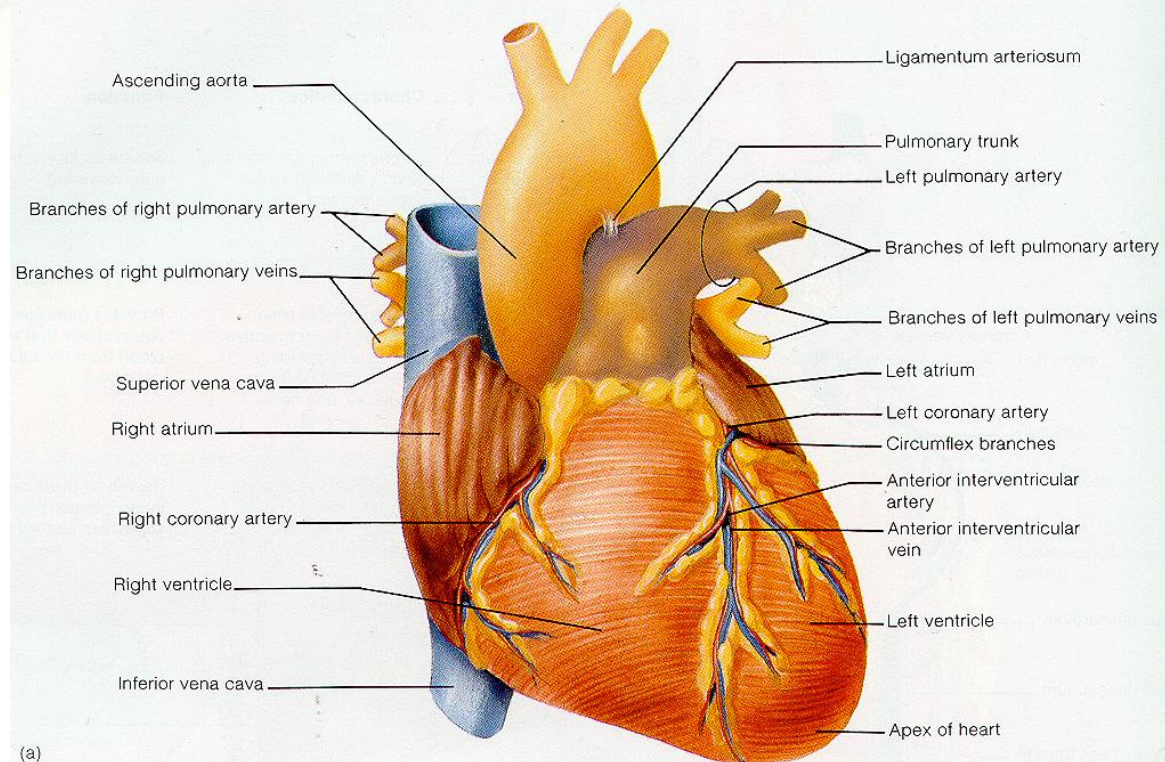
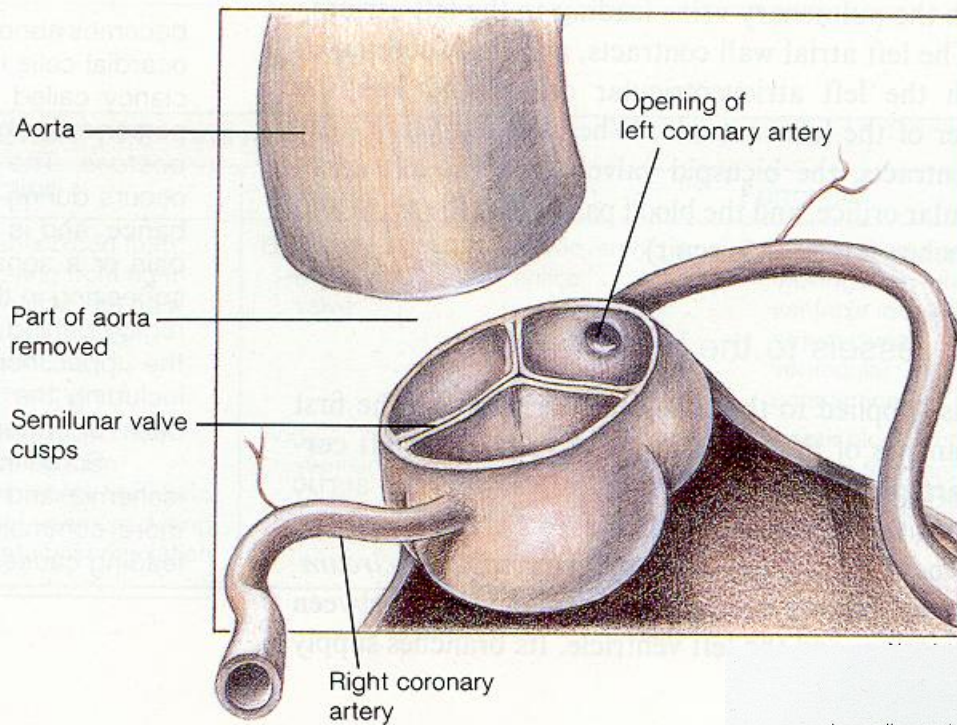
# Coronary Circulation

Ischemia

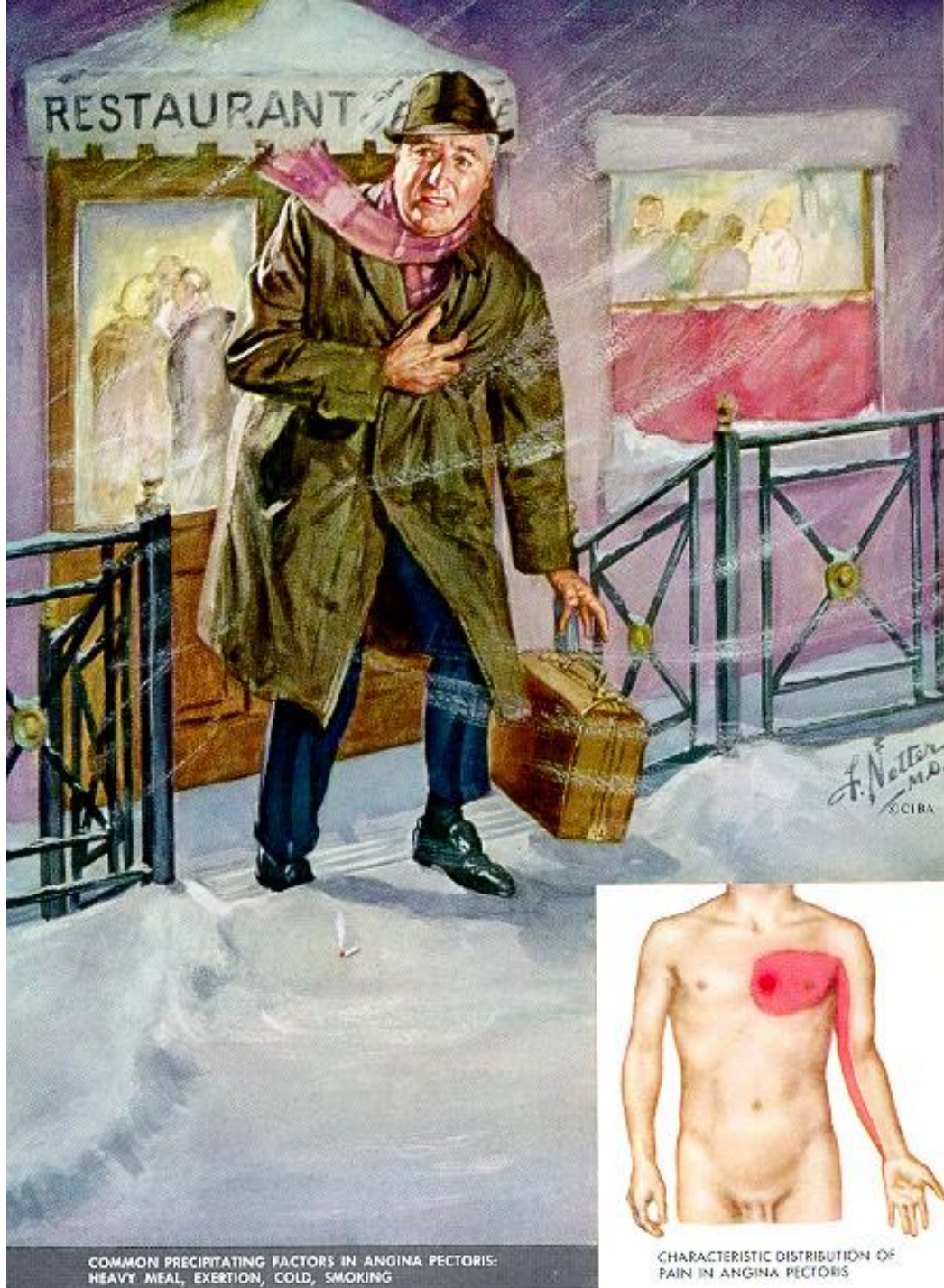
Angina pectoris

Myocardial infarction

Ventricular fibrillation



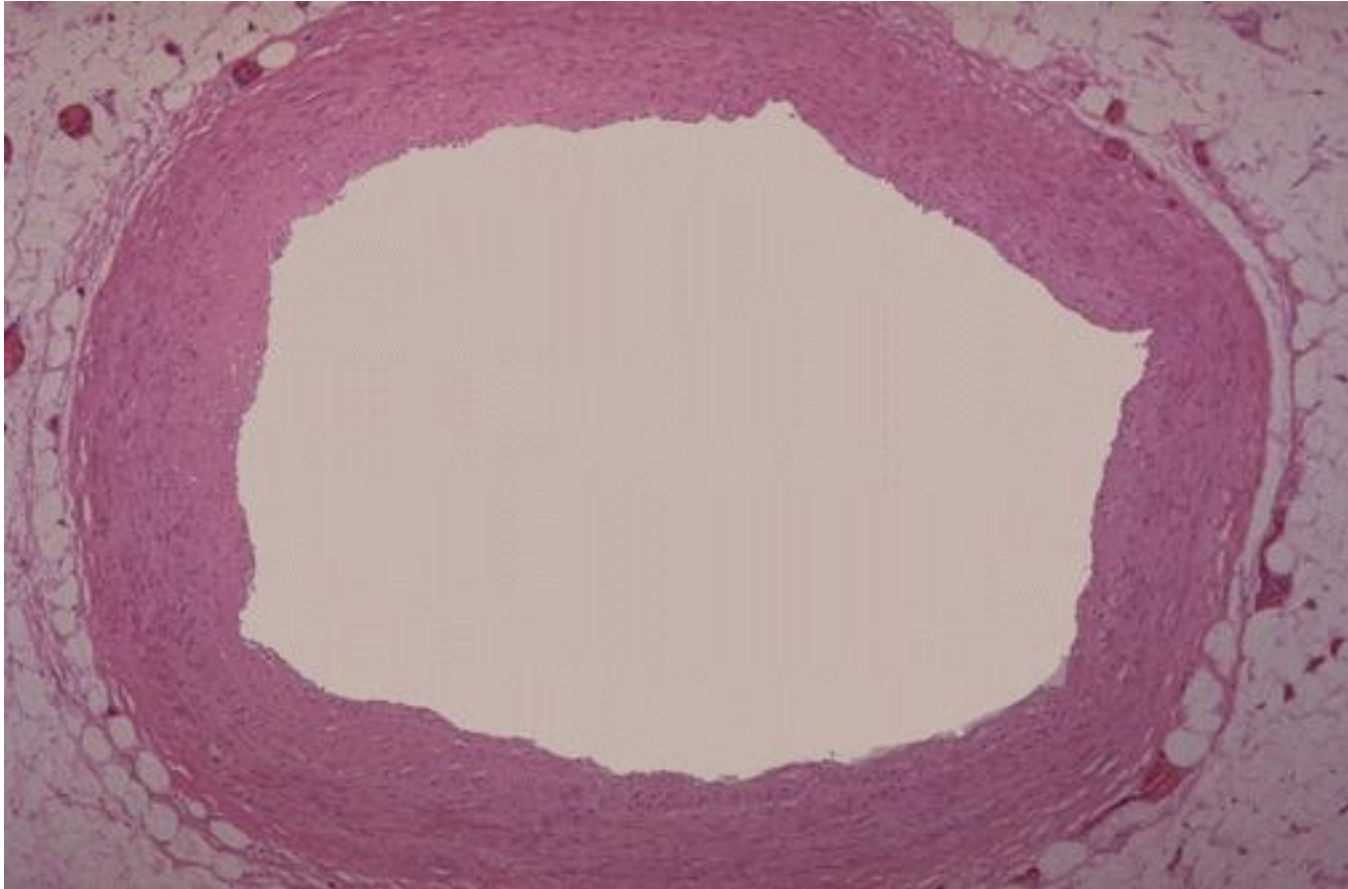




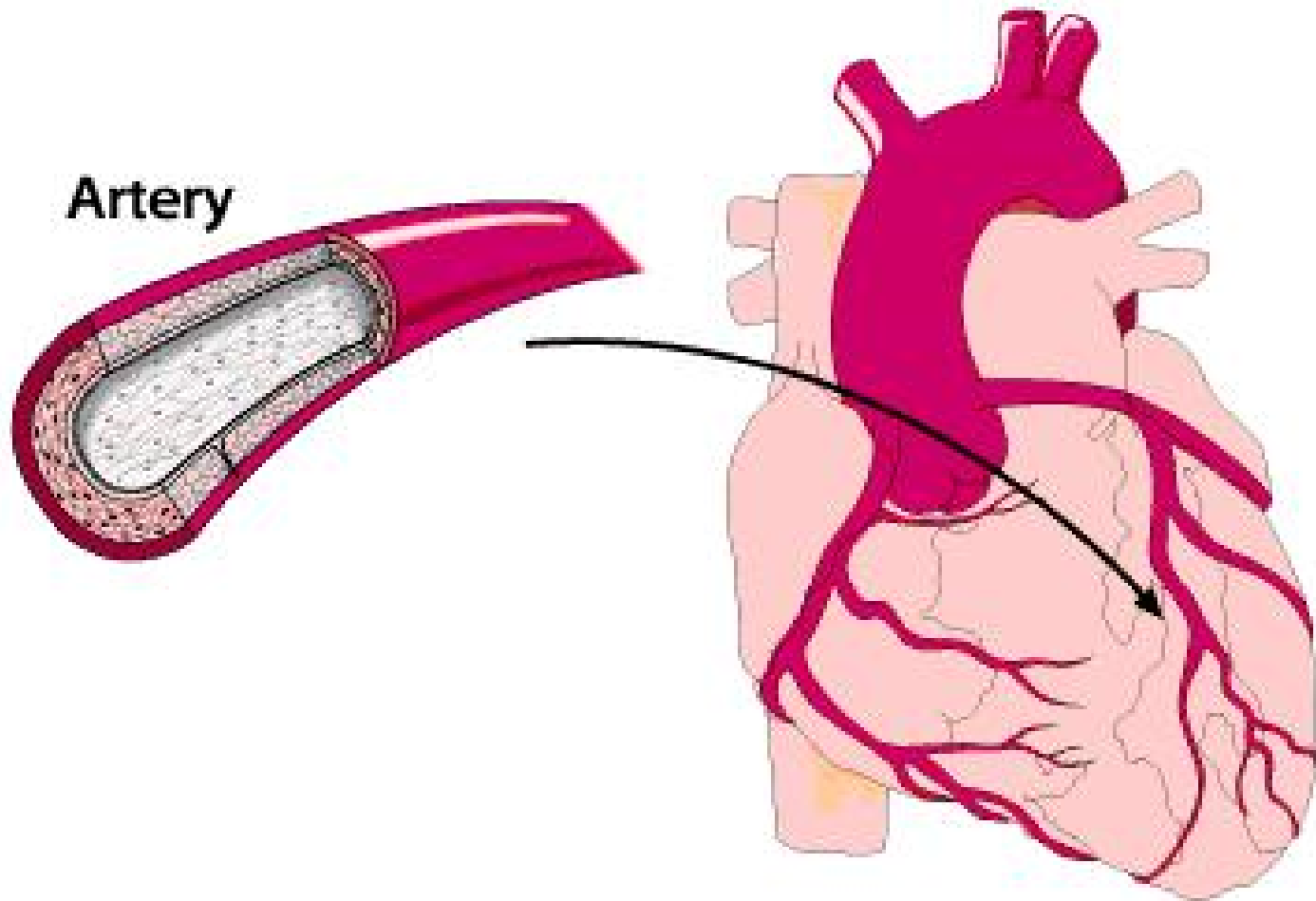
COMMON PRECIPITATING FACTORS IN ANGINA PECTORIS:  
HEAVY MEAL, EXERTION, COLD, SMOKING

CHARACTERISTIC DISTRIBUTION OF  
PAIN IN ANGINA PECTORIS

# Normal Coronary Artery



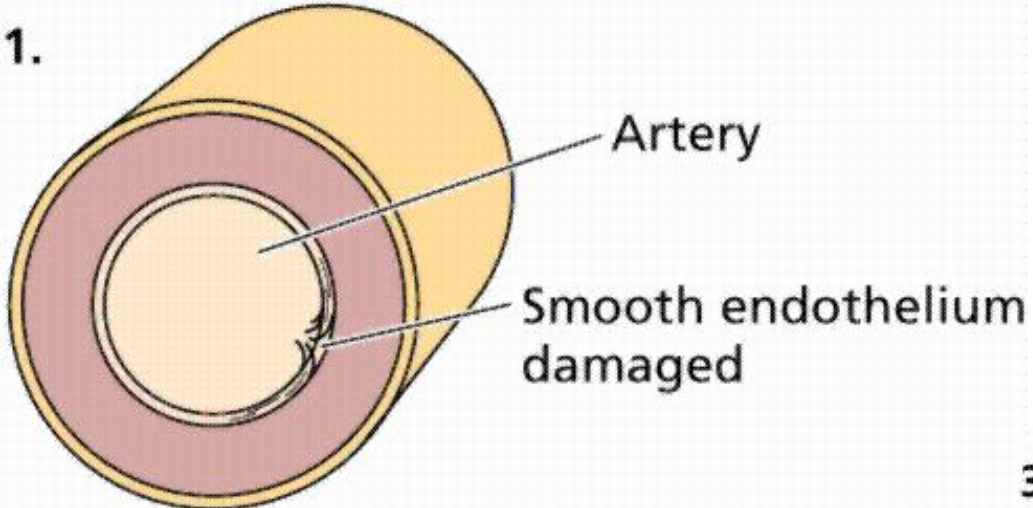
- This is a normal coronary artery with a nice, big, unobstructed lumen for supplying plenty of blood to the myocardium.



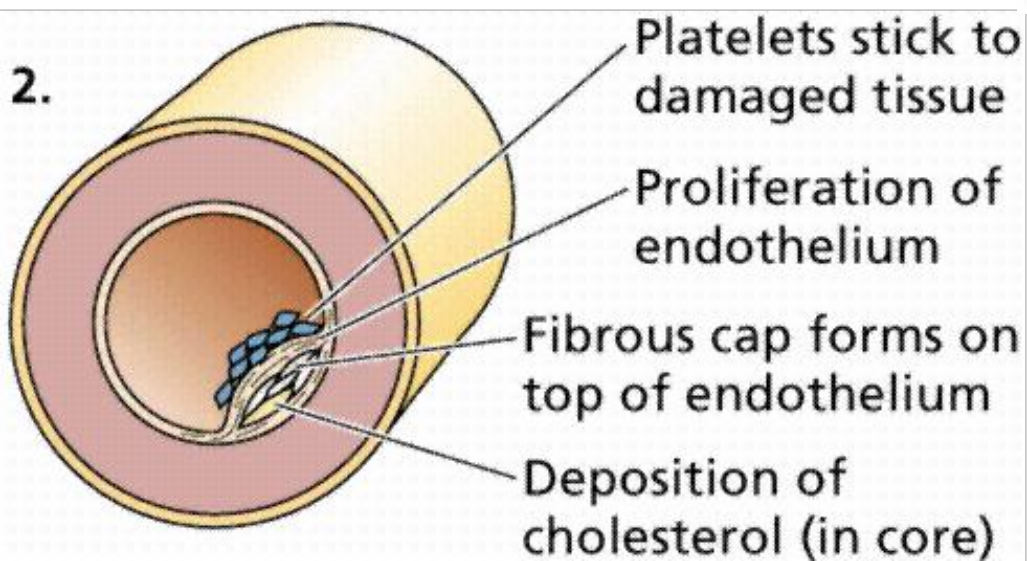
"Myocardial Infarction" (abbreviated as "MI") means there is death of some of the muscle cells of the heart as a result of a lack of supply of oxygen and other nutrients. This lack of supply is caused by closure of the artery ("coronary artery") that supplies that particular part of the heart muscle with blood. This occurs 98% of the time from the process of arteriosclerosis ("hardening of the arteries") in coronary vessels.



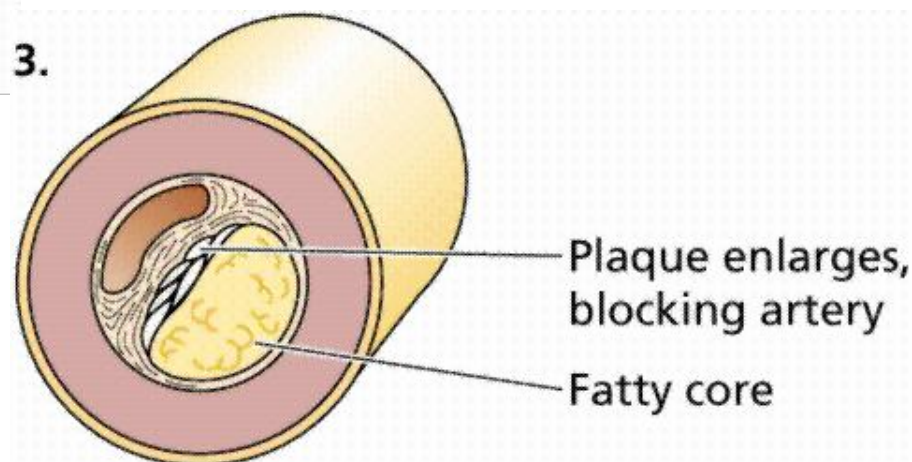
1.



2.



3.



# Serial Section of Coronary Artery



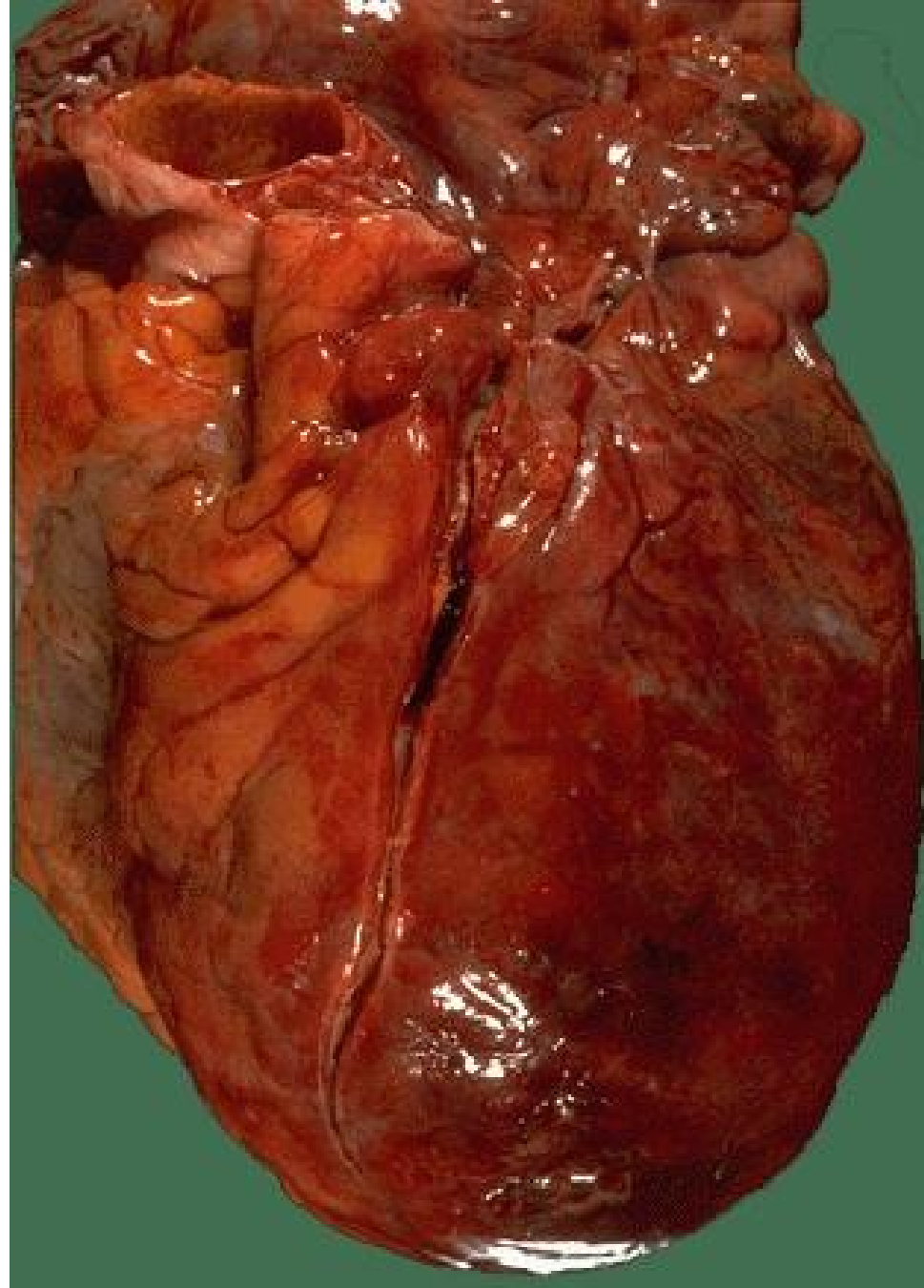
These serial sections of a coronary artery demonstrate grossly the appearance of luminal narrowing with atherosclerosis.

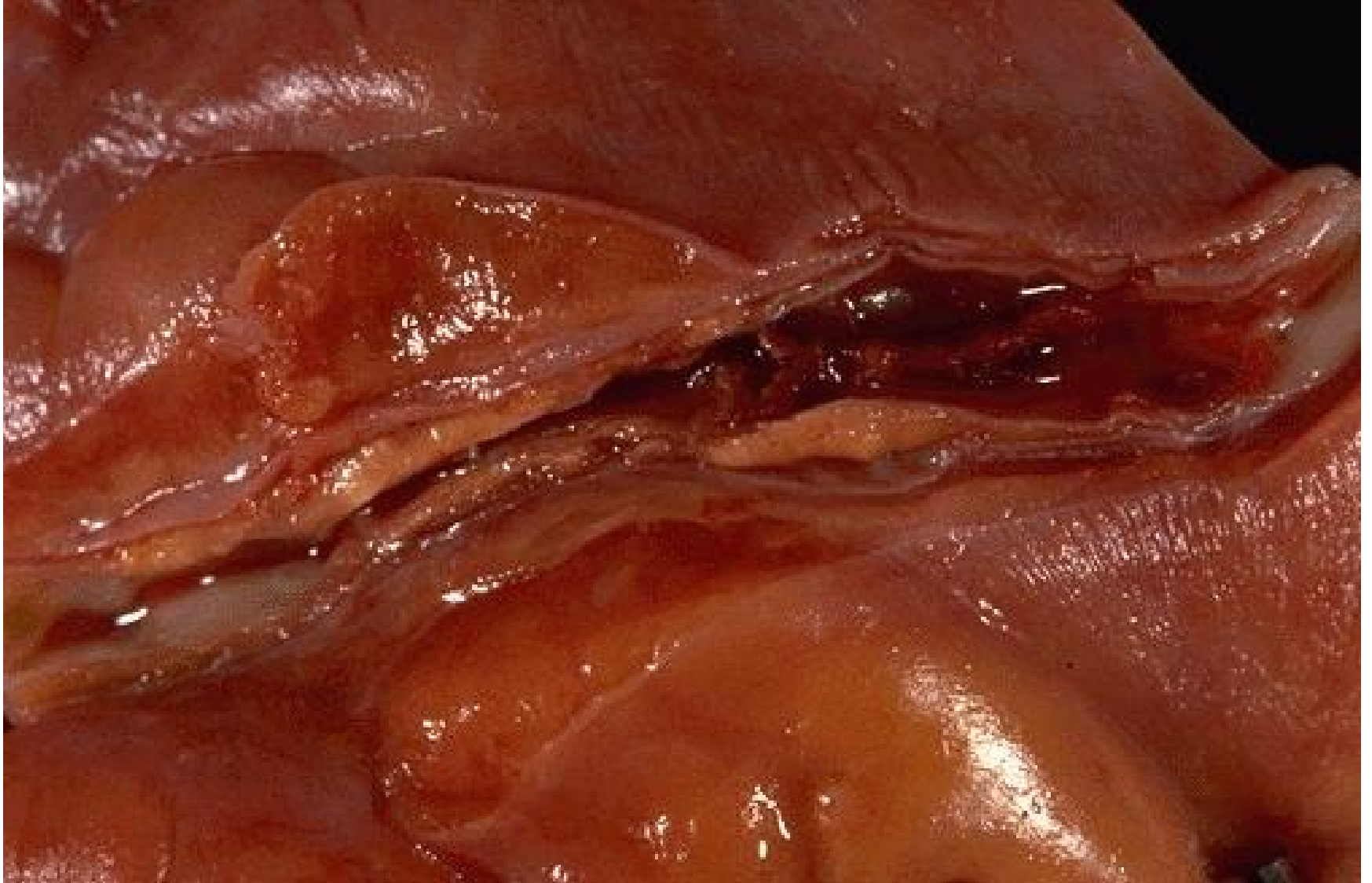


- This coronary artery opened longitudinally demonstrates severe atherosclerosis



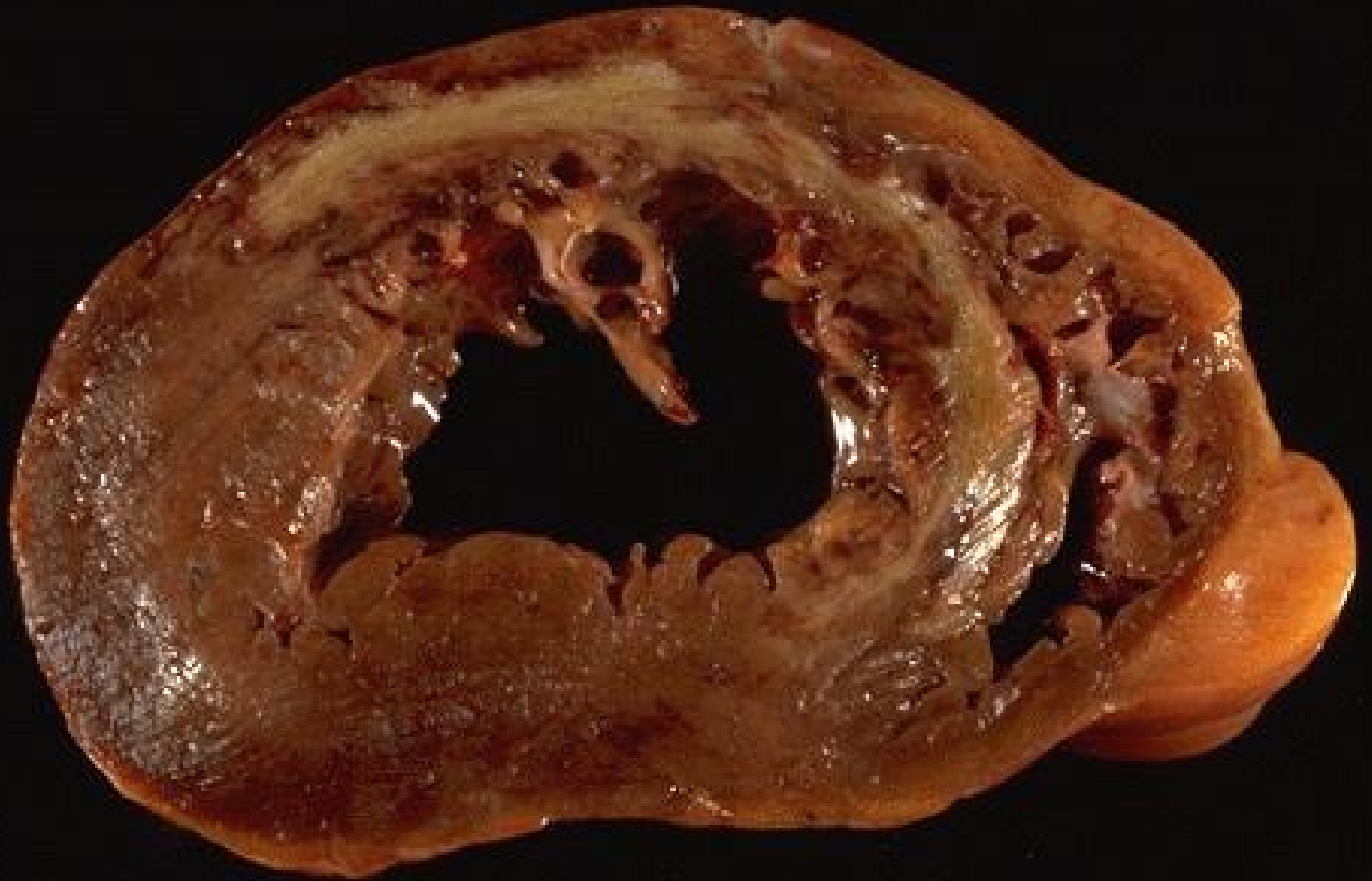
The anterior surface of the heart demonstrates an opened left anterior descending coronary artery. Within the lumen of the coronary can be seen a dark red recent coronary thrombosis. The dull red color to the myocardium as seen below the glistening epicardium to the lower right of the thrombus is consistent with underlying myocardial infarction.





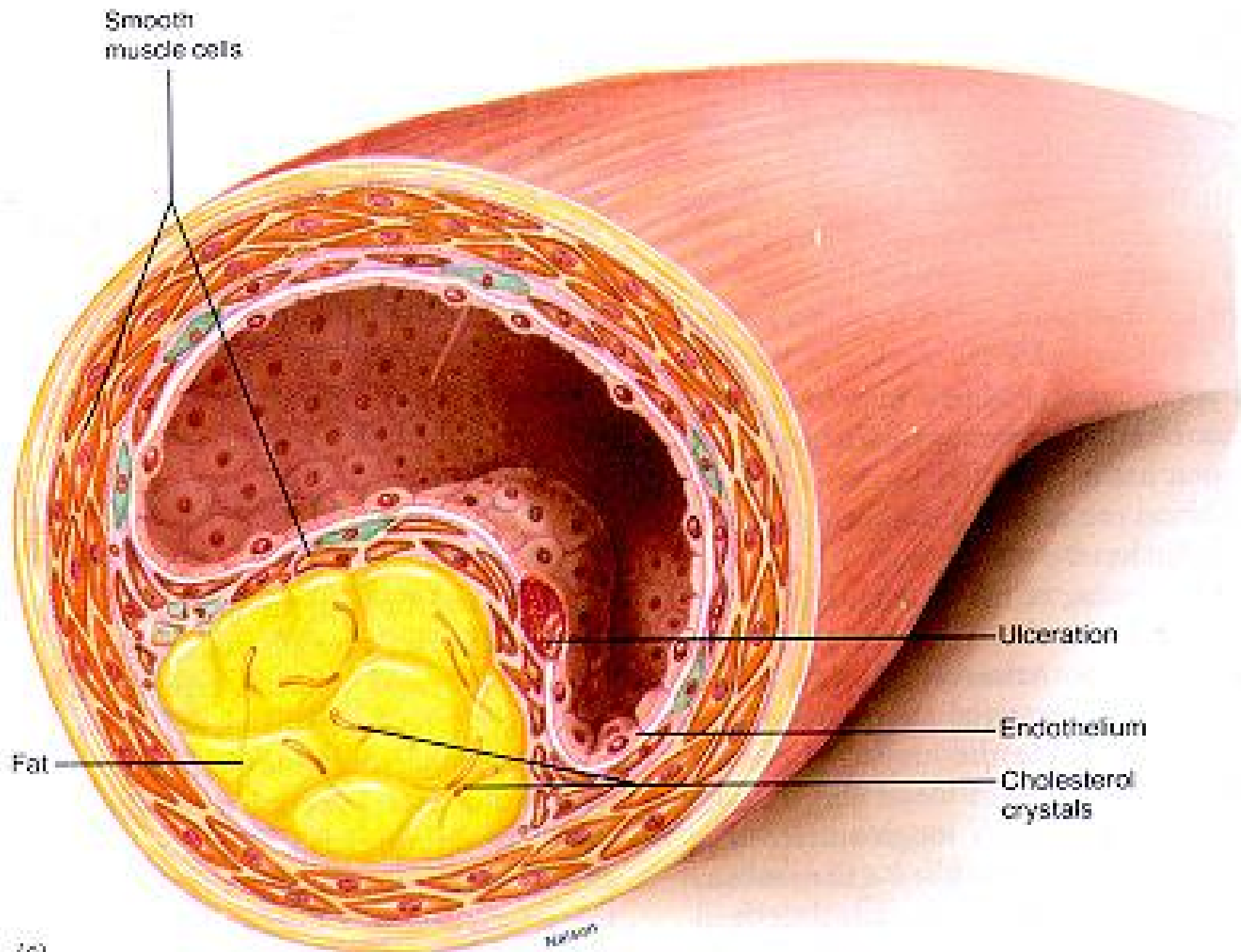
- This is thrombosis in a coronary artery. Such a thrombus severely narrows or occludes the lumen and can produce a sudden ischemic event. "Sudden death" as well as infarction can occur.

# Myocardial infarction of the left ventricular wall and septum



# Risk Factors in Heart Disease

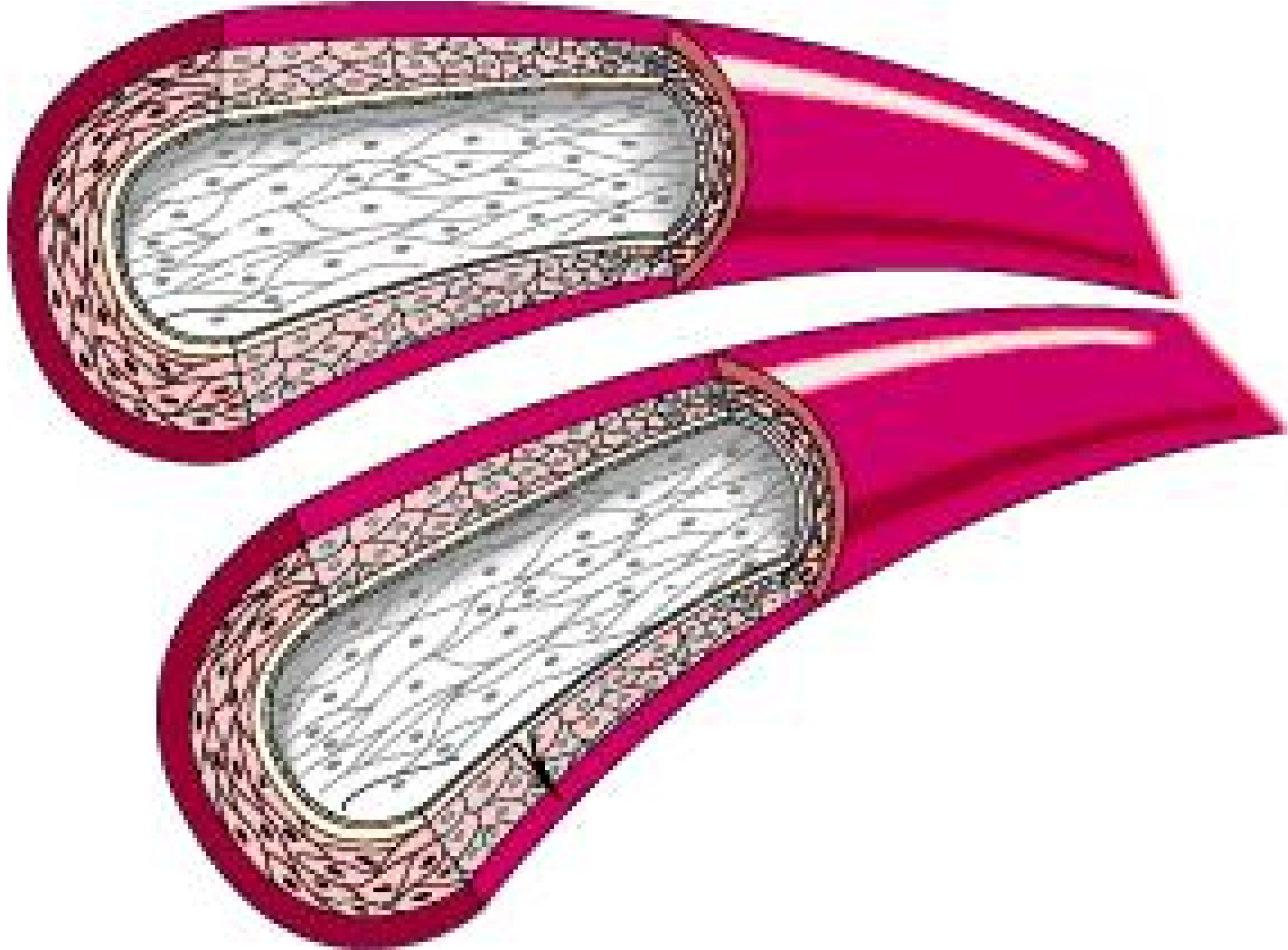
- Hypercholesterolemia
- Hypertension
- Cigarette smoking
- Obesity
- Lack of exercise
- Diabetes mellitus
- Genetic predisposition
- Male gender
- Alcohol



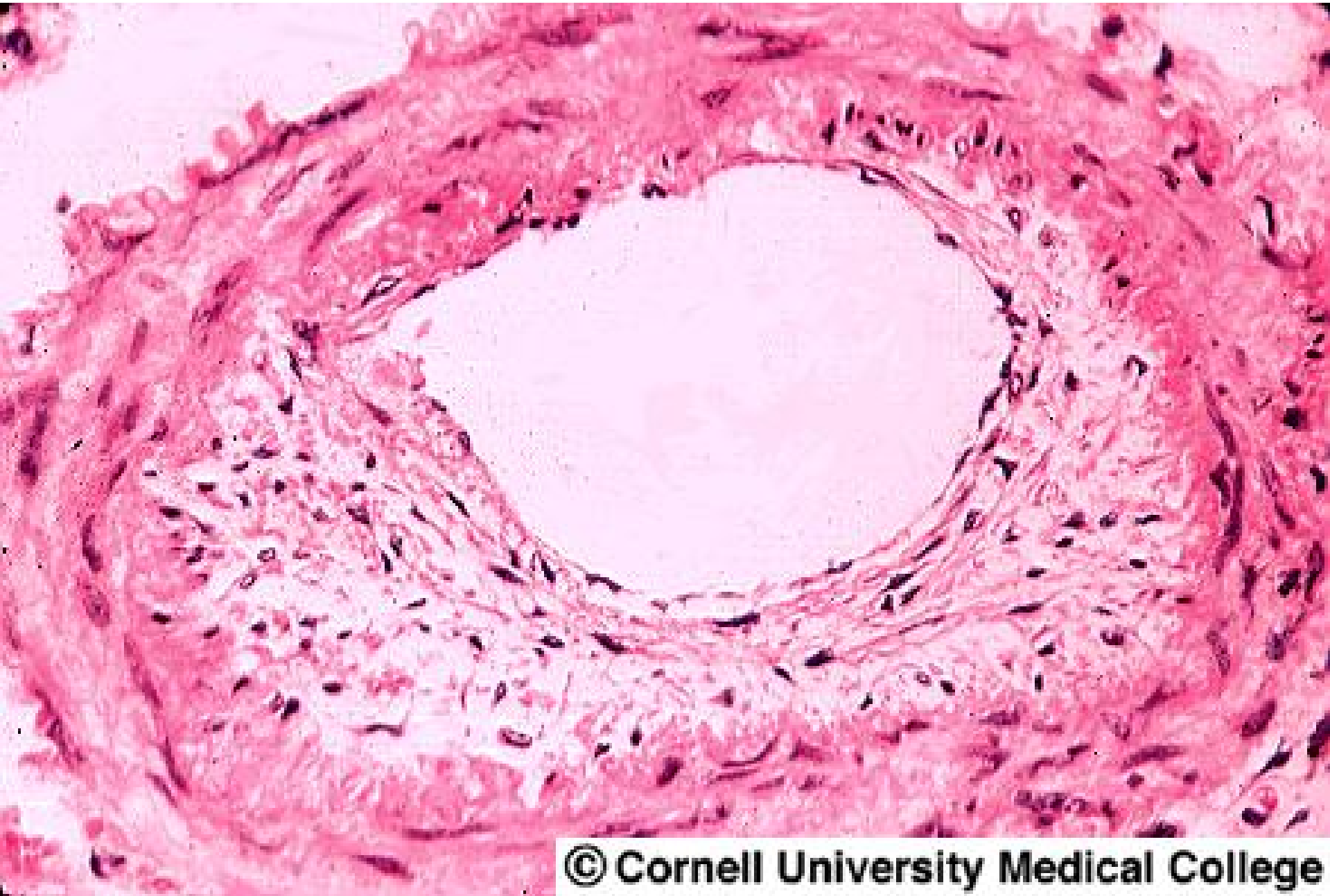
(c)



# Atherosclerosis and Blood Clotting

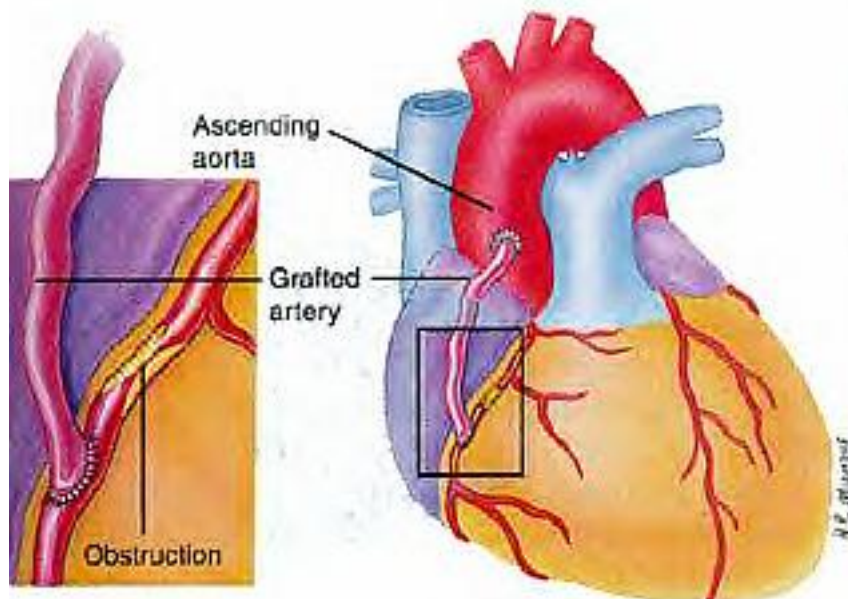


# Organized Thrombi

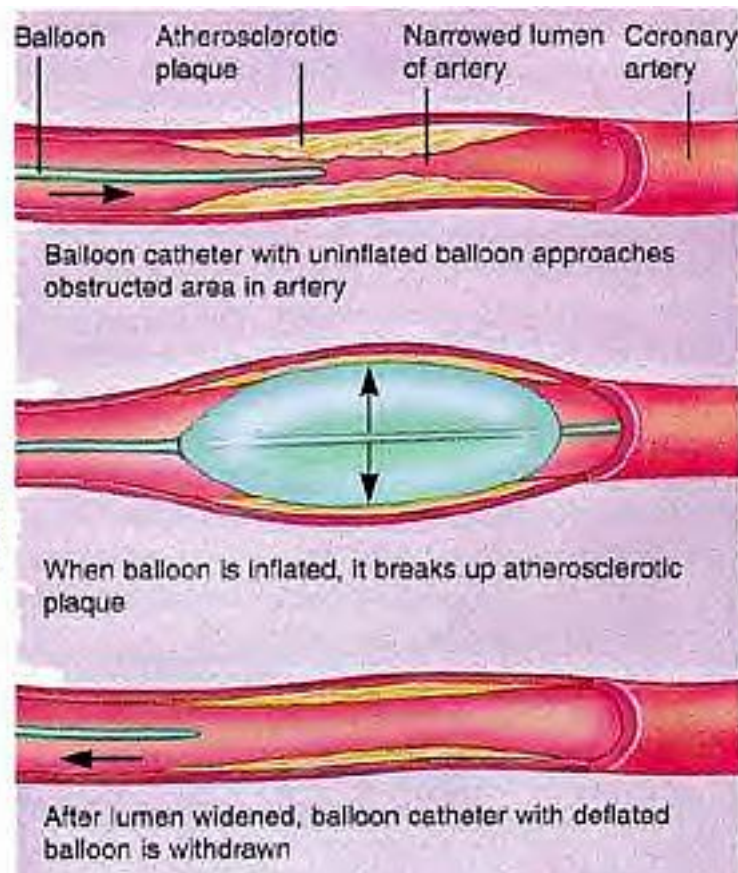




Martin M. Rotker/Science Source



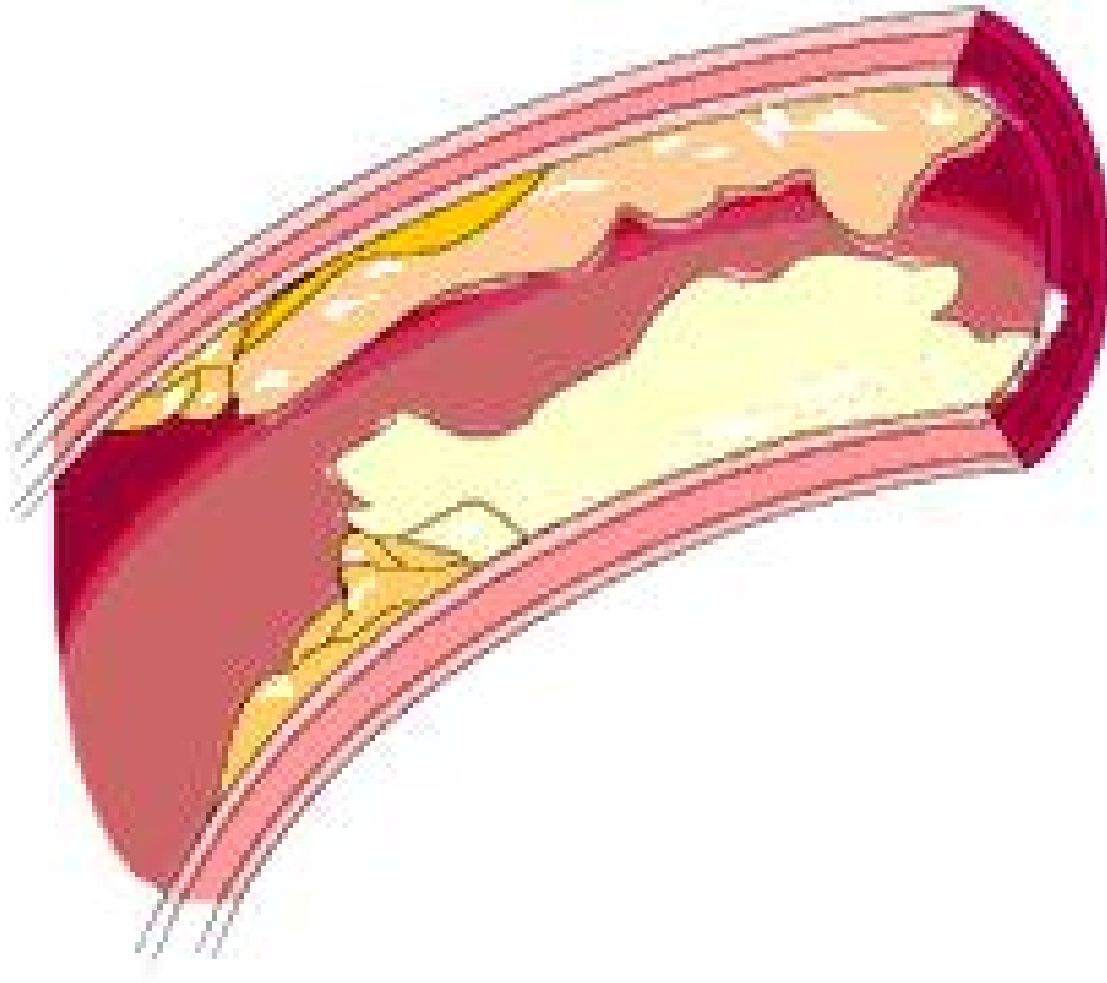
Coronary artery bypass grafting (CABG)

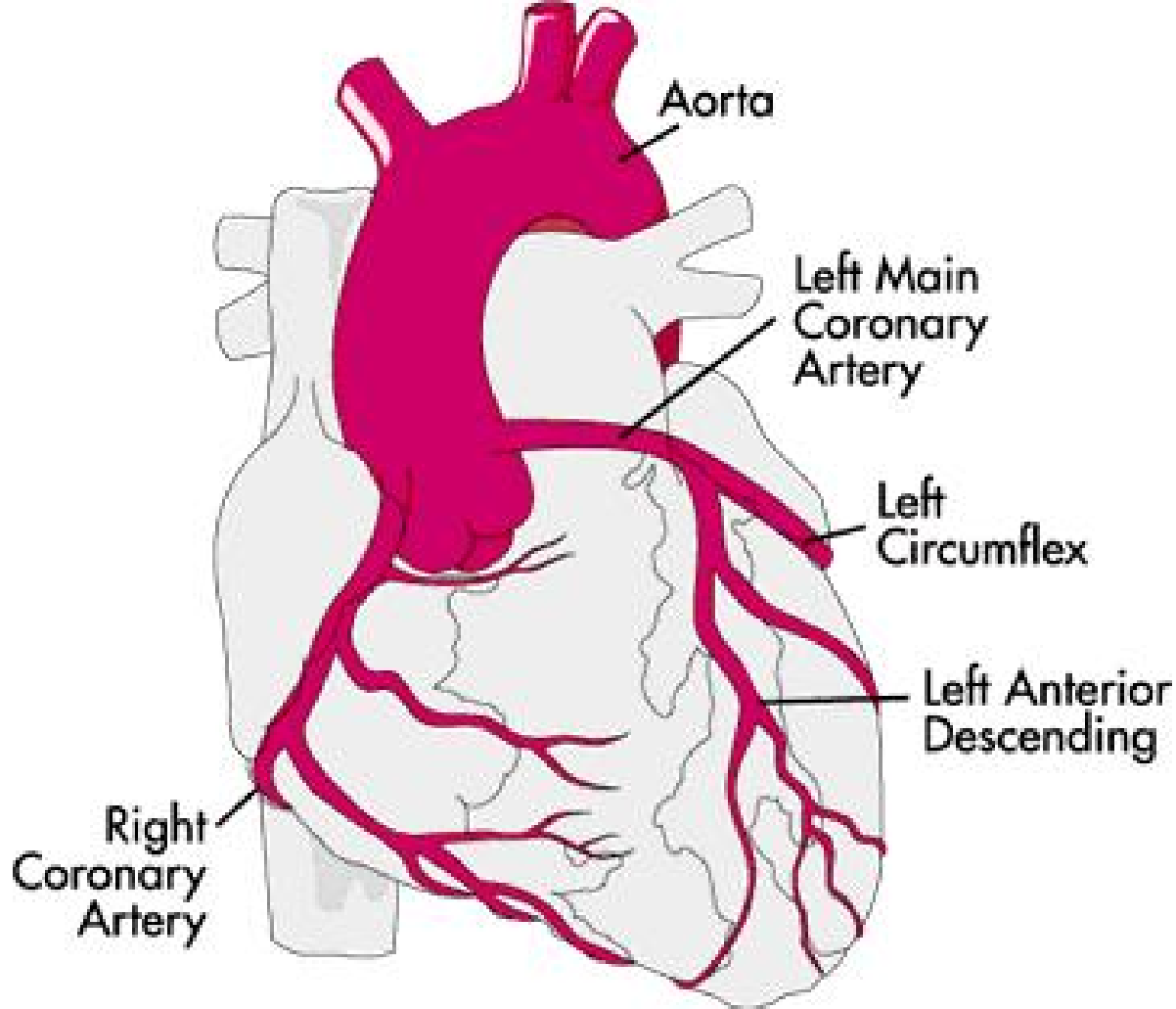


Balloon angioplasty (percutaneous transluminal coronary angioplasty or PTCA) is widely used for treatment of the blockages of coronary artery disease. This procedure is relatively simple, with only a small incision in the groin needed to introduce the equipment. As shown above, the balloon is inflated to compress the plaque and enlarge the artery, and provide an adequate area for blood to flow through. To learn more about PTCA and similar procedures, read on.

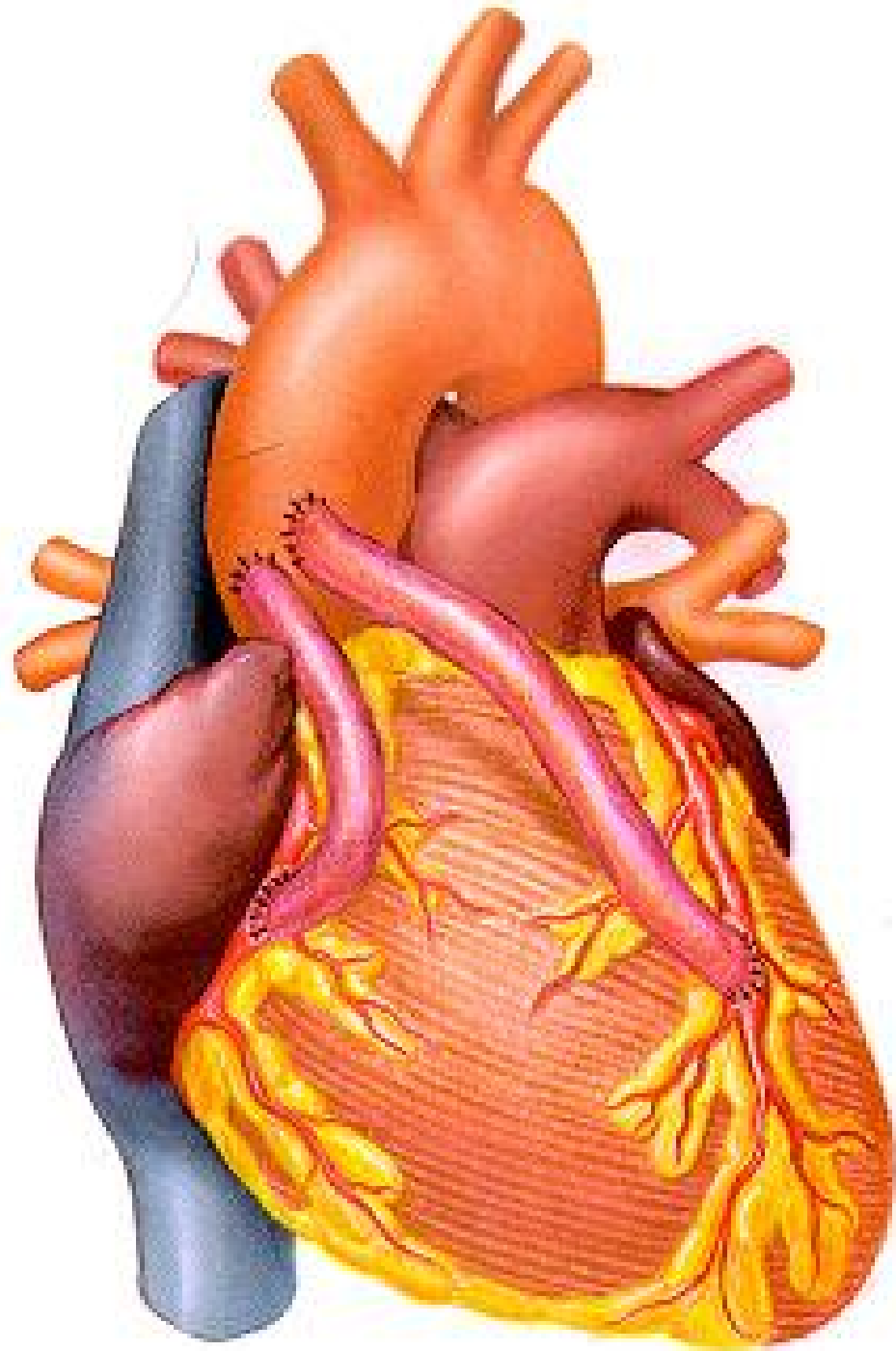


# Balloon Angioplasty

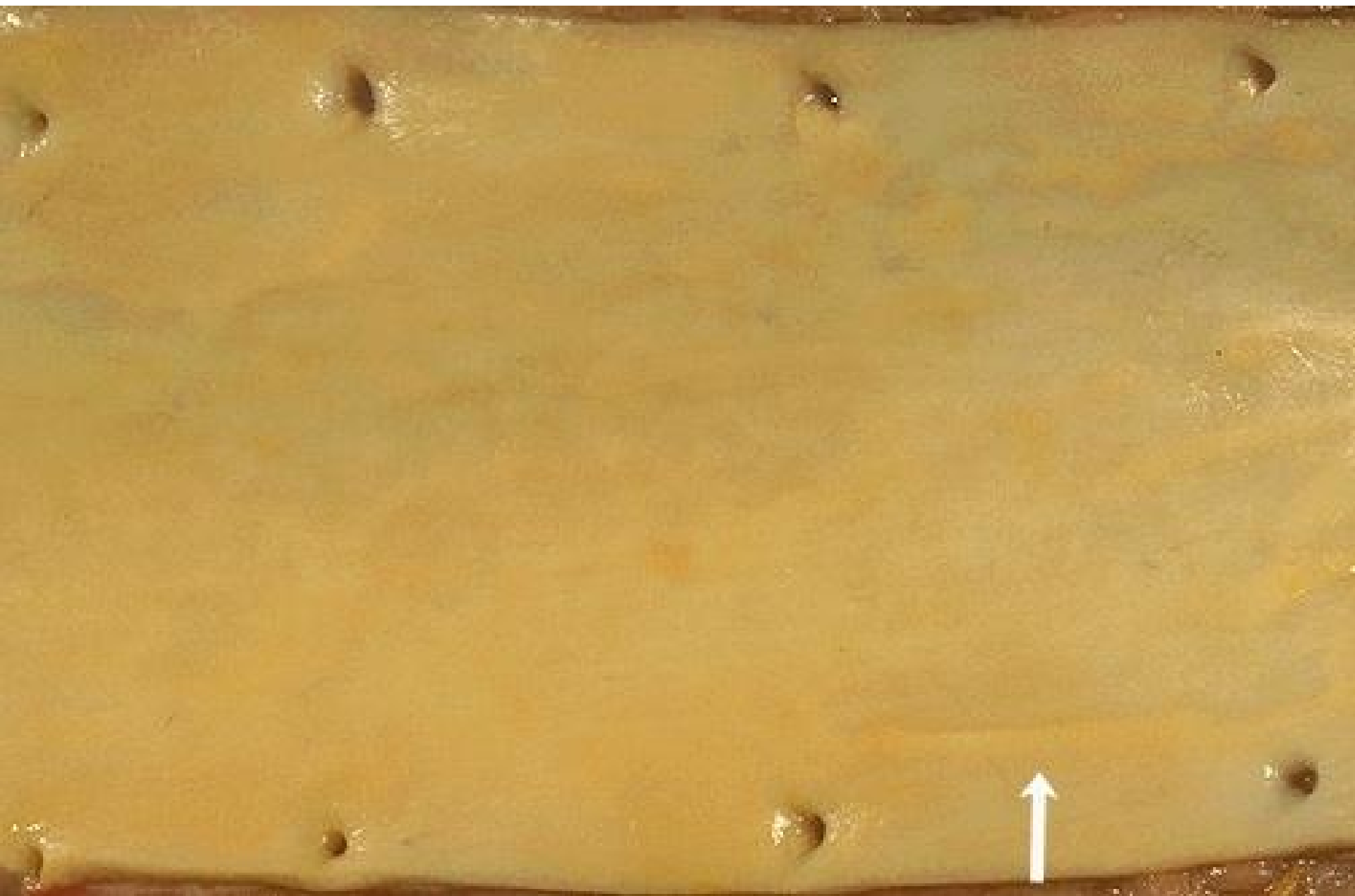




When the heart builds up blockages, it is often in more than one place. In this illustration, the patient has developed a 100% blockage of one artery (the Right Coronary Artery), while another vessel has several blockages including one at a branch point between the LAD and circumflex. Catheter techniques such as PTCA are of limited utility in both of these types of situations. In this illustration, a bypass surgery is being performed. Veins are taken from the leg, and sewn to the aorta, and then to the coronary arteries beyond the blockages.



## Fatty Streaks in Aorta



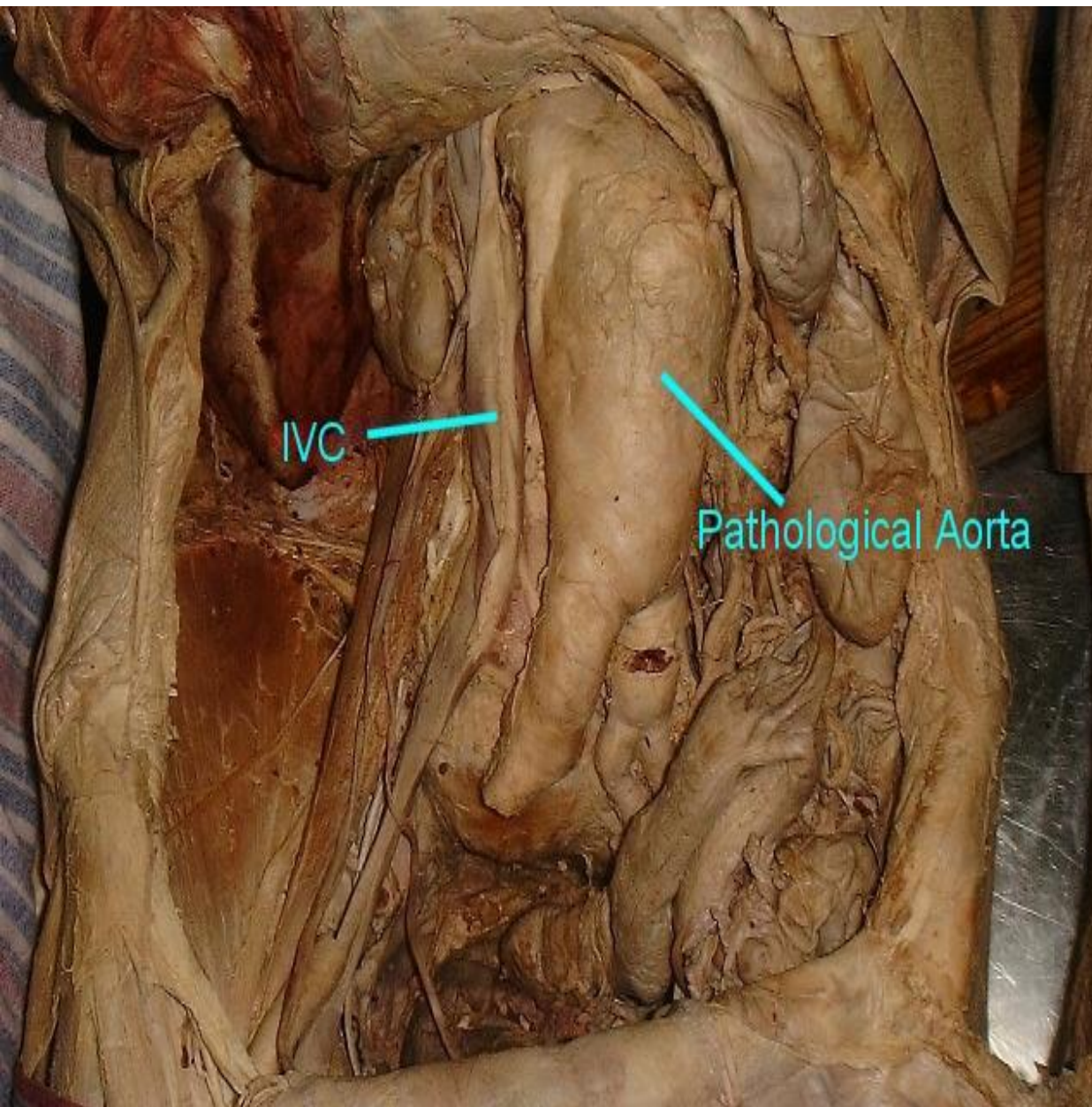


# Atherosclerosis, Mild, Moderate, and Severe



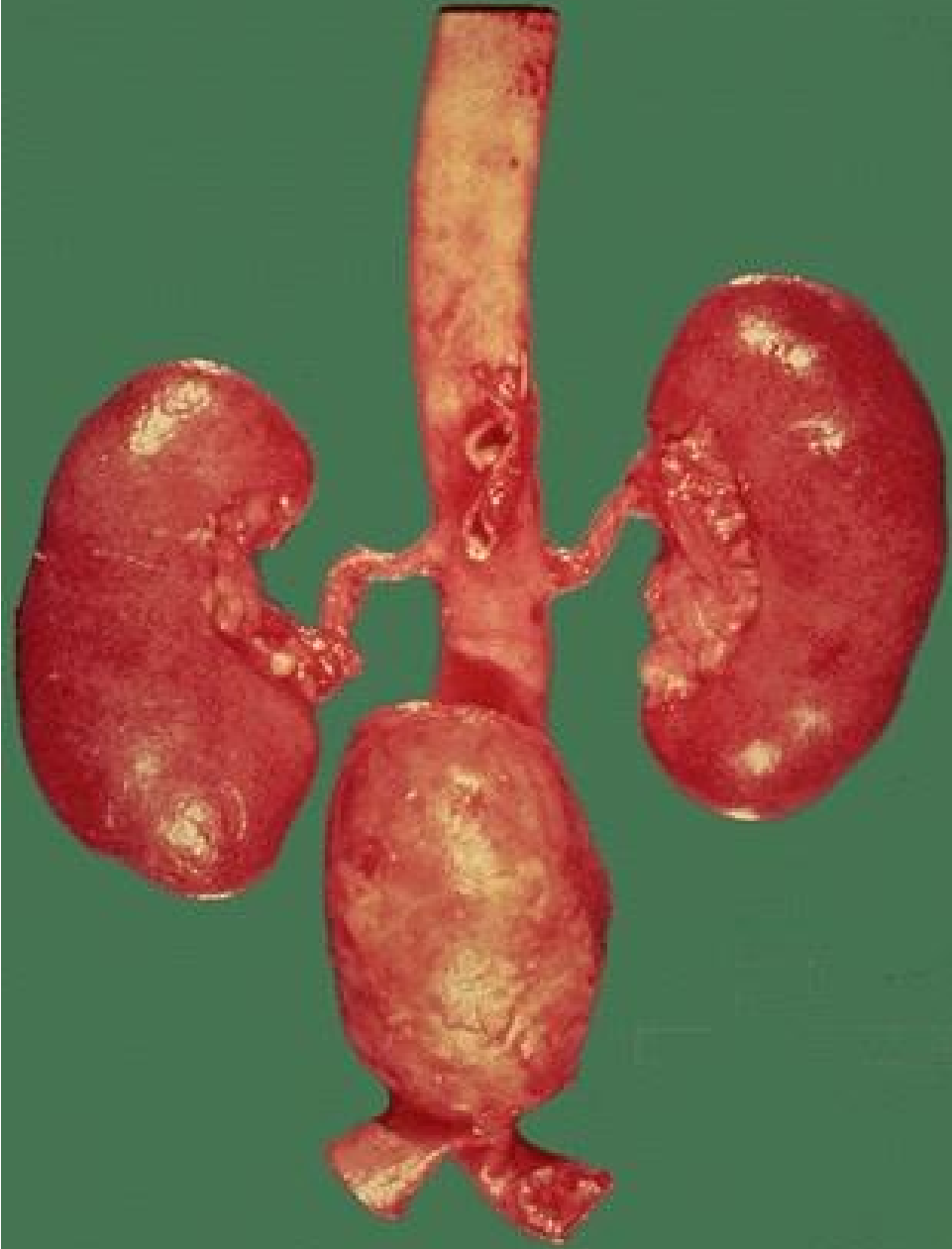
# Atherosclerosis of the aorta w/ mural thrombi





AAA stands for abdominal aortic aneurysm. An aneurysm is a permanent, localised dilatation of an artery and may involve several or all of the layers. There are various types and they are commonly found in the elderly population. They are thought to arise from a systemic collagen synthetic or structural defect such as atheromatous plaques. Ruptured AAA has a very high mortality rate - in excess of 80%. Yet, prior to rupture many are asymptomatic. If they are diagnosed before presentation with rupture they are treated dependent on size and rate of growth by surgical prosthetic grafting.

# Abdominal Aortic Aneurysm



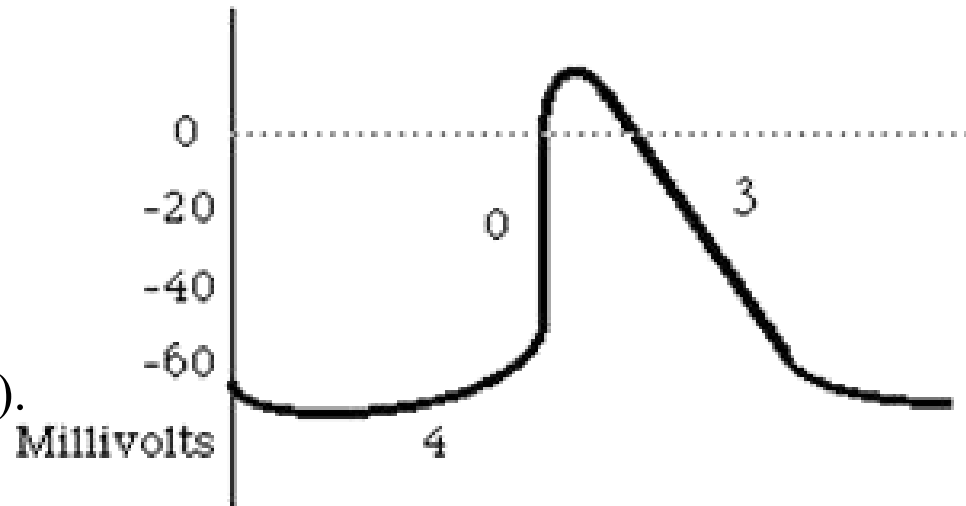




This aorta has been opened longitudinally to reveal an area of fairly limited dissection that is organizing. The red-brown thrombus can be seen in on both sides of the section as it extends around the aorta. The intimal tear would have been at the left. This creates a "double lumen" to the aorta. This aorta shows severe atherosclerosis which, along with cystic medial necrosis and hypertension, is a risk factor for dissection.

# In cardiac muscle, there are two types of cells:

- Contractile cells
- Autorhythmic (or automatic) cells.
- **Autorhythmic cells** exhibit **PACEMAKER POTENTIALS**.
  - **Depolarization is due to the inward diffusion of calcium** (not sodium as in nerve cell membranes).
  - Depolarization begins when:
    - the slow calcium channels open (4),
    - then concludes (quickly) when the fast calcium channels open (0).
    - Repolarization is due to the outward diffusion of potassium (3).



# Myocyte



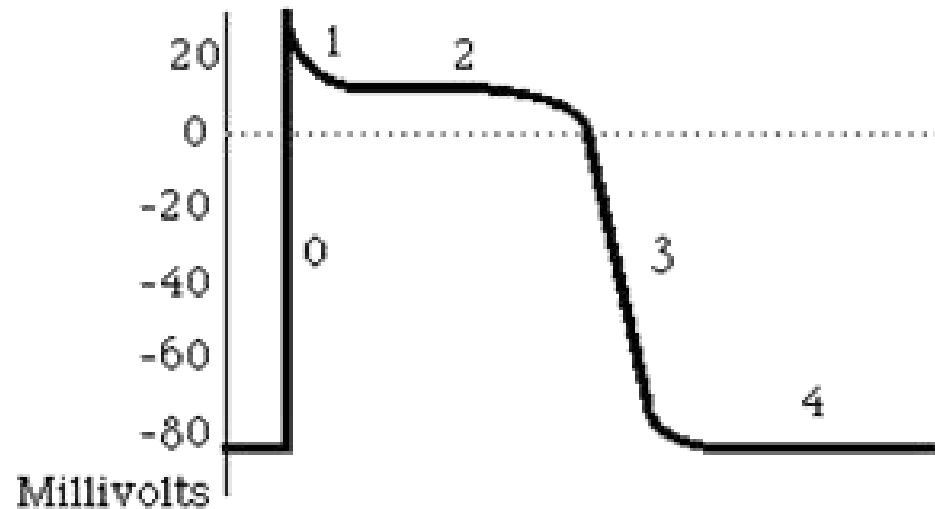
**Autorhythmic cells**



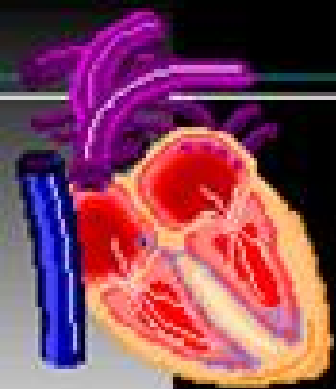
**Sheet of cells**

## In Contractile cells:

- **depolarization** is very rapid & is due to the **inward diffusion of sodium** (0).
- **repolarization** begins with a slow outward diffusion of potassium, but that is largely offset by the slow inward diffusion of calcium (1 & 2). So, repolarization begins with a plateau phase. Then, potassium diffuses out much more rapidly as the calcium channels close (3), and the membrane potential quickly reaches the 'resting' potential (4).



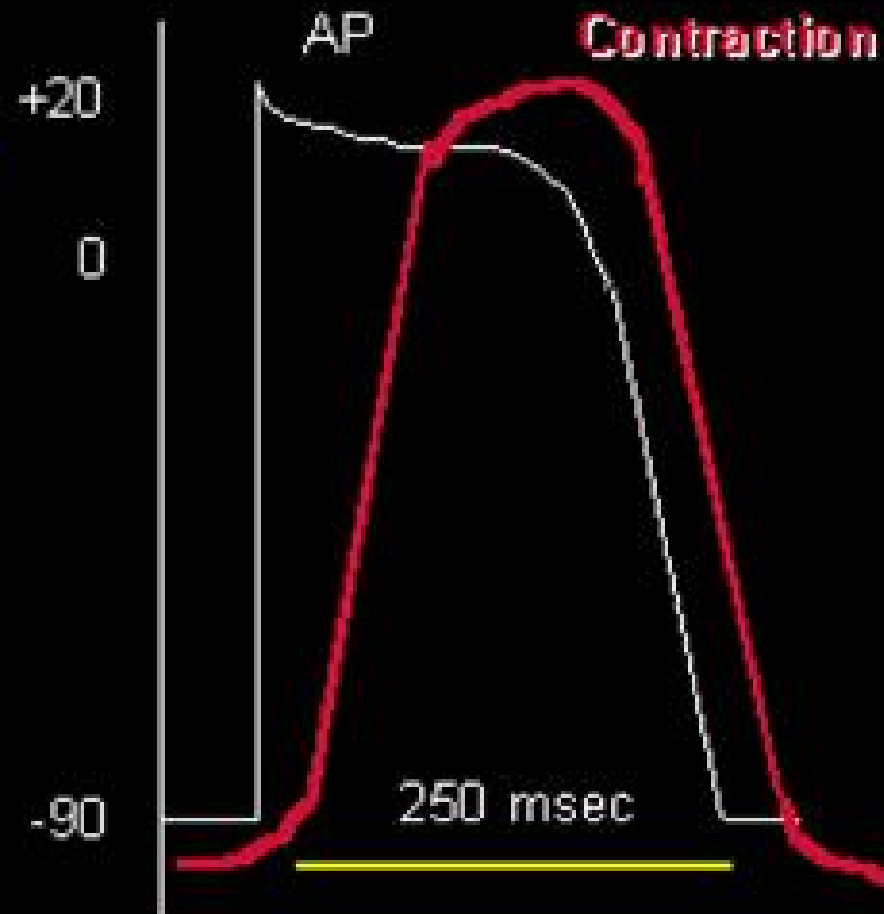




# Refractory Period

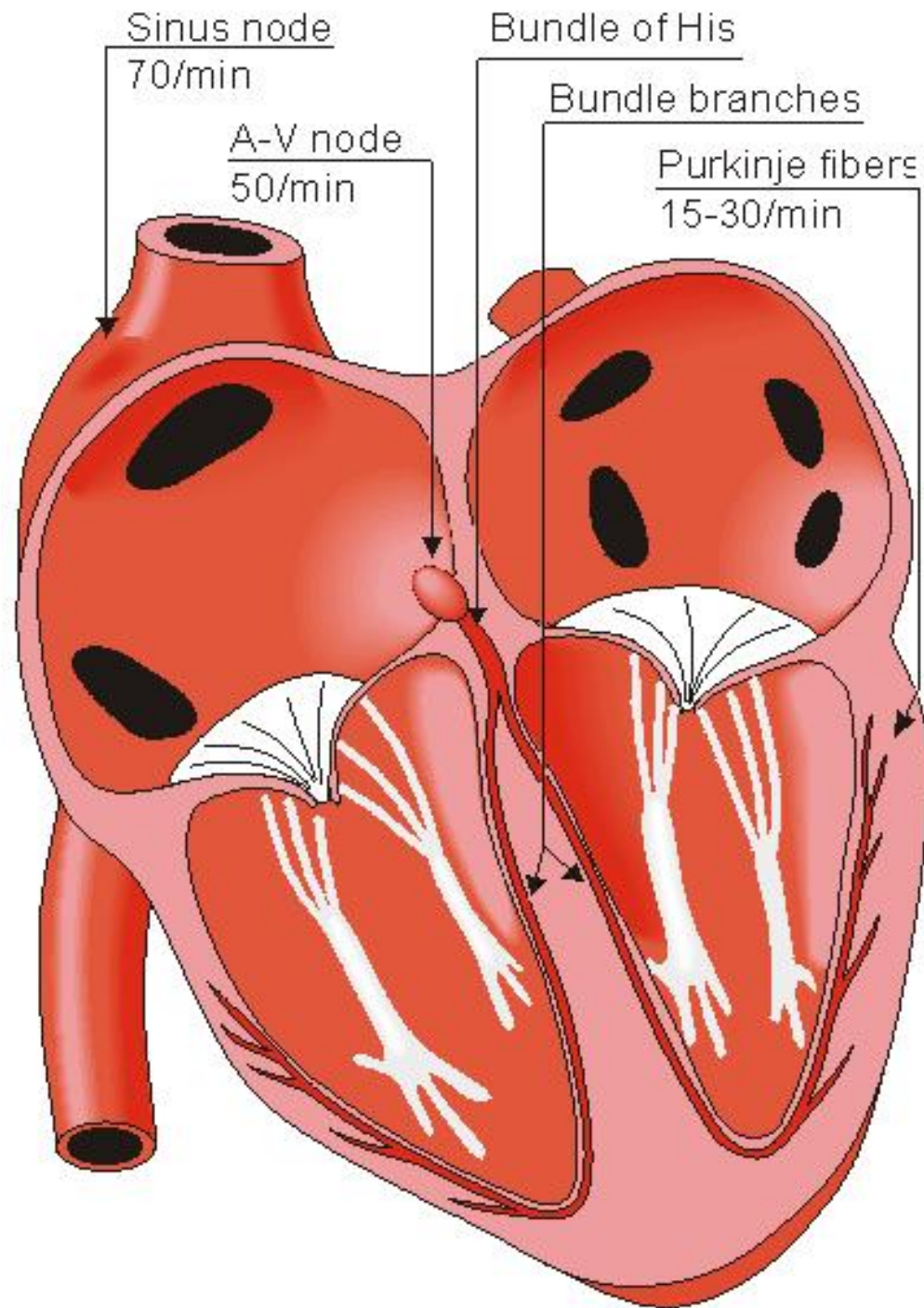
- **LONG** (*250 msec*)
- membrane is refractory to further stimulation until the contraction is over

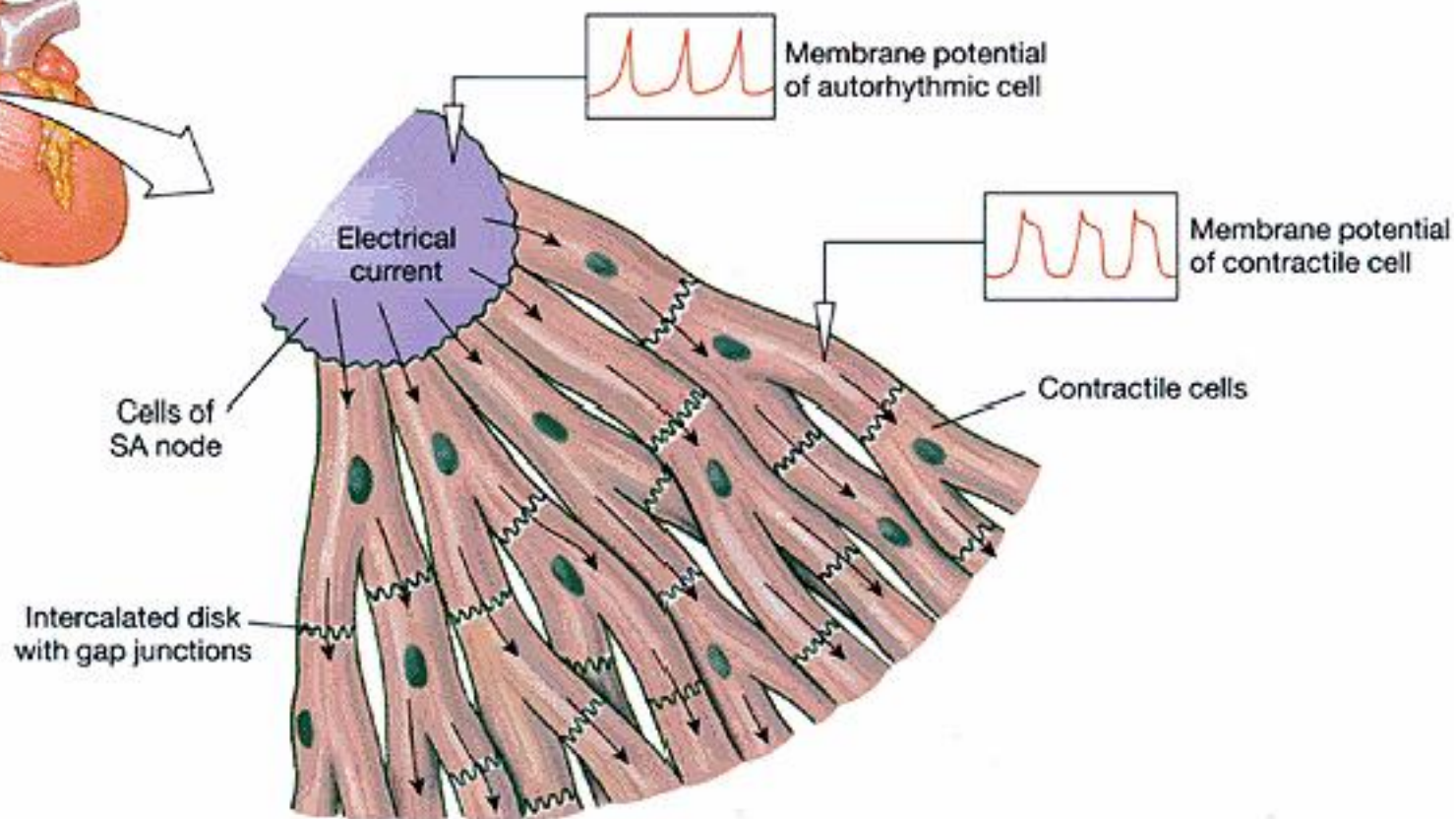
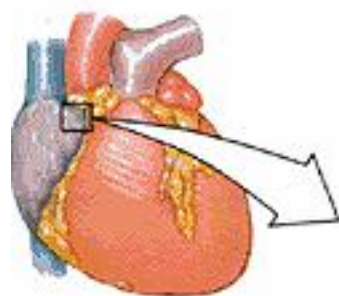
➡ **NO SUMMATION OR TETANY POSSIBLE!**



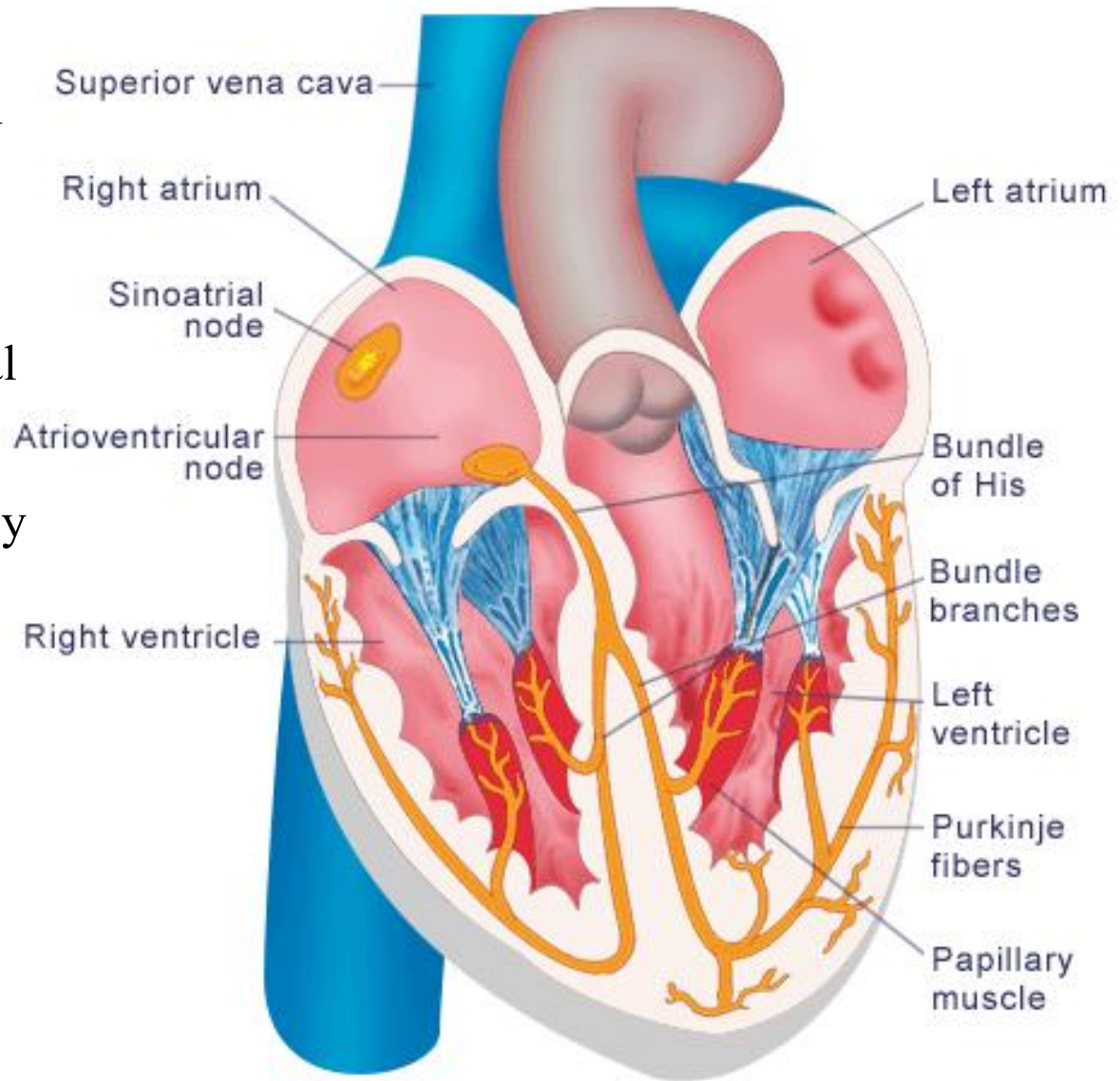
**Most of the muscle cells in the heart are contractile cells. The autorhythmic cells are located in these areas:**

- **Sinoatrial (SA), or sinus, node** - 60 - 100 per minute (usually 70 - 80 per minute)
  - **Atrioventricular (AV) node** - 40 - 60 per minute
  - Atrioventricular (AV) bundle (also sometimes called the bundle of His)
  - Right & left bundle branches
  - Purkinje fibers
- 20 - 40 per minute

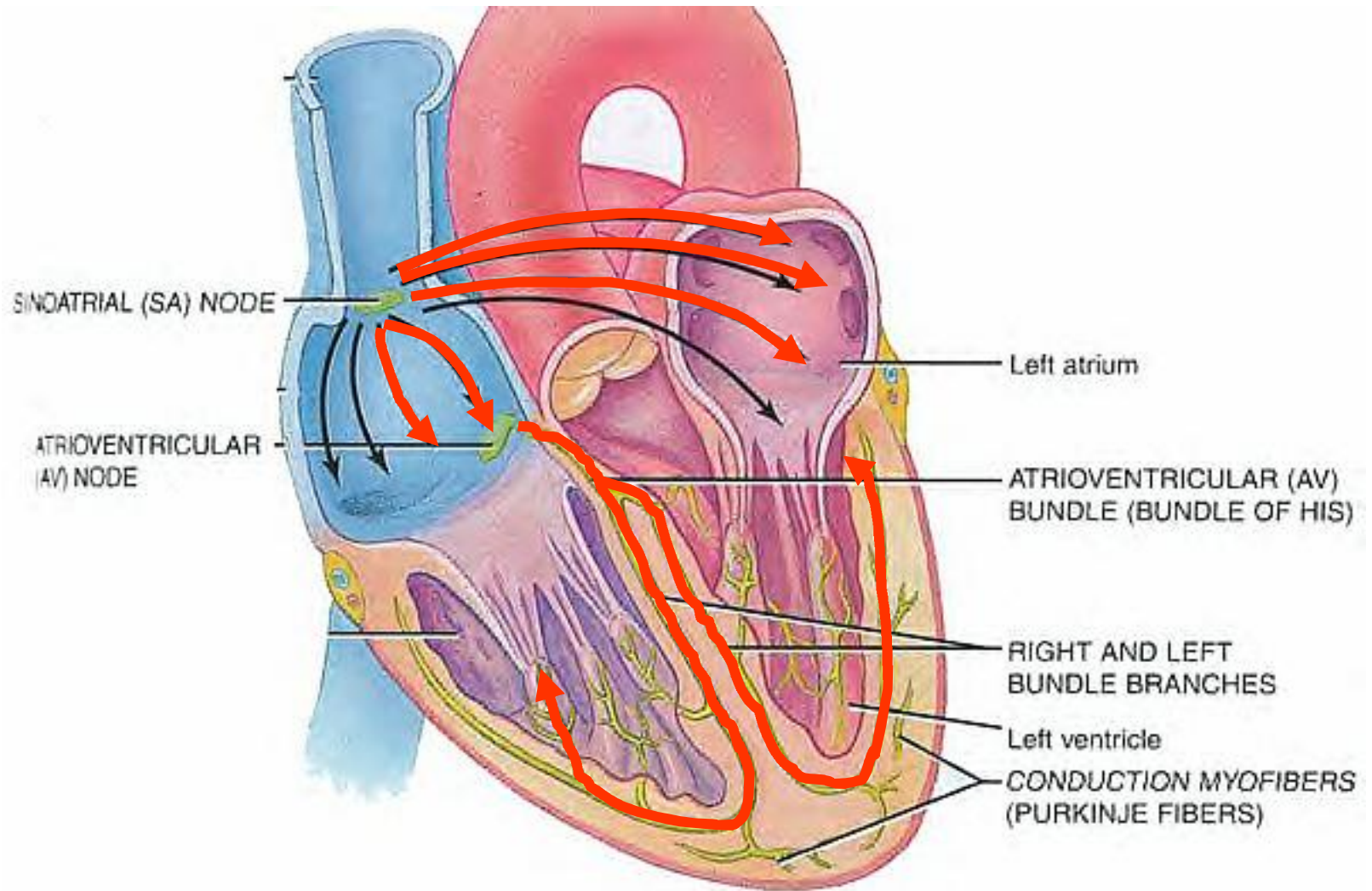


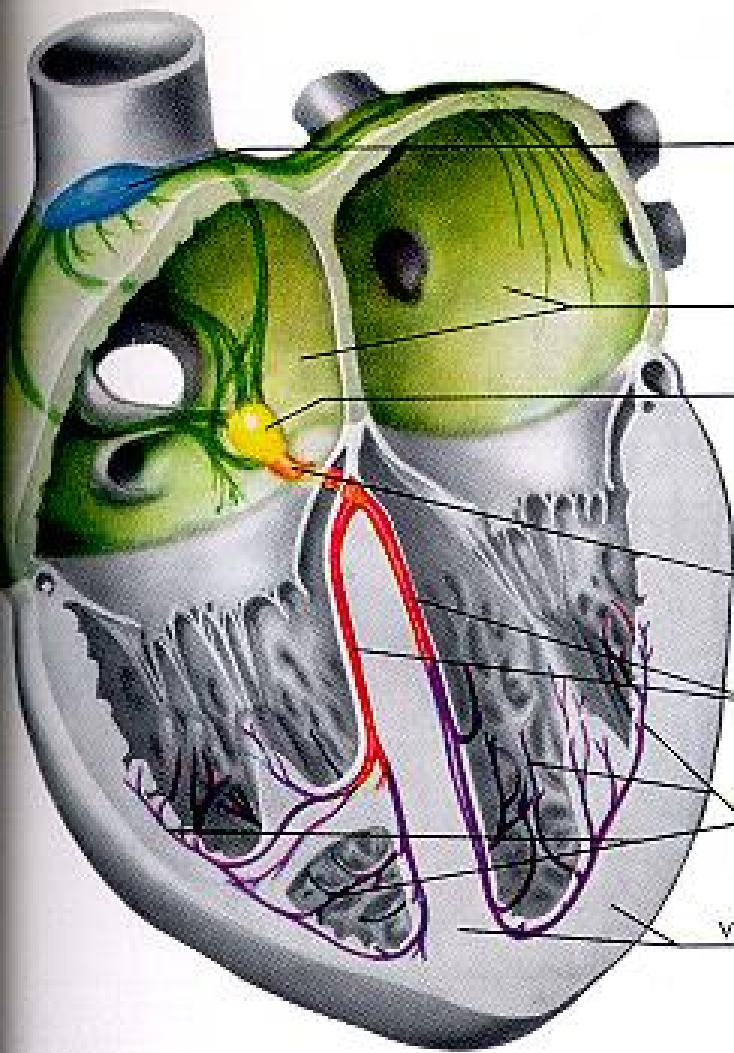


- **Sinoatrial node-** a patch of modified myocytes in the right atrium that is the pacemaker that initiates each heartbeat and determines the heart rate.
- **Atrioventricular node-** located near the right AV valve and acts as an electrical gateway to the ventricles.
- **atrioventricular bundle** (bundle of His)- a pathway by which signals leave the AV node
- **Right and left bundle branches-** divisions of the AV bundle that enter the interventricular septum and descend toward the apex.
- **Purkinje fibers-** nerverlike processes that arise from the bundle of branches that distribute the electrical excitation to the myocytes of the ventricles









S-A NODE

ATRIAL MUSCLE

A-V NODE

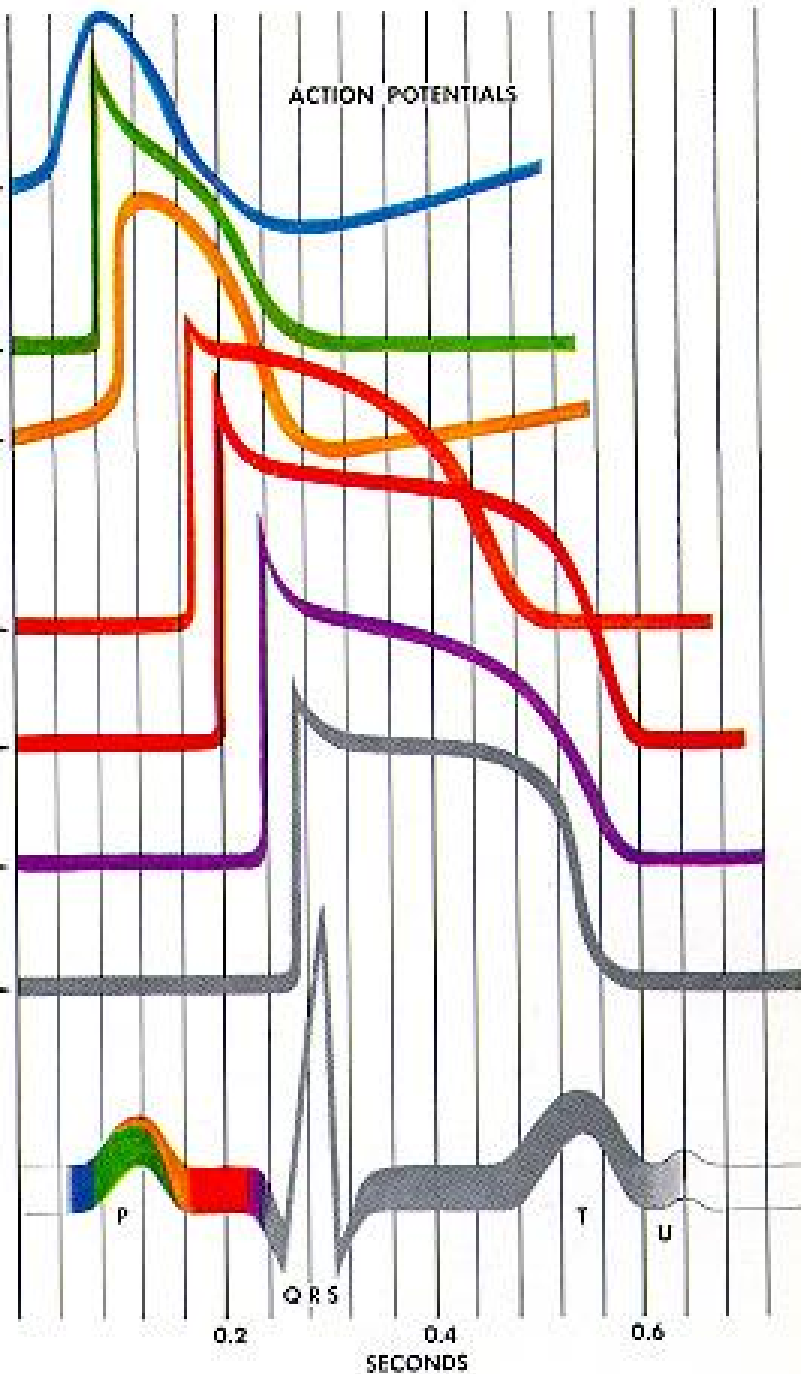
COMMON BUNDLE

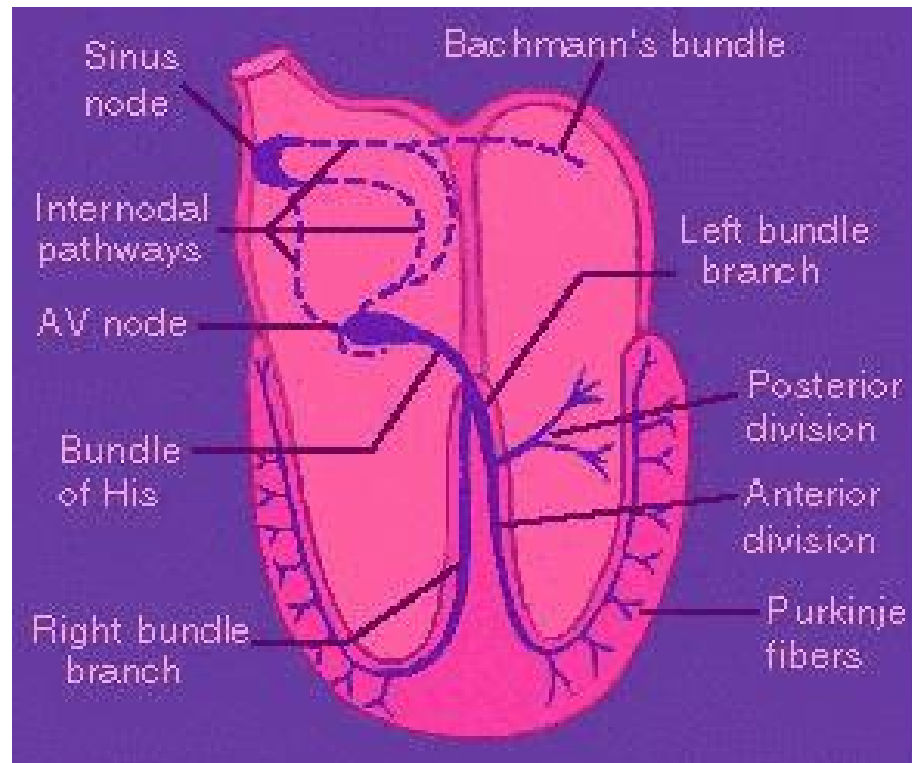
BUNDLE BRANCHES

PURKINJE FIBERS

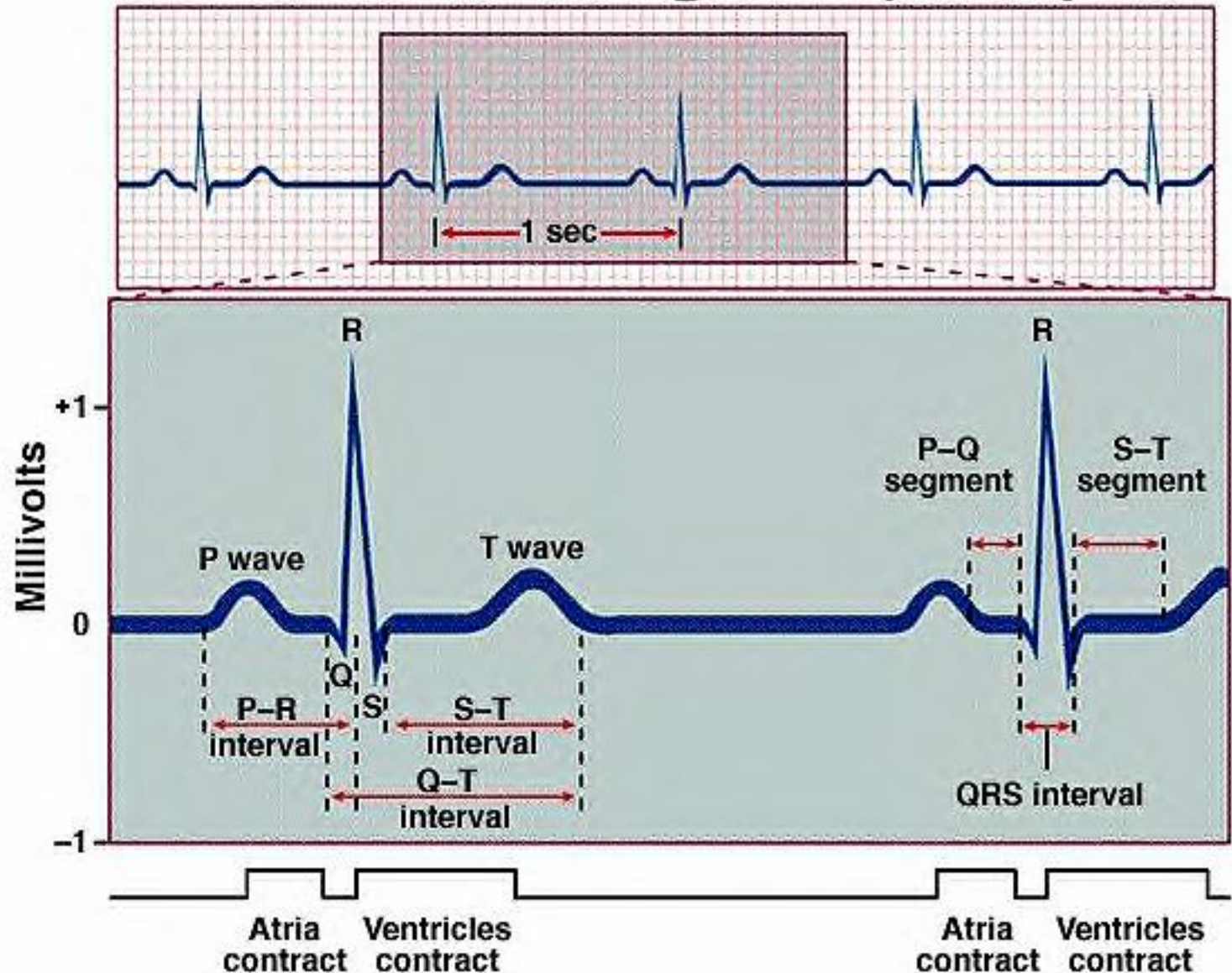
VENTRICULAR MUSCLE

ACTION POTENTIALS





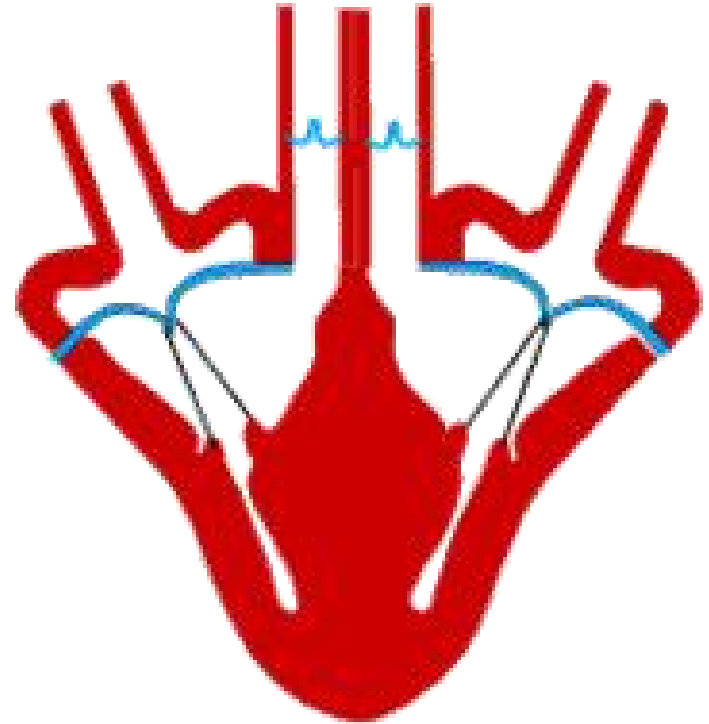
# Electrocardiogram (ECG)

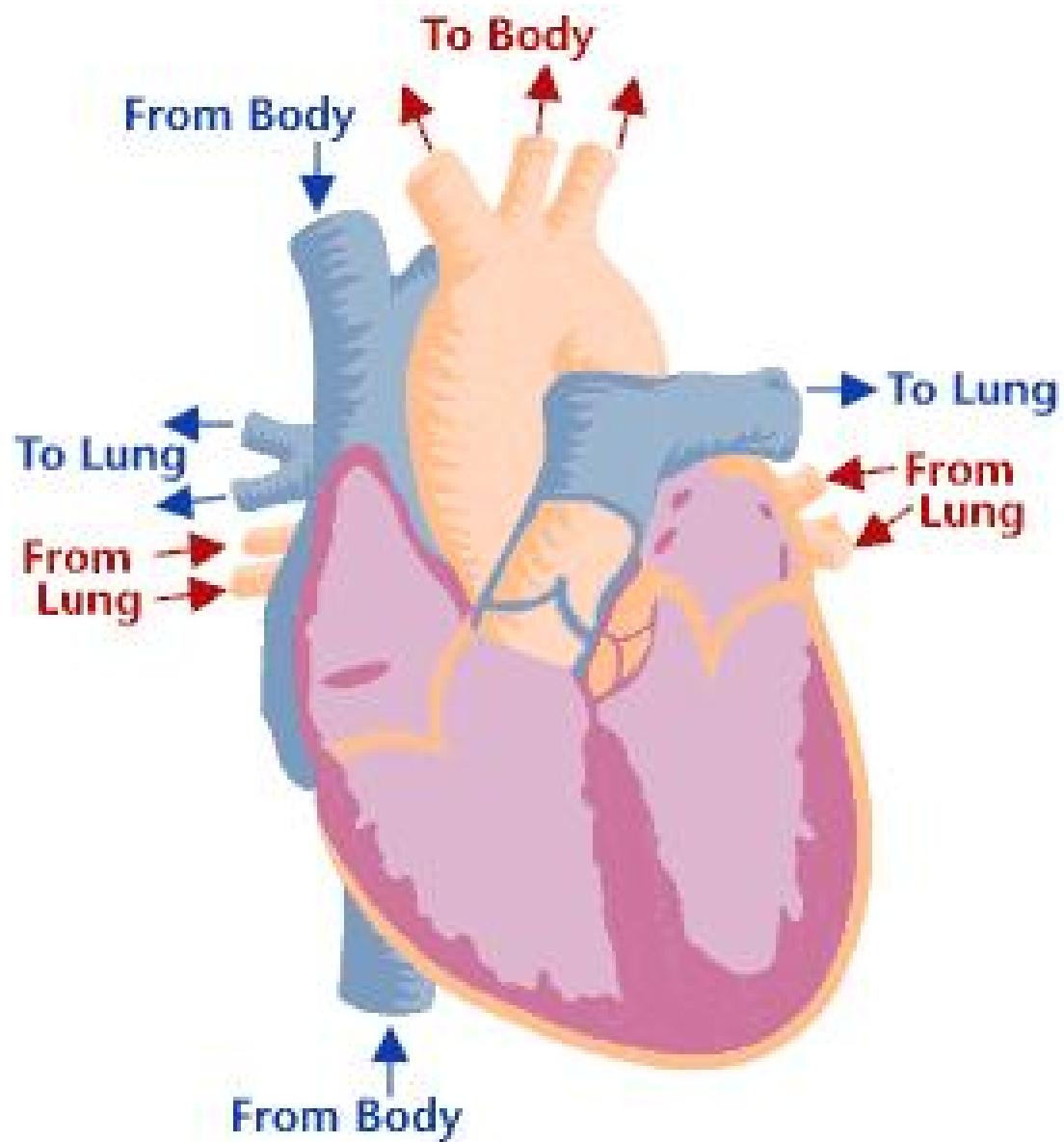


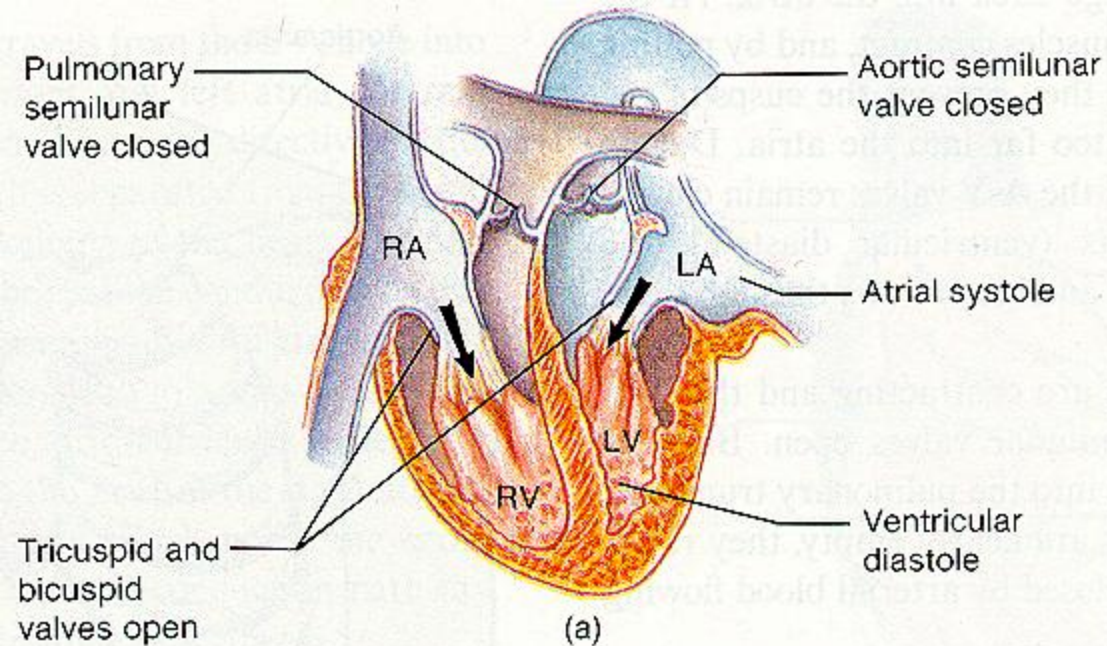




- **End diastolic volume (EDV)-** blood in the ventricles at the end of diastole
- **Ejection fraction-** fraction of the EDV that is ejected (%), used to measure heart efficiency
- **End systolic volume (ESV)-** blood that remains in the ventricle after ejection







## Cardiac Cycle Phases

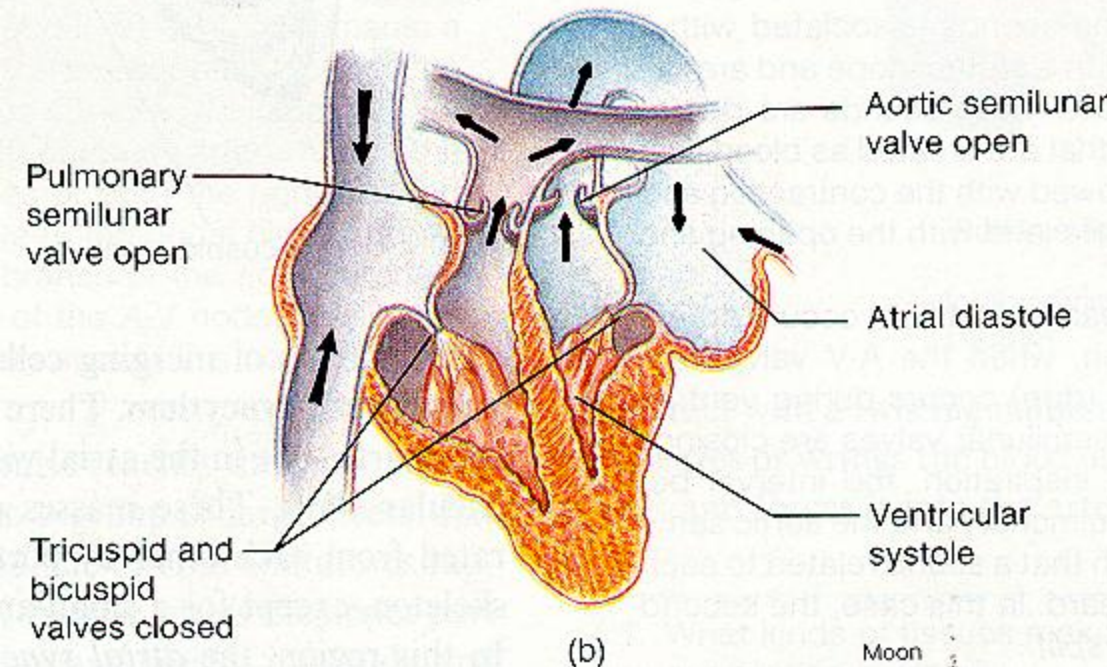
- relaxation (diastole)---dupp (S2)
- ventricular filling
- ventricular contraction (systole)---lubb (S1)

### •Heart murmur- abnormal flow noise

- Mitral Stenosis- narrowing of the Mitral valve opening
- Mitral valve prolapse- when a portion of the valve pushes back to far into the atria upon contraction

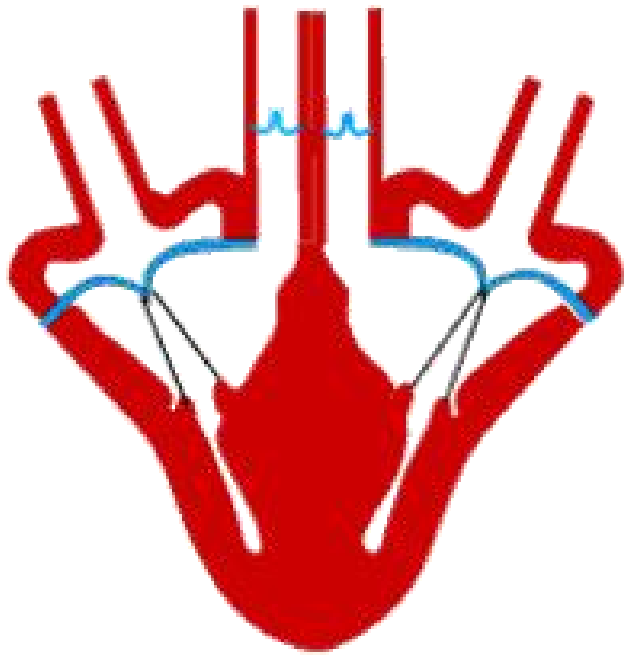
### Cardiac Output- the amount of blood ejected from the heart per minute

- determined by the stroke volume (the amount of blood pumped each stroke) and the heart rate (beats per minute)
- average CO is 5.25L/min

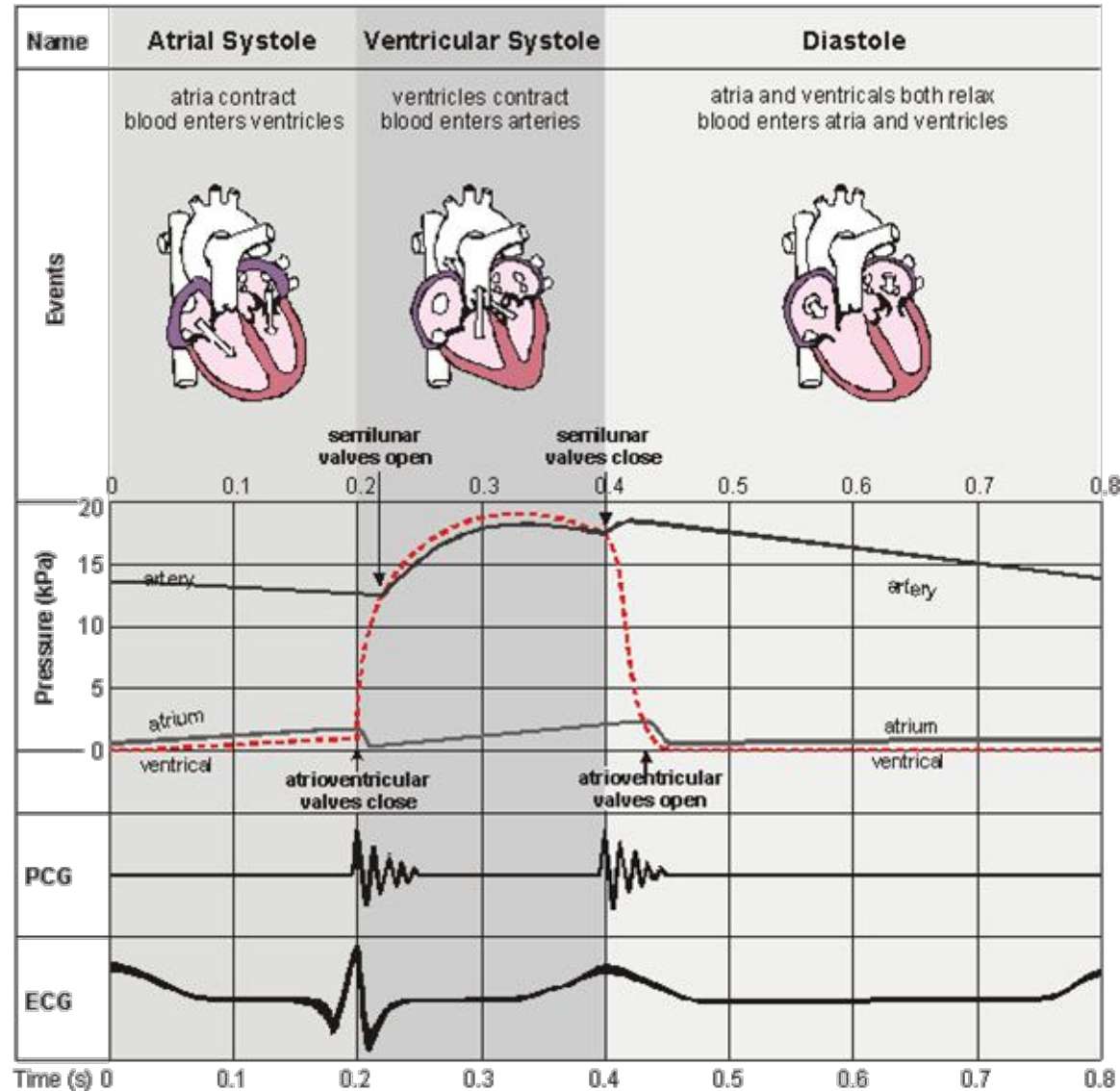


**Starlings law of the heart-** the more cardiac fibers are stretched by the filling of a chamber with blood, the stronger the walls will contract to eject the blood





1. Ventricular filling
2. Ventricular contraction
  - a. av valves close (S1)
  - b. isovolumetric contraction
  - c. semilunar valves open
3. Ejection
4. Begin diastole
5. Semilunar valves close (S2)
6. Isovolumetric relaxation
7. Av valves open

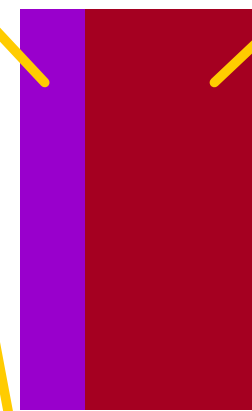


# Stroke Volume

- Governed by preload, contractility, and afterload
- **Preload**- the work imposed on the heart before contraction begins
  - represents the amount of blood that the heart must pump with each beat
    - determined by venous return and the accompanying stretch of the muscles fibers
  - established on Frank-Starling law of the heart-
    - based on the fact that there is an optimal anatomic arrangement of the actin and myosin filaments (when the fibers are stretched 2 ½ times there normal resting length) that generates maximum contraction.

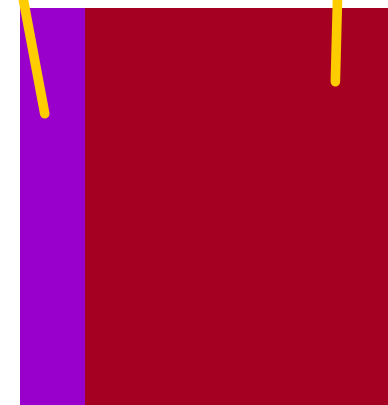
# Starling's Law of the Heart

**End-diastolic volume**  
(volume in ventricle  
at end of relaxation)



Residual

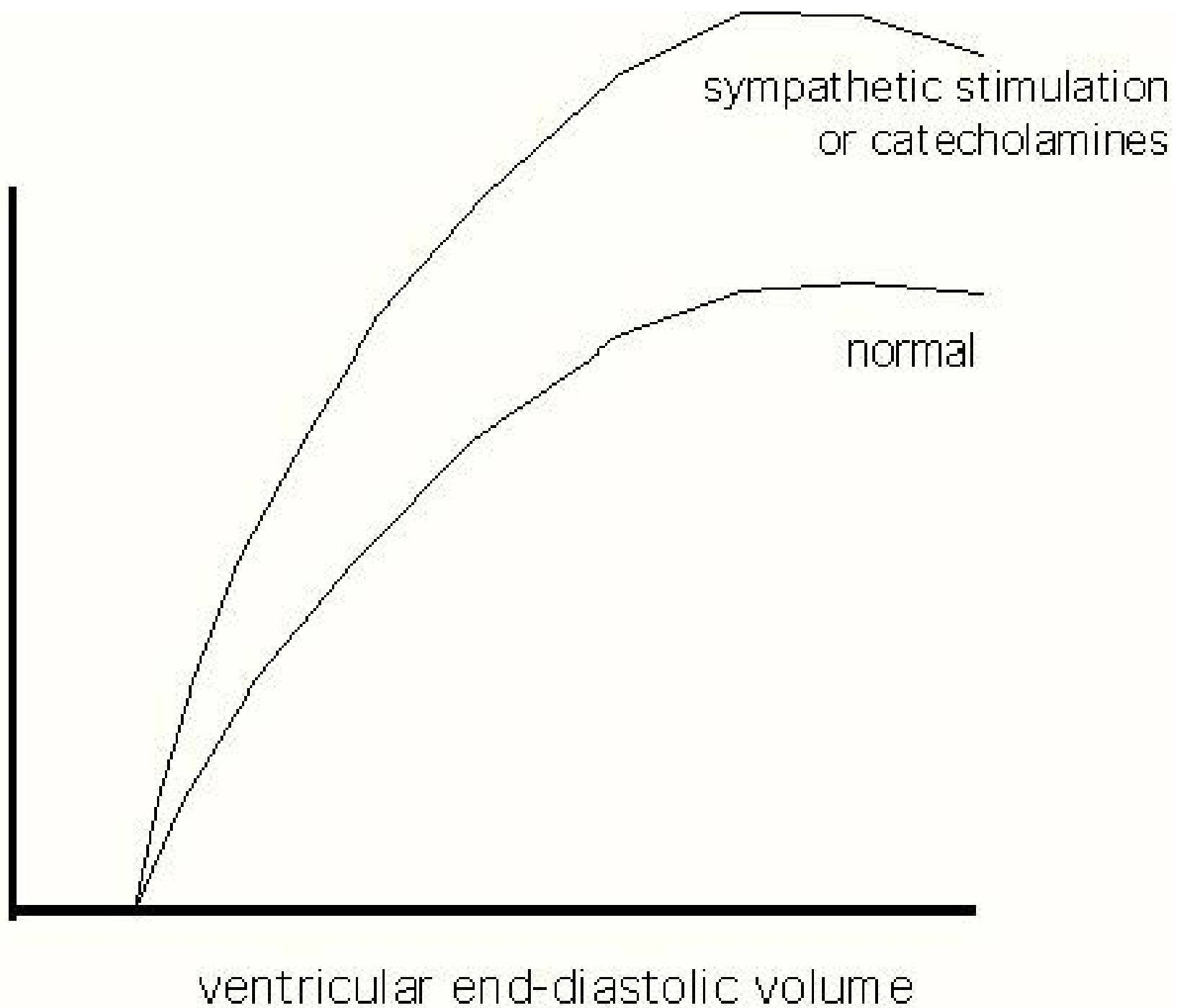
Stroke volume



contraction

contraction

cardiac output





**After load-** the work presented to the heart after the contraction has commenced

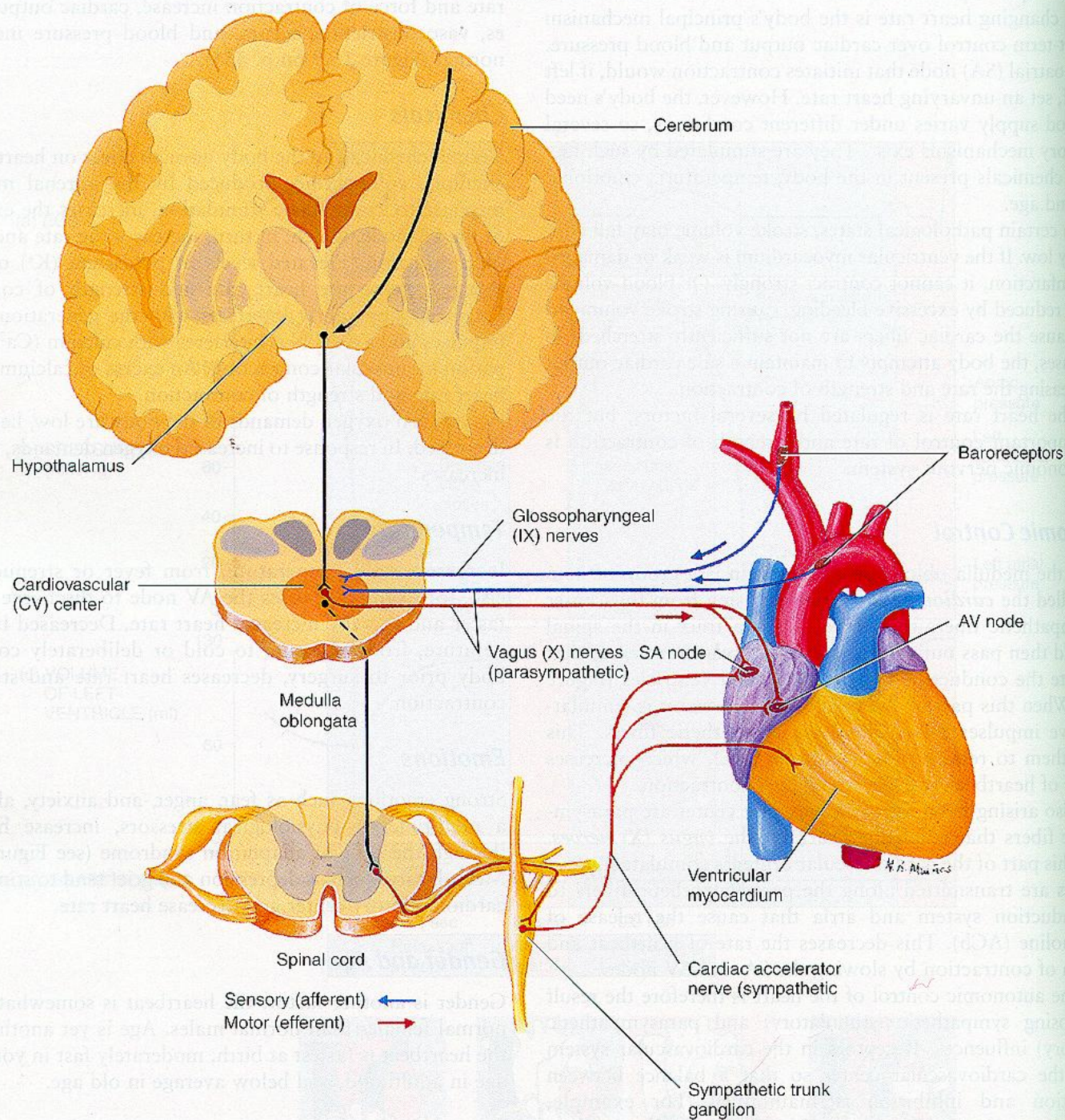
- represents the pressure that the heart must generate to move blood into the aorta
- The systemic arterial blood pressure is the main source of afterload work on the left heart and the pulmonary artery pressure for the right heart.

- **Contractility**- refers to the ability of the heart to change its force of contraction without changing its resting fiber (diastolic) length.
- The state of the muscle is strongly influenced by the number of calcium ions that are available to participate in the contraction process.
- **Inotropic** influence is one that modifies the contractile state of the myocardium independent of Frank-Starling mechanism.
  - ex: sympathetic stimulation produces a positive inotropic effect by increasing the calcium that is available for interaction between the actin and myosin filaments

# Heart Rate

- Determines the frequency with which blood is ejected from the heart
- As heart rate increases, cardiac output tends to increase
- As the HR increases the time spent in diastole is reduced and there is less time for ventricular filling but the time spent in systole remains fairly constant
- This leads to a decrease in stroke volume at high heart rates
  - ex: one of the dangers of **ventricular tachycardia** is a reduction in cardiac output because the heart does not have time to fill adequately.

- **Tachycardia**- adult heart rate above 100 bpm
- **Bradycardia**- adult heart rate below 60 bpm
- **Positive chronotropic agents**- factors that raise the heart rate
- **Negative chronotropic agents**- factors that lower the heart rate



## Heart rate control

sympathetic

parasympathetic

chemical-  $K^+$ ,  $Na^+$ ,  $Ca^{++}$

temperature

emotions

gender/age



# Chronotropic Effects of the Autonomic Nervous System

- The nervous system only modulates the heart beat but does not initiate it.
- 1. cardiac center of the medulla consist of two neuronal pools
  - **cardioacceleratory center**- sends signals by way of the sympathetic cardiac accelerator nerves to the SA node, AV node, and myocardium.
  - These nerves secrete norepinephrine, which bind to B-adrenergic receptors that tend to increase heart rate and contractility

# Chronotropic Effects of the Autonomic Nervous System

2. Cardioinhibitory center- sends signals by way of the parasympathetic fibers in the vagus nerves to the SA and the AV nodes]
  - the right vagus innervates mainly the SA node and the left vagus, the AV node
  - nerve secretes acetylcholine which binds to muscarinic receptors to opens  $K^+$  channels ( $K^+$  leaves the cell) to hyperpolarize the cell which causes the cell to fire less frequently, thereby slowing the heart rate.
  - vagal tone- continuous background vagal input to the nodes that holds the heart rate between 70 and 80 bpm.
    - maximal vagus stimulation can reduce the heart rate to as low as 20 bpm
    - severing the vagus allows the SA node to fire at its own intrinsic frequency of about 100 bpm

# Input to the Cardiac Centers

- Sensory and emotional input via the cerebral cortex, limbic system, and hypothalamus
- **Proprioceptors**- in the muscles and joints inform the cardiac center of changes in physical activity
- **Baroreceptors** (pressure receptors)- pressure sensors in the aorta and internal carotid arteries
- **Chemoreceptors**- sensitive to blood pH, carbon dioxide, and oxygen concentration are found in the aortic arch, carotid arteries, and medulla oblongata
  - respond to **hypercapnia** (excess CO<sub>2</sub>) and **acidosis** (increased H<sup>+</sup>) and **hypoxia** (oxygen deficiency)

# Chronotropic Effects of Chemicals

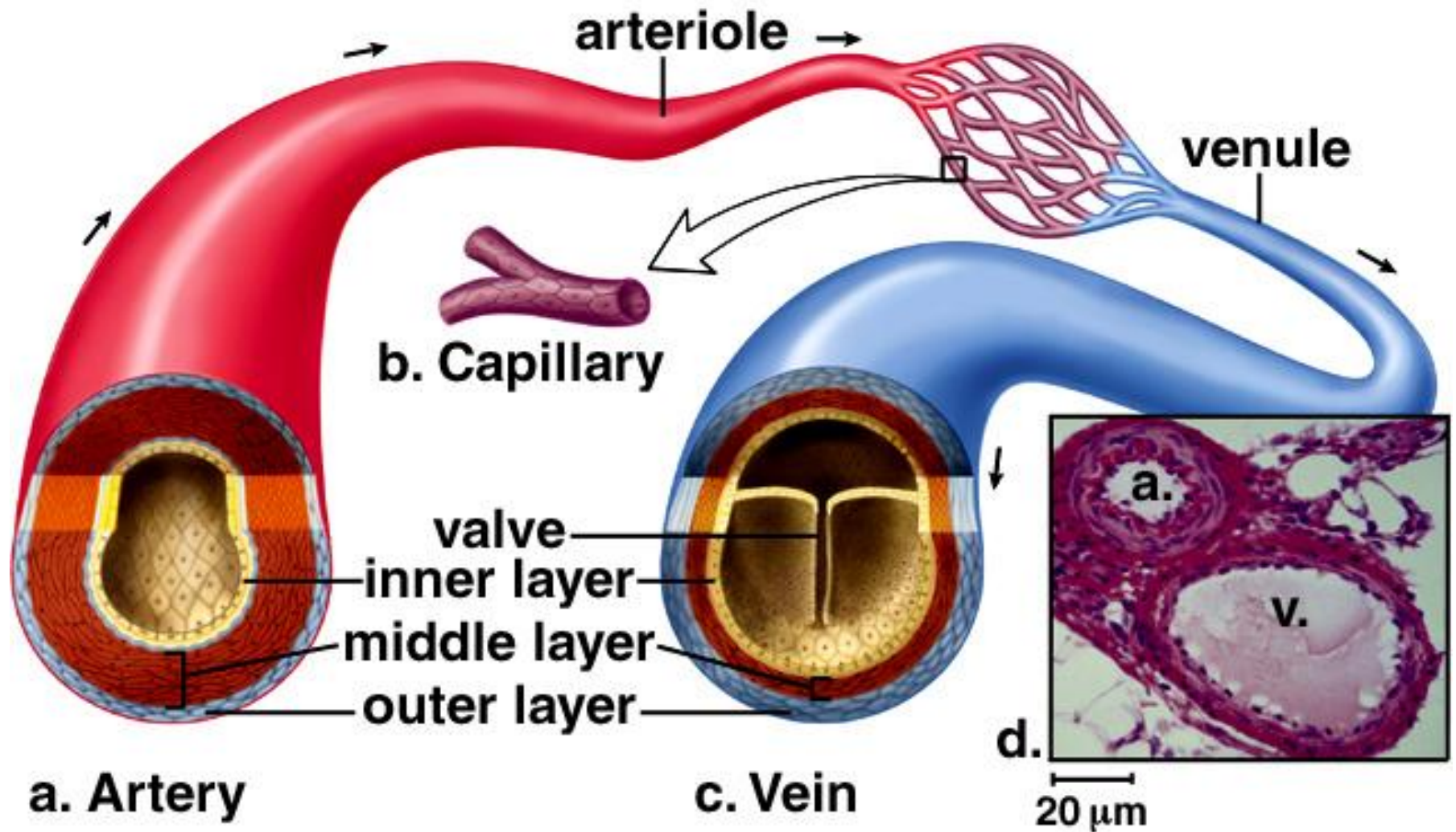
- The catecholamines, Epinephrine and norepinephrine, are potent cardiac stimulants
  - they act through cAMP
- Caffeine and the related stimulants in coffee, tea, and chocolate produce positive chronotropic effects by inhibiting the breakdown of cAMP
- Nicotine accelerates heart rate by stimulating catecholamine secretion
- Thyroid hormone increases the number of adrenergic receptors in the cardiac muscles, thereby making the more responsive to sympathetic stimulation (hyperthyroidism causes tachycardia)

# Chronotropic Effects of Chemicals

- **Hyperkalemia** (excess  $K^+$ )
  - rapid rise makes the myocardium unusually excitable and subject to systolic arrest (ventricles contract and fail to rest)
  - slow rise makes it less excitable than normal; the heart rate becomes slow and irregular and may arrest in diastole
- **Hypokalemia** ( $K^+$  deficiency)- myocytes become hyperpolarized which makes it more difficult to stimulate the cells to threshold
- **Hypercalcemia**- reduces the heart rate and strengthens contraction strength
- **Hypocalcemia**- increases the heart rate and weakens contraction

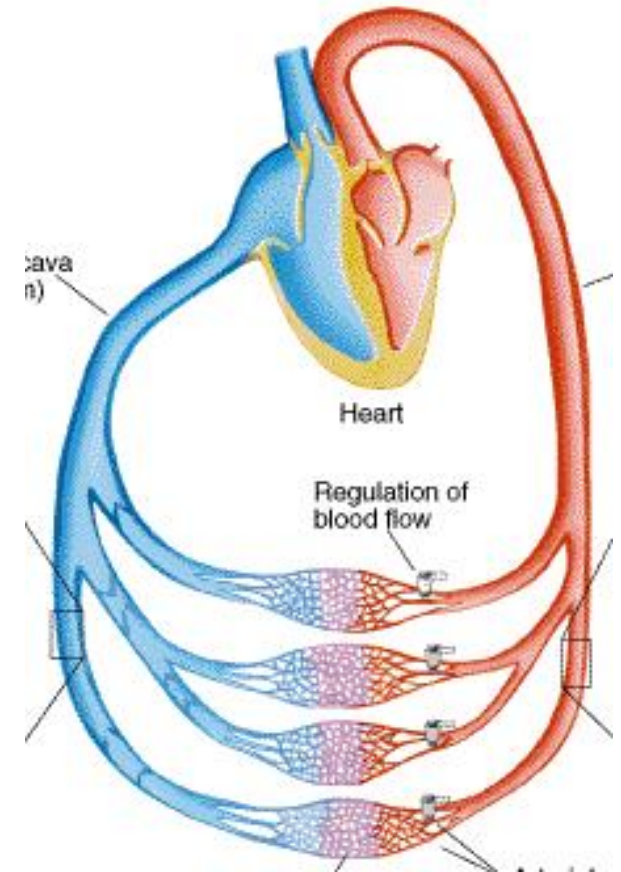


# Blood vessels



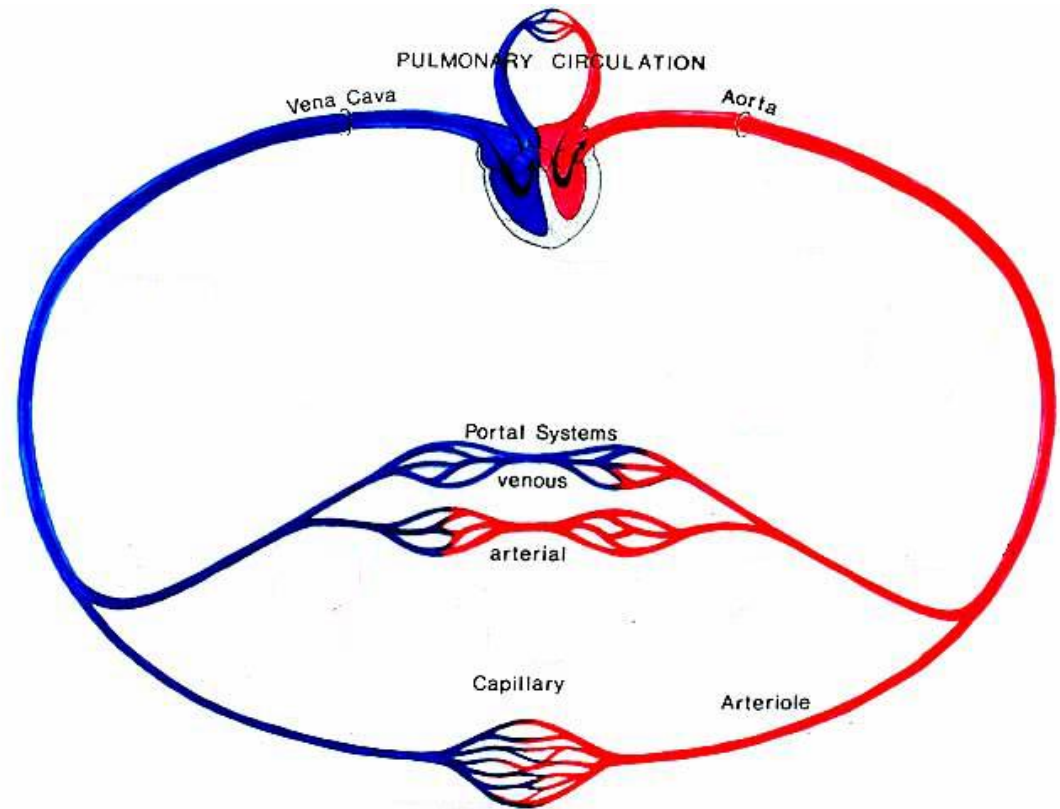
# Circulatory Routes

- Usual route of blood is
  - heart
  - arteries
  - arterioles
  - capillaries
  - venules
  - veins
  - heart



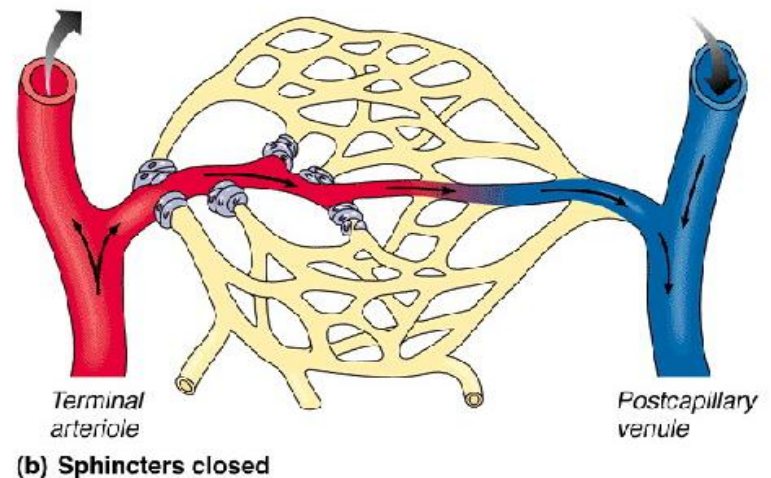
# Circulatory Routes

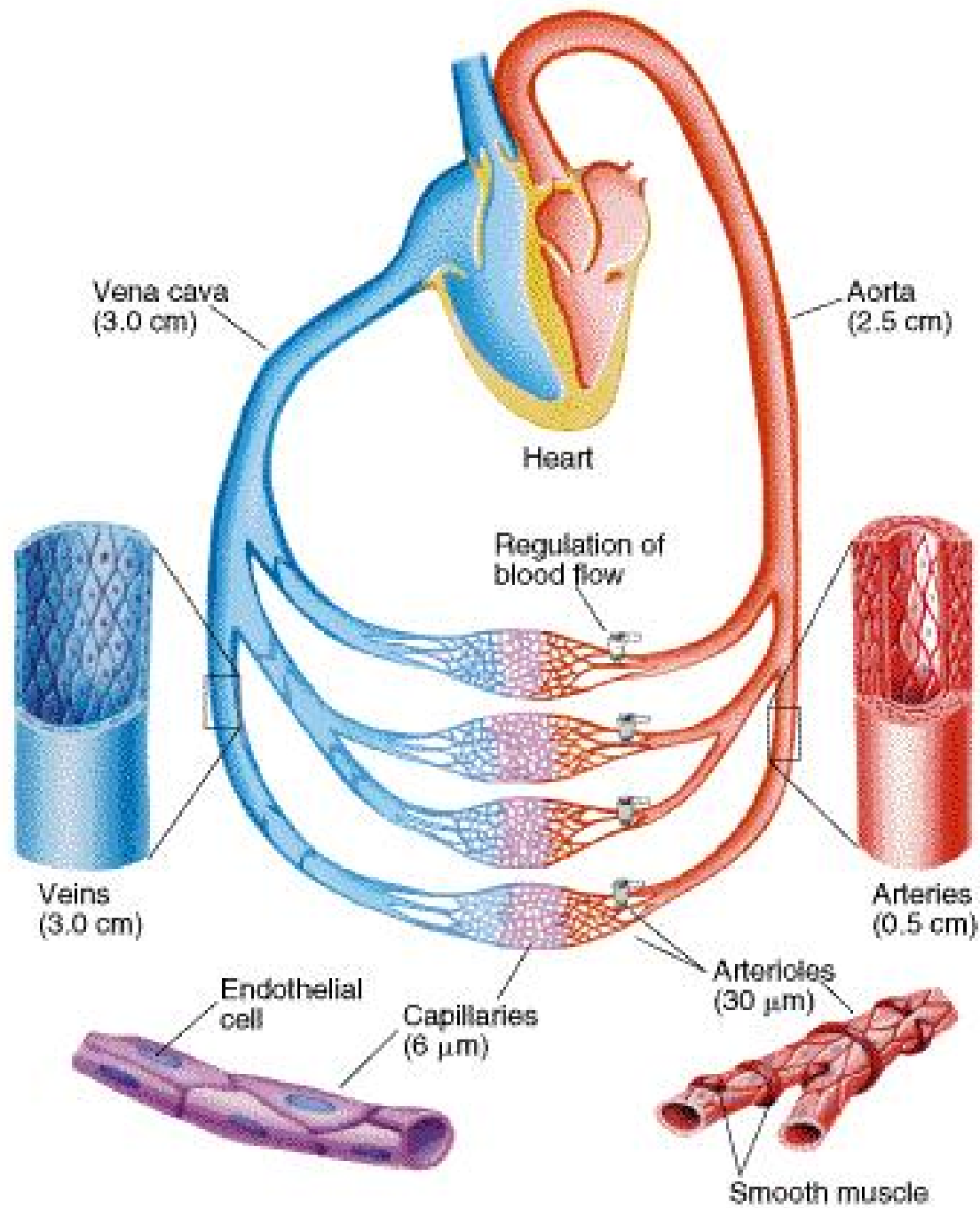
- Portal system- blood flows through two consecutive capillary beds before returning to the heart



# Circulatory Routes

- **Anastomosis**- a point where two blood vessels merge
- **Arteriovenous anastomosis (shunt)**- when blood flows from an artery directly into a vein and bypasses the capillaries
  - occurs in cold weather in fingers, palms, toes, and ears when body is trying to conserve heat
  - makes areas more susceptible to frostbite
- **Arterial anastomosis**- when two arteries merge and provide collateral (alternative) routes of blood supply to a tissue
  - common around joints where movement may temporarily obstruct one pathway
- **Venous anastomosis**-
  - are more common
  - provide several alternative routes of drainage from an organ, therefore, venous blockage is rarely as life-threatening as arterial blockage



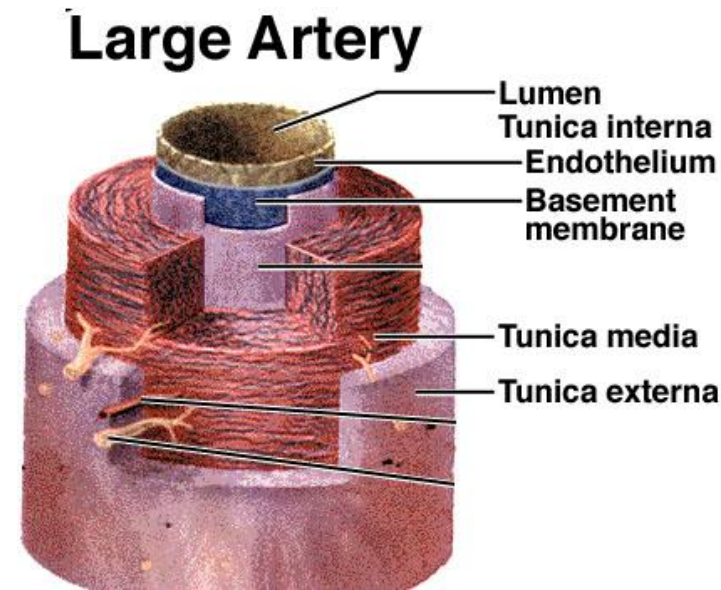
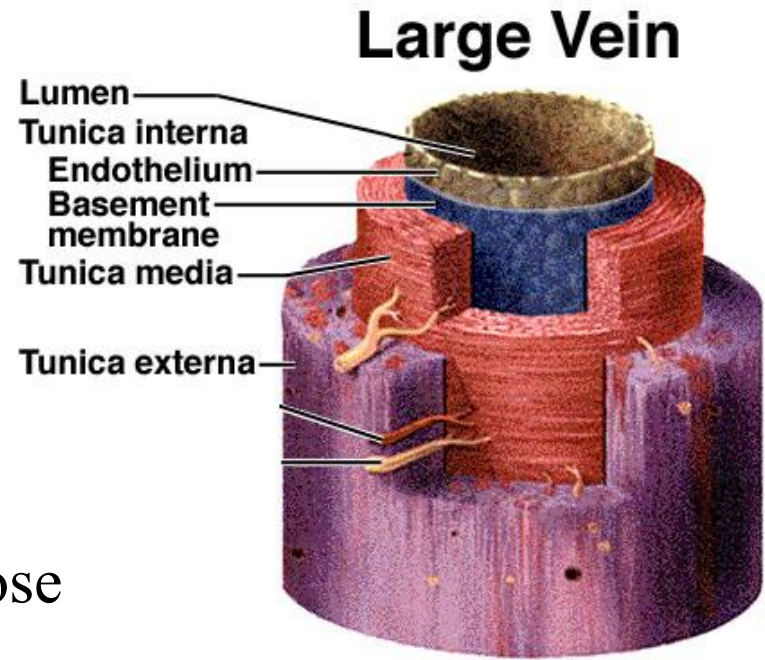




# The Vessel Wall

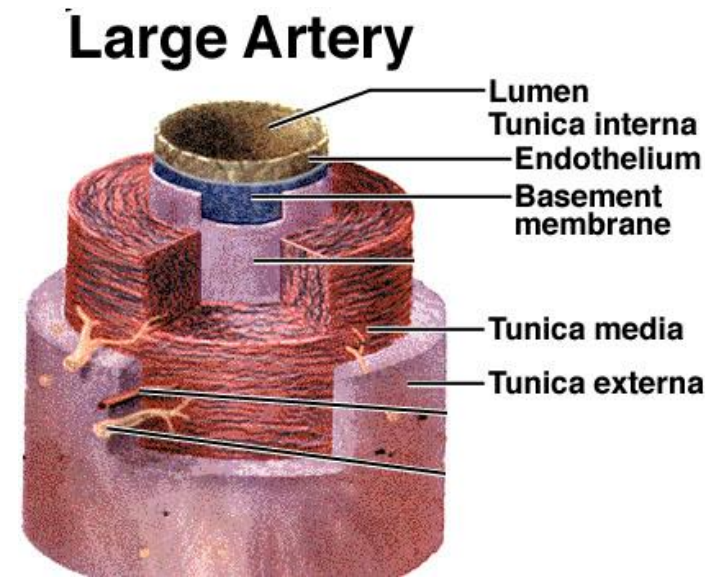
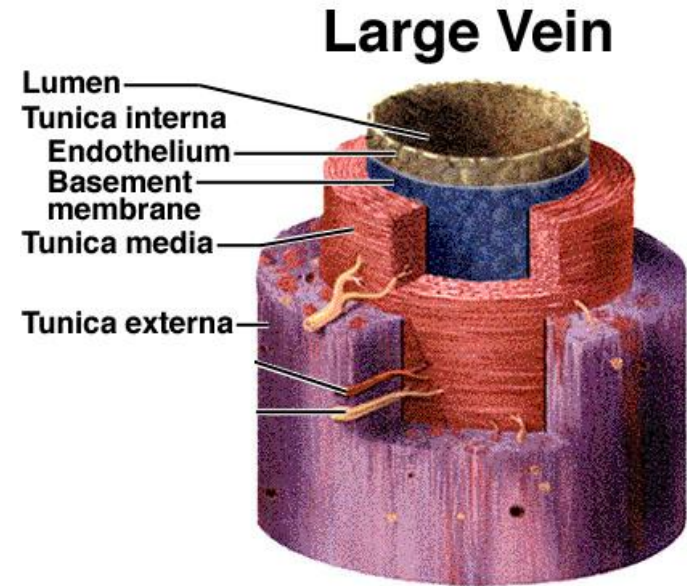
Vessel walls has three layers (tunics)

- **tunica externa (adventitia)**-
  - outer most layer that consist of loose c.t. that anchors the vessel
  - provides passage for small nerves, lymphatics, and smaller blood vessels (**vasa vasorum**) that supply blood to outer half of wall
  - inner half nourished by diffusion of luminal blood



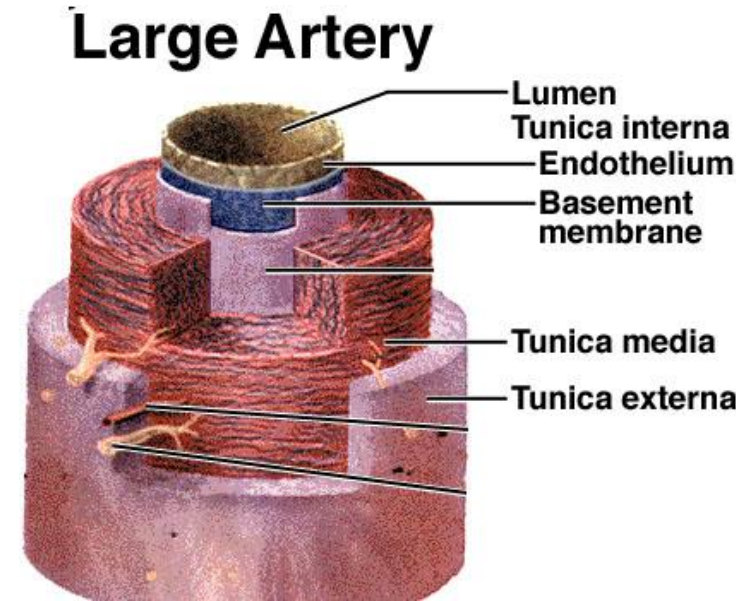
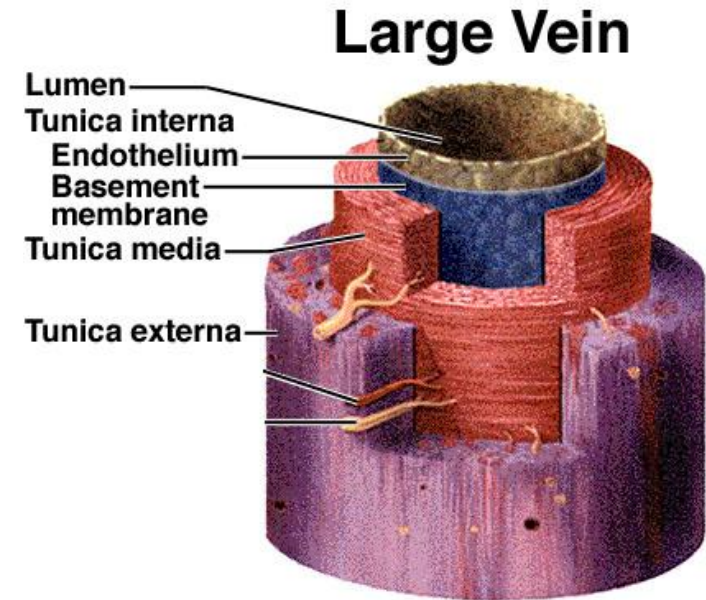
# The Vessel Wall

- Tunica media-
  - the middle layer
  - usually the thickest layer
  - consist of smooth muscle, collagen, and sometimes elastic tissue
  - smooth muscle responsible for vasoconstriction and vasodilation



# The Vessel Wall

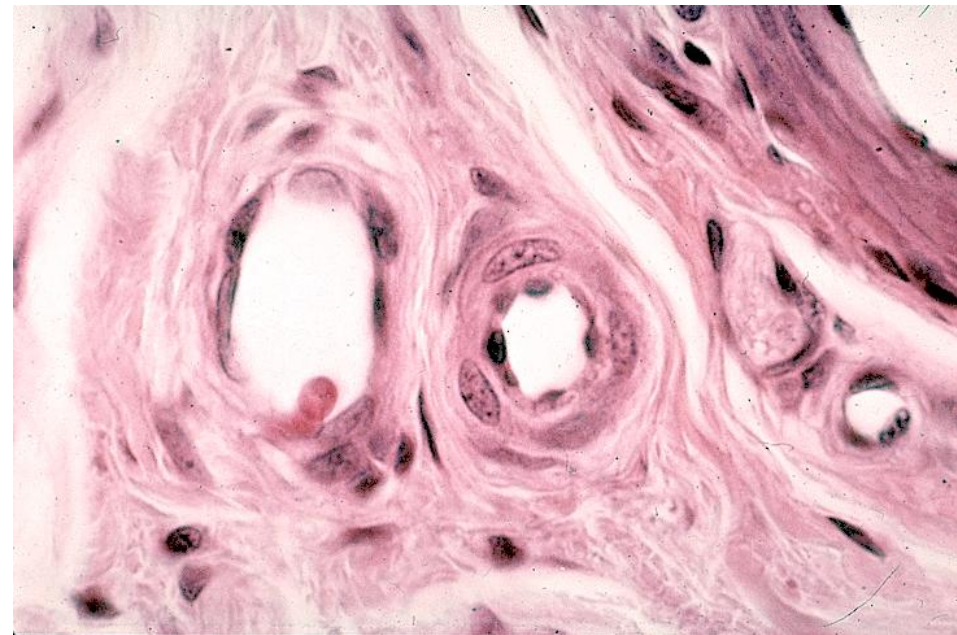
- **Tunica intima (tunica interna)**- inner layer that is exposed to blood
  - consists of a simple squamous endothelium overlaying a basement membrane and a sparse layer of fibrous c.t.
  - acts as a selectively permeable barrier to blood solutes
  - secretes vasoconstrictors and vasodilators
  - provides a smooth inner lining that repels blood cells and platelets
    - platelets adhere to damaged endothelium
    - during inflammation, leukocytes adhere to endothelium by means of **cell-adhesion molecules** produced by the endothelium cells





# Arteries and Metarterioles

- Arteries are constructed to withstand surges of blood pressure generated by the heart
  - more muscular than veins and appear relatively round in tissue sections
- Arteries are divided into three categories by size
  - conducting (elastic)
  - distributing (muscular)
  - resistance (small)



# Arteries and Metarterioles

- **Conducting (elastic) arteries-**
  - are the largest (pulmonary, aorta, common carotids)
  - tunica media consist of lots of elastic tissue and smooth muscle
  - expand when the ventricles pump blood into them during systole and recoil during diastole



# Arteries and Metarterioles

- **Distributing (muscular) arteries**
  - are smaller branches farther away from the heart that distribute blood to specific organs
  - smooth muscle layers consist of  $\frac{3}{4}$  of the wall thickness
  - most arteries to which names are given are of this class (as well as conducting arteries)
    - brachial, femoral, etc.
- **Resistance (small) arteries-** are usually too variable in number and location to be given names
  - lots of smooth muscle in tunica media
  - the smallest of these are the arterioles
  - are the primary points at which the body controls the relative amounts of blood directed to various organs

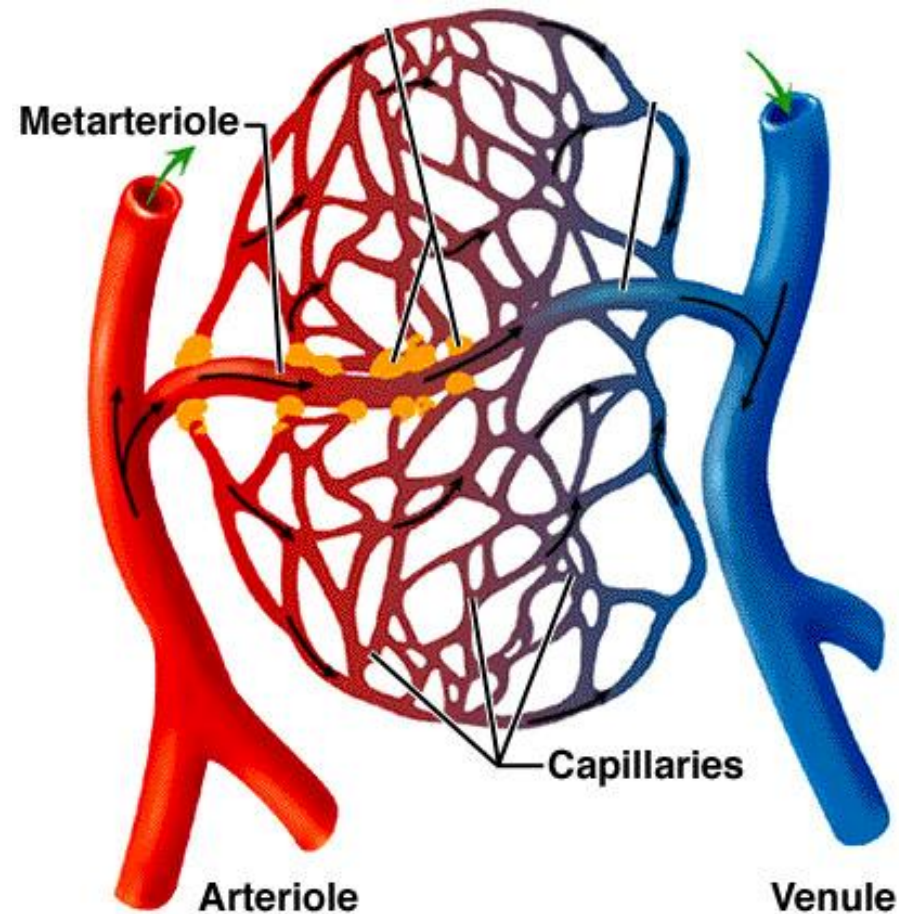
# Metarterioles & Capillaries

## Metarterioles

- short vessels that link arterioles and capillaries
- have smooth muscle pre-capillary sphincter that encircles the entrance to a capillary
- continues through the capillary bed as a thoroughfare channel leading directly to the venule

## Capillaries

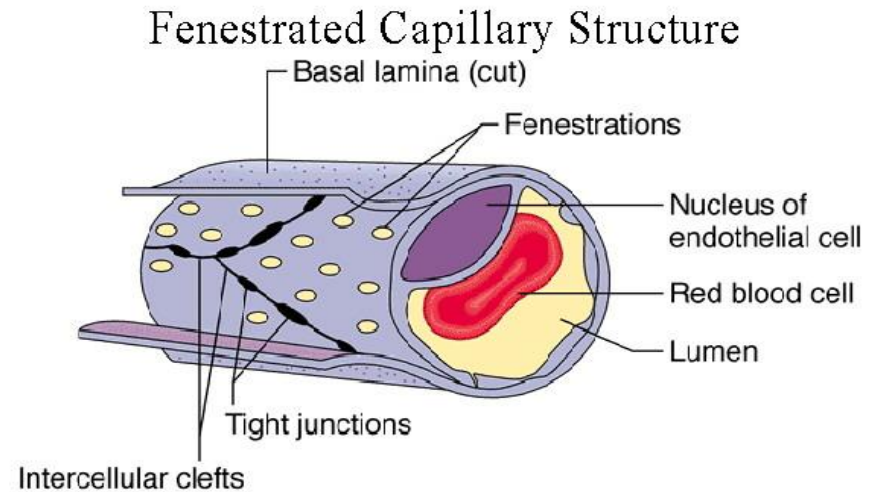
- only point where materials are exchanged between blood and tissue
- consist of only endothelium (one cell thick) and basement membrane
- has very large total surface area ( $6,300 \text{ m}^2$ )



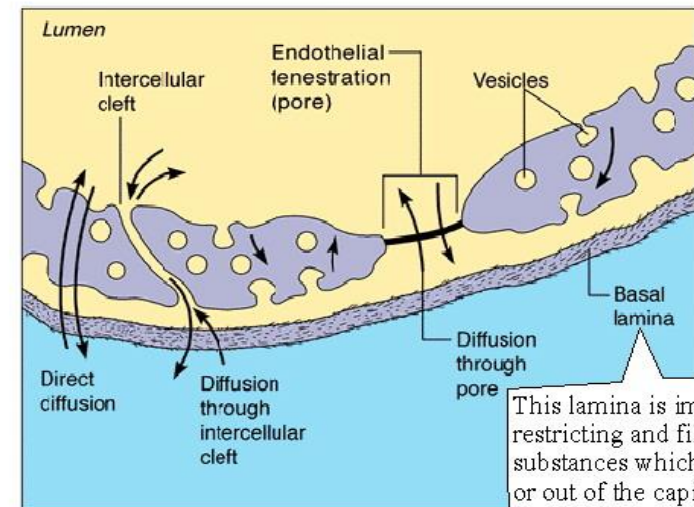
# Types of Capillaries

## Continuous capillaries

- occurs in most tissues
- endothelial cells held together by tight junctions
- cells have narrow intercellular clefts which can pass small solutes such as glucose, but large particles (plasma proteins) are held back
  - capillaries of the blood brain barrier lack intercellular clefts and more complete tight junctions



(a)



(b)

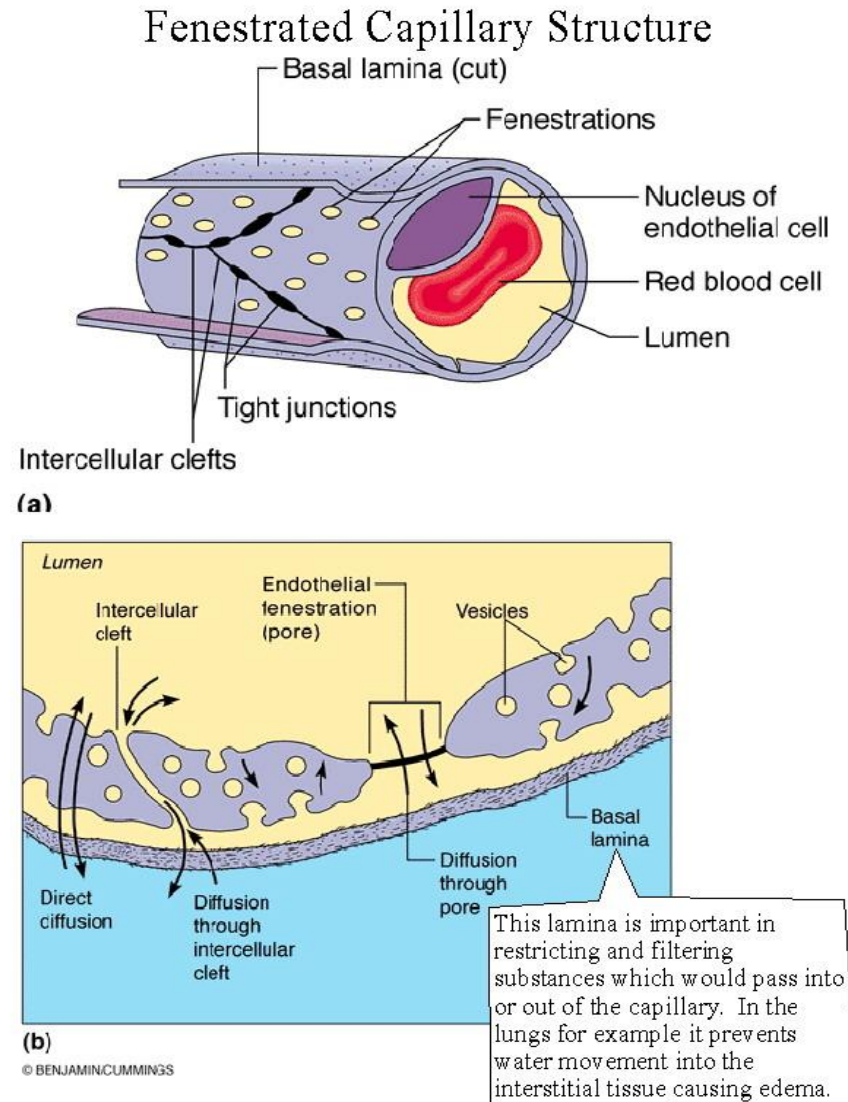
© BENJAMIN CUMMINGS

This lamina is important in restricting and filtering substances which would pass into or out of the capillary. In the lungs for example it prevents water movement into the interstitial tissue causing edema.

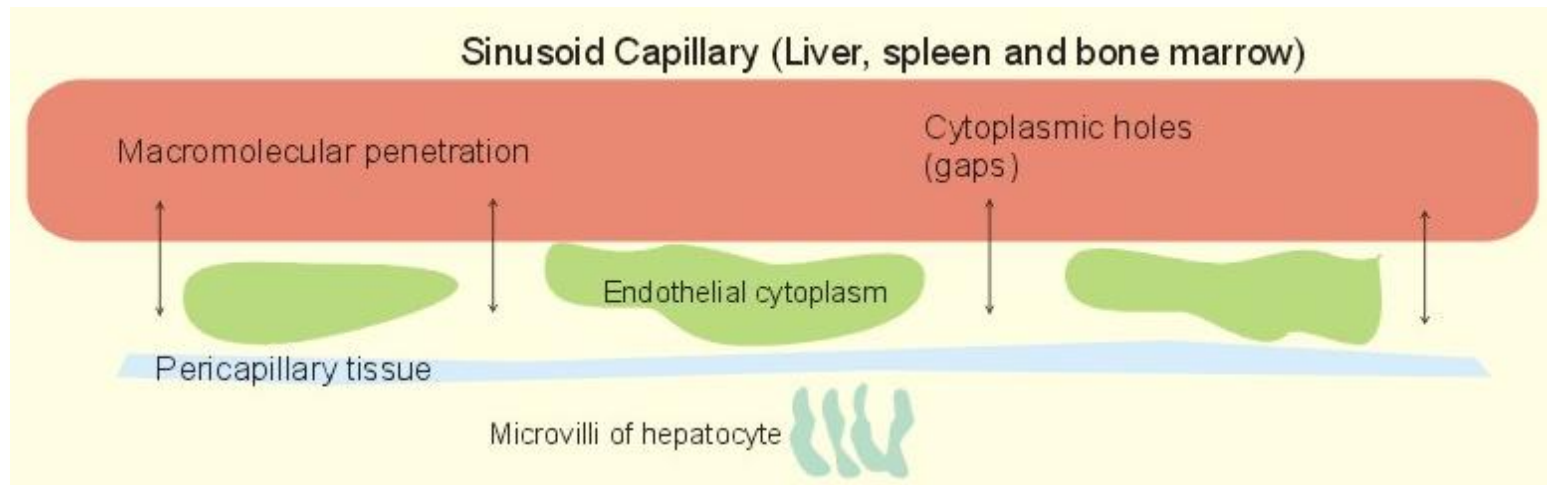
# Types of Capillaries

## Fenestrated capillaries

- cells are riddled with holes called fenestrations (filtration pores)
- fenestrations are covered by a thin mucoprotein diaphragm
- they allow for the rapid passage of small molecules but retain proteins and larger particles in the blood stream
- are important in organs that engage in rapid absorption or filtration (kidneys, endocrine glands, small intestine, choroid plexus of brain)



# Types of Capillaries



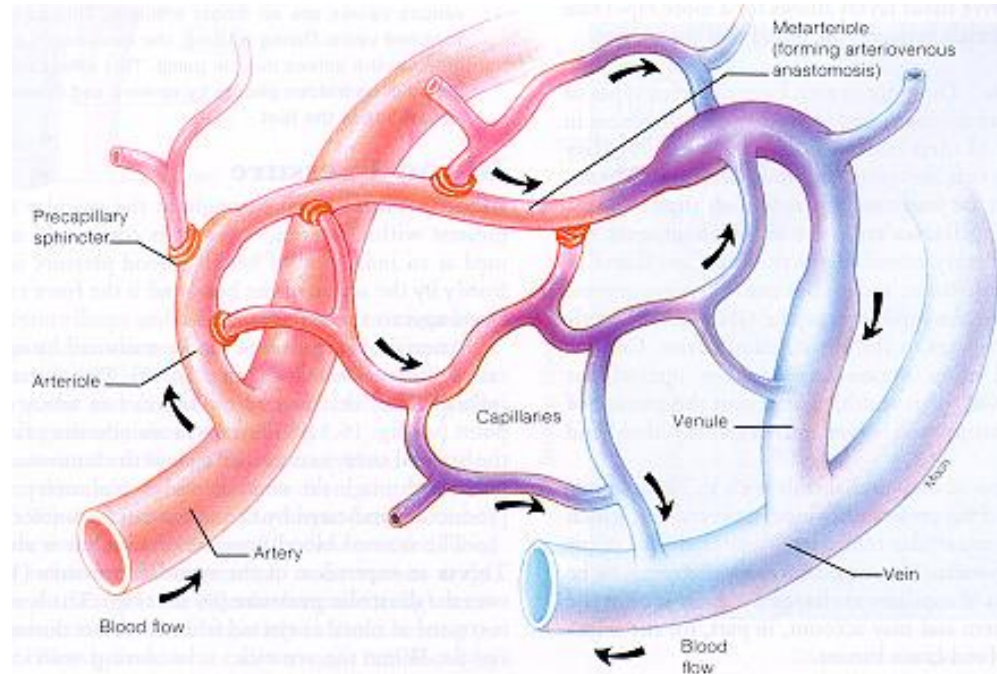
## Sinusoid capillary

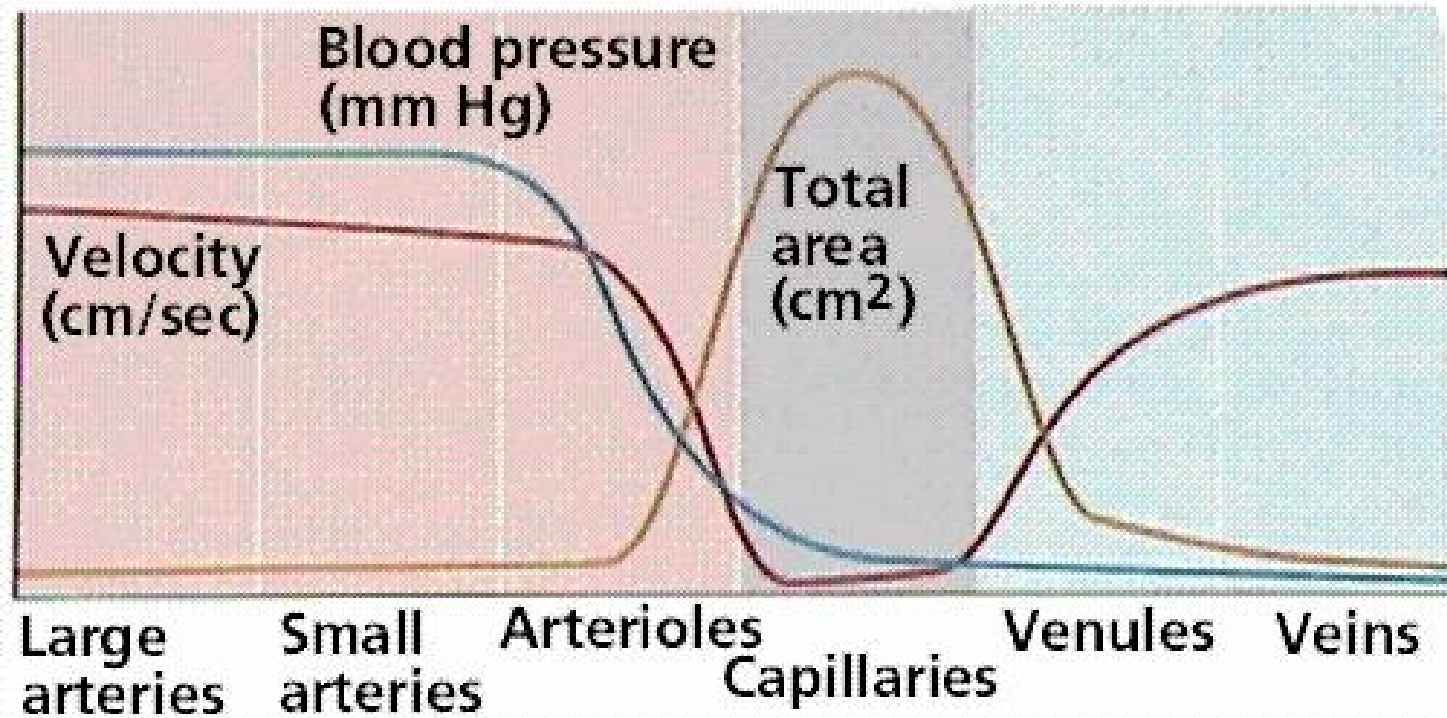
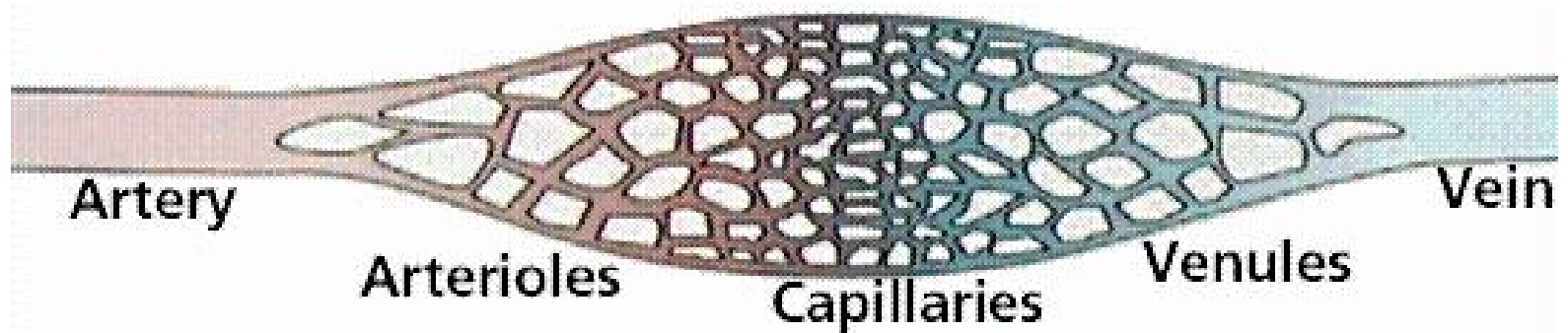
- irregular blood filled spaces that serves as passageways that allow blood plasma to come into direct contact with the parivascular cells
- Pores allow passage of proteins and blood cells
  - how albumin, clotting factors, and other large proteins synthesized by the liver enter the blood
  - how newly formed blood cells enter the circulation from the bone marrow and lymphatic organs



# Veins

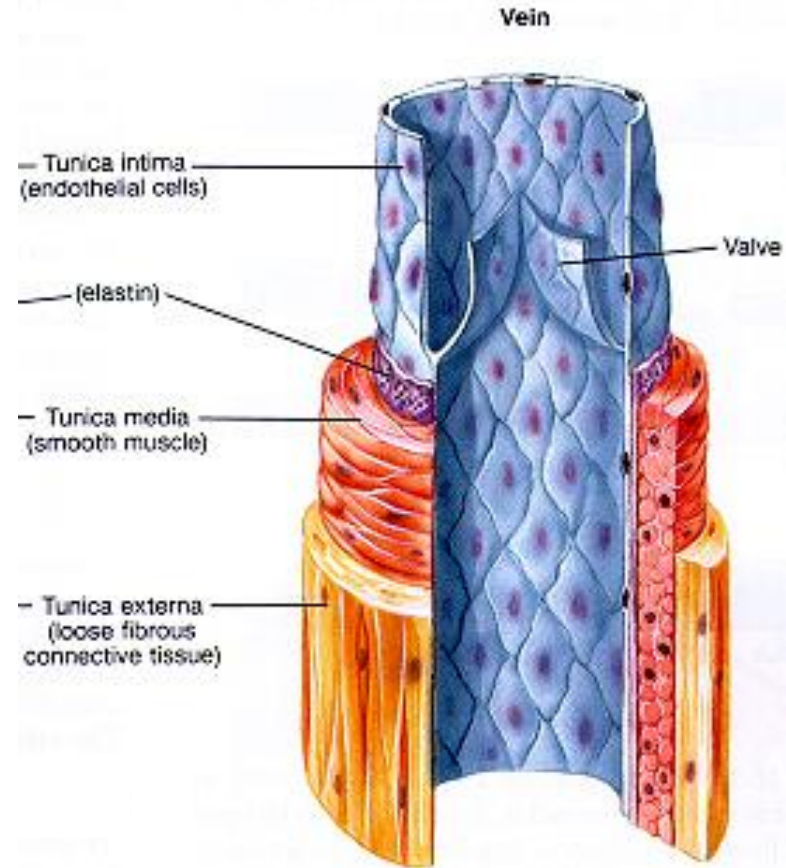
- Blood collects from the capillaries to the venules that are able to exchange fluid with the surrounding tissues
- veins have much lower blood pressure than arteries (avg. 10 mmHg vs. 100mmHg)
- Have thinner walls than arteries with less muscular and elastic tissue
- They collapse when empty and look flattened or irregular in histologic sections
- Expand more easily and accommodate more blood than do arteries
  - approx. 54% of the blood is found in the systemic veins at rest (called **capacitance vessels**)





# Veins

- Pressure in the veins is not high enough to push blood upward against gravity to the heart
- flow of blood back to the heart depends on the
  - **messaging action of skeletal muscle**
  - **one-way valves** that keep blood from dropping down again when the muscles relax
  - **thoracic (respiratory) pump**
  - **cardiac suction**- chordae tendeneae pulls AV valves cusps downward, slightly expanding the atrial space, creating a slight suction
  - **gravity**- blood flow from head
- Valves occur especially in medium sized veins of the arms and legs
- They are absent from very small and very large veins of the ventral body cavity and brain



To heart

Valve open

Vein

Valve closed

Contracted skeletal muscles

Relaxed skeletal muscles



# Circulatory Shock

- Is any state in which cardiac output is insufficient to meet the body's metabolic needs
- All forms of shock fall in to two categories
  - **cardiogenic shock**- caused by inadequate pumping by the heart usually as result of myocardial infarction



# Circulatory Shock

- **low venous return (LVR) shock** has three principal forms
  - hypovolemic shock-
    - most common form
    - produced by a loss of blood volume as a result of hemorrhage, trauma, bleeding ulcers, burn, or dehydration
  - Obstructed venous return shock
    - occurs when something compresses vein and impedes its blood flow
      - ex: a growing tumor or aneurysm

# Circulatory Shock

- Venous pooling (vascular ) shock-
  - occurs when the body has a normal total blood volume, but too much of it accumulates in the limbs
  - can result from long periods of standing or sitting or from widespread vasodilation
  - neurogenic shock- form of venous pooling caused by a sudden loss of vasomotor tone, allowing the vessels to dilate
    - can be cause by severe brainstem trauma or emotional shock

# Circulatory Shock Caused by Both Venous Pooling and Hypovolemic Shock

- **Septic shock-** occurs when bacterial toxins trigger vasodilation and increased capillary permeability
- **Anaphylactic shock-** results from exposure to an antigen to which a person is allergic which triggers release of histamine causing generalized vasodilation and increased capillary permeability

# Principles of Blood flow

Blood flow is determined by two factors

- a pressure difference between the two ends of a vessel or group of vessels
- the resistance that blood must overcome as it moves through the vessel or vessels
- The relationship between pressure, resistance, and flow is expressed by the equation

$$F=P/R$$

F is the blood flow, P is the difference in pressure between the two ends of the system, and R is the resistance to flow through the system

# Principles of Blood flow

- Peripheral resistance is the resistance that the blood encounters in the vessels as it travels away from the heart
- It results from the friction of blood against the walls of the vessels and is proportional to three variables
  - blood viscosity
  - vessel length
  - vessel radius



# Principles of Blood flow

## **Blood viscosity**

- thickness of the blood
- due mainly to erythrocytes and albumin
- A deficiency of red blood cells (anemia) or albumin (hypoproteinemia) decreases peripheral resistance and speeds up blood flow
- Viscosity increases as a result of polycythemia or dehydration, therefore, resistance increases and flow declines

# Principles of Blood flow

## **Vessel length**

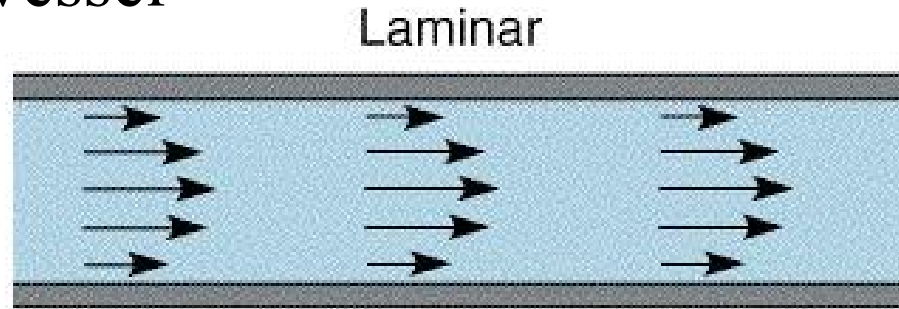
- The farther a liquid travels through a tube, the more cumulative friction it encounters (in the body the length is constant)

## **Vessel Radius**

- Is the only significant way of controlling peripheral resistance
- vasomotion- a change in vessel radius
  - vasoconstriction- smooth muscle contraction
  - vasodilation- smooth muscle relaxation

# Principles of Blood flow

- The effects of vessel radius on blood flow is related to the friction of the moving blood against the walls of the vessel



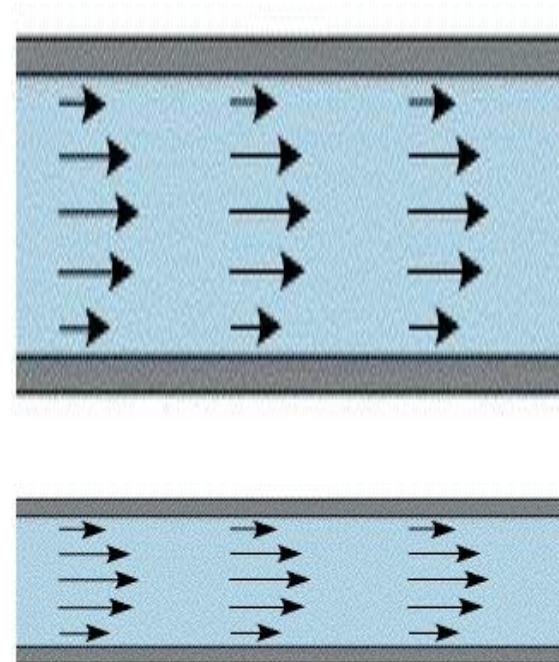
- Blood flow is laminar, meaning, it flows in layers that is faster near the center of the vessel where it encounters less friction, and slower near the walls where it drags against the vessel.

# Principles of Blood flow

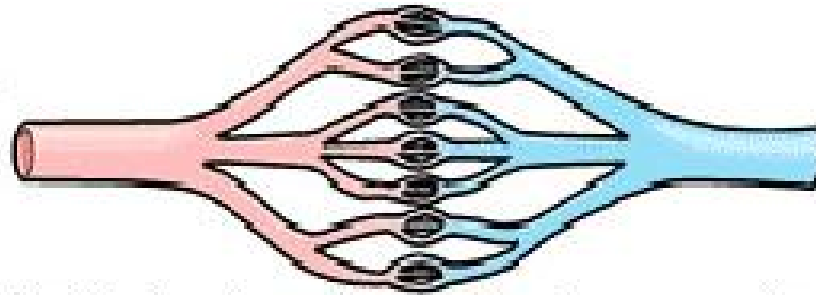
- When a blood vessel dilates, a greater portion of the blood is in the middle of the stream and the average flow is swift
- When the vessel constricts, more blood is closer to the wall and average flow is slower
- Blood flow is proportional to the fourth power of the radius, making a slight change in diameter resulting in a great change in flow

ex:  $r = 2 \text{ mm}$     $F = 16 \text{ mm/sec}$

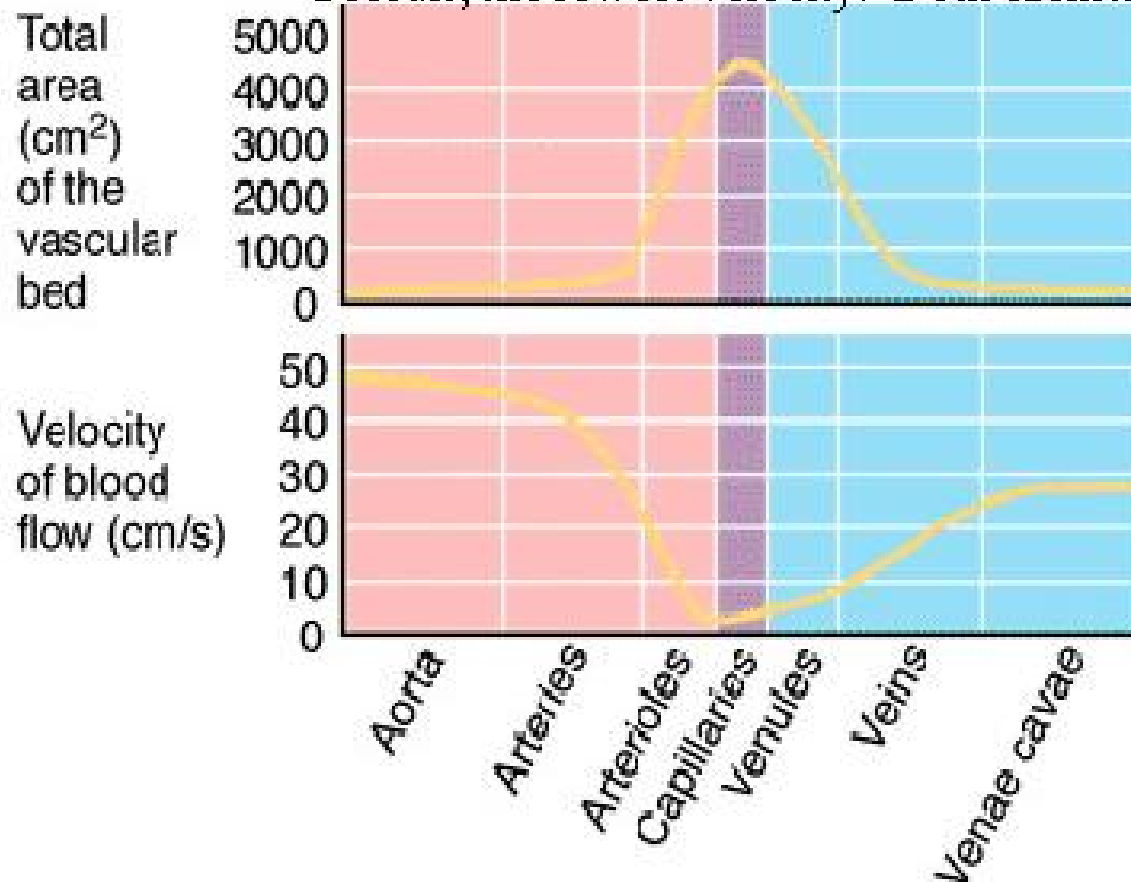
$r = 3 \text{ mm}$     $F = 81 \text{ mm/sec}$



Relative cross-sectional area of different vessels of the vascular bed



Capillaries have the greatest cross-sectional area, and as a result, the lowest velocity. Both facilitate transport.





# Regulation of Blood Pressure and Flow

## Local control

- **Autoregulation**- the ability of tissue to regulate their own blood supply according to its metabolic needs

Ex: in inadequately perfused tissue, waste products accumulate ( $\text{CO}_2$ ,  $\text{H}^+$ ,  $\text{K}^+$ , lactic acid) which stimulates vasodilation which tend to increase perfusion.

- Established through **vasoactive chemicals** secreted by blood platelets, endothelial cells, and the perivascular tissue.
  - Ex: vasodilators- histamine, bradykinin, prostaglandins in inflammation, trauma, and exercise
  - endothelial cells secrete prostacyclin and nitric oxide – vasodilators and **endothelins**- vasoconstrictors

# Regulation of Blood Pressure and Flow

## Local control (cont.)

- **Reactive hyperemia**- increase flow above normal to a tissue when flow is cut off for a time
  - due an accumulation of metabolites during the period of ischemia]
    - ex: when skin flushes after a person comes in from the cold
- **Angiogenesis**- the process where a tissue increases its own perfusion by the growth of new blood vessels
  - three situations in which this is demonstrated
    - regrowth of the uterine lining after menstruation
    - generation of blood vessels in the muscles of well-conditioned athletes
    - growth of arterial bypasses around obstruction in the coronary circulation
    - malignant tumors secrete growth factors to provide more nourishment

# Regulation of Blood Pressure and Flow

## Neural control

- control by hormones and the autonomic nervous system
- vasomotor center-
  - give off sympathetic fibers stimulate most blood vessels to constrict but they dilate the vessels of skeletal and cardiac muscle.
  - integrating center for autonomic reflexes
    - baroreflexes
    - chemoreflexes
    - medullary ischemic reflex- response to a drop in perfusion to the brain
      - increase in heart rate and contraction force
      - widespread vasoconstriction

# Hormonal Control of Blood Pressure

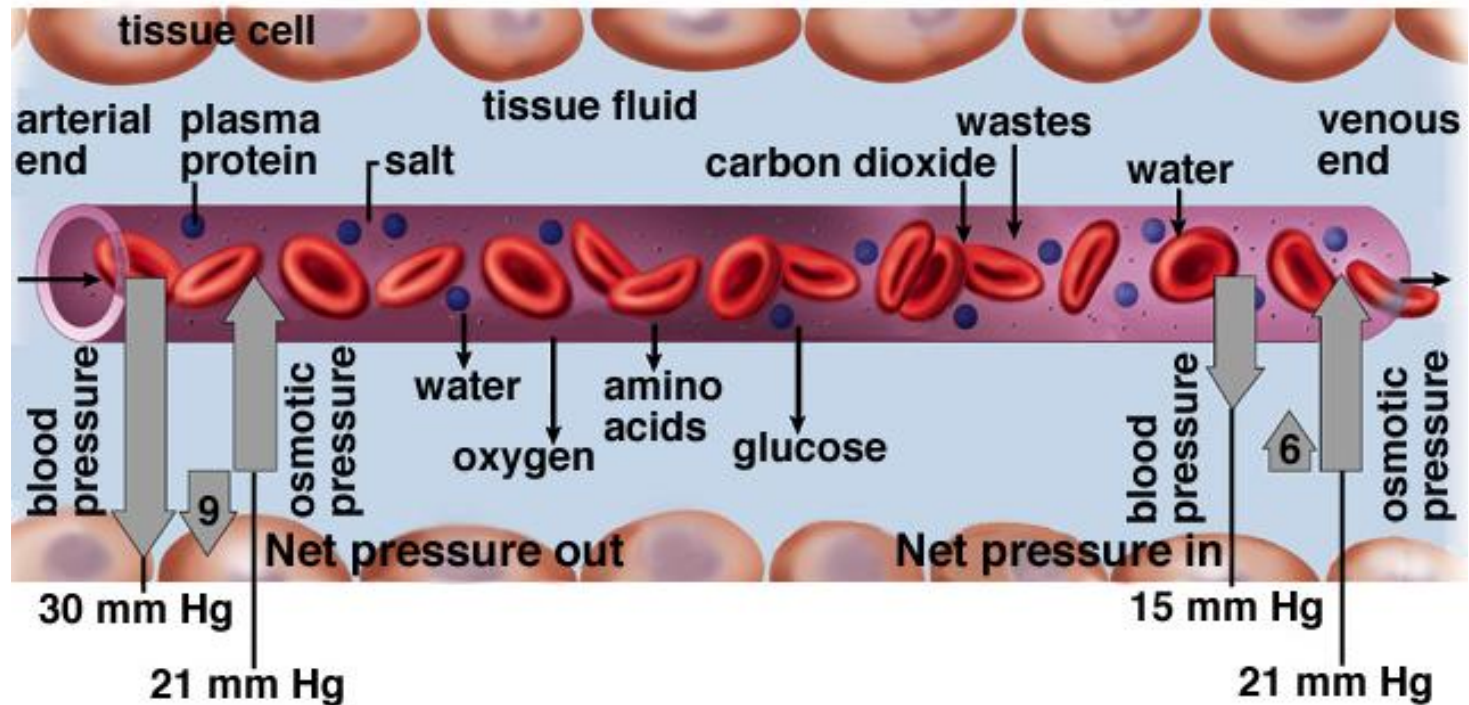
- **Angiotensin II**- potent vasoconstrictor produced by angiotensin converting enzyme (ACE)
  - hypertension often treated with ACE inhibitors
- **Aldosterone**- salt retaining hormone
  - promotes  $\text{Na}^+$  retention by the kidney along with water following
- **Atrial natriuretic peptide (ANP)**- is secreted by the heart and antagonizes aldosterone
  - it increases  $\text{Na}^+$  excretion by the kidney, thus reducing blood volume and pressure
  - also has a generalized vasodilator effect
- **Antidiuretic hormone (ADH)**- promotes water retention but at very high concentrations causes vasoconstriction (hence the name vasopressin)
- **Epinephrine and norepinephrine**- adrenal and sympathetic catecholamine that bind to alpha- adrenergic receptors on smooth muscles of vessels, causing vasoconstriction
  - in skeletal muscles and coronary vessels, they bind to beta- adrenergic receptors causing vasodilation, resulting in increased blood flow to the myocardium

# Capillary Exchange

- Substances pass between the blood and tissue fluid by three routes
  - through the intercellular clefts between endothelial cells
  - through the fenestrations (pores) of fenestrated capillaries
  - through the endothelial cell cytoplasm
- The mechanisms of exchange involve
  - diffusion
  - transcytosis
  - filtration
  - reabsorption



# Capillary Exchange



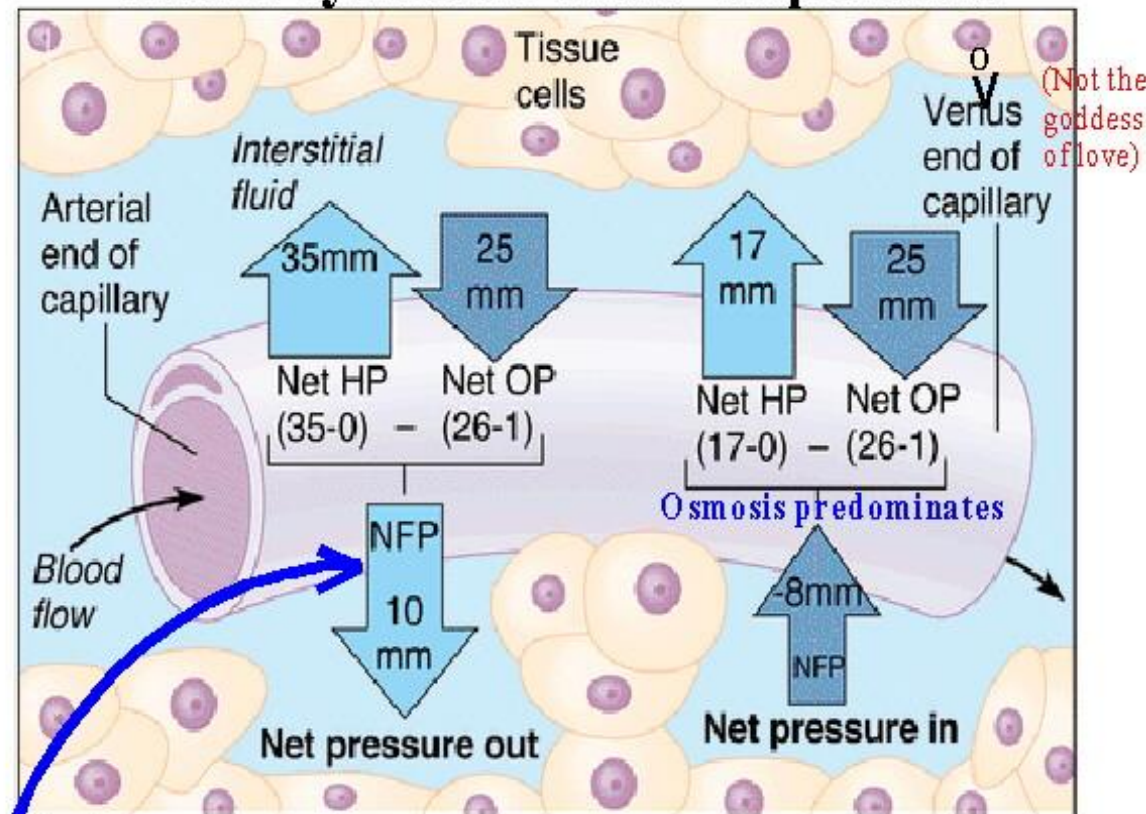
## Diffusion

- most important mechanism for exchange
- glucose and oxygen diffuse from vessels to tissue
- CO<sub>2</sub> and other wastes products diffuse from tissue to vessels

## Other forces in capillary fluid dynamics

- blood pressure (**hydrostatic pressure**) causing filtration
- **Oncotic pressure**- the difference between the interstitial pressure (colloid osmotic pressure of the tissue) and the colloid osmotic pressure of the plasma
- Pressures change from arterial end to venous end

## Fluid Dynamics in the Capillaries



Key to pressure values:

HP<sub>c</sub> at arterial end = 35 mmHg    HP<sub>if</sub> = 0 mmHg    OP<sub>if</sub> = 1 mmHg  
 HP<sub>c</sub> at venous end = 17 mmHg    OP<sub>c</sub> = 26 mmHg

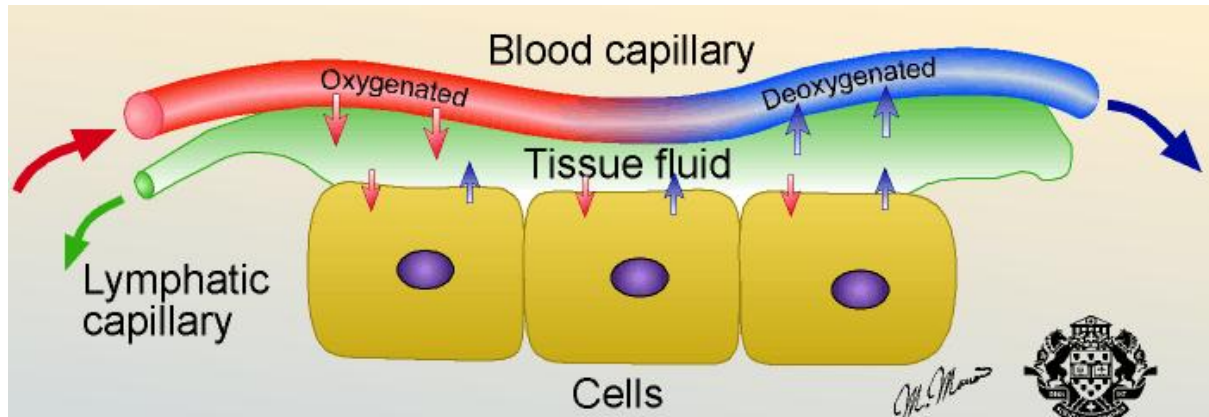
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**Filtration** - movement of water and dissolved substances out of the capillary due to hydrostatic pressure

HP = hydrostatic pressure (blood pressure)

OP = osmotic pressure

# Edema



- the accumulation of excess fluid in a tissue causes by
  - increased capillary filtration caused by increased permeability or blood pressure
    - poor venous return causes back pressure
      - congestive heart failure, incompetent heart valves
    - prolonged confinement to bed or wheelchair (insufficient muscular activity)
    - kidney failure- causing water retention and hypertension
- Reduced capillary reabsorption caused by a reduction in oncotic pressure
  - hypoproteinemia caused by liver damage
- Obstructed lymphatic drainage or surgical removal

# Edema

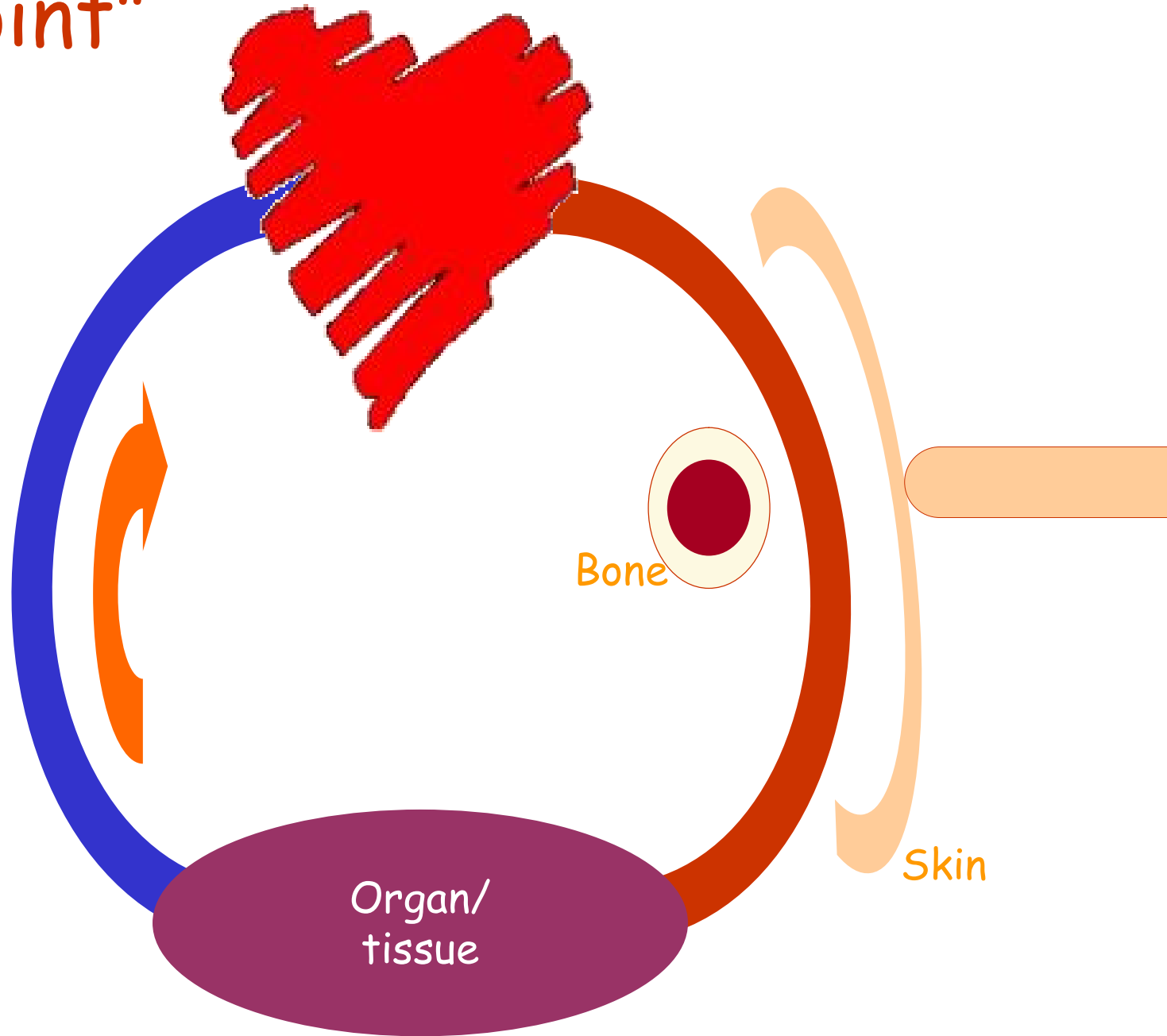
- Severe edema
  - may lead to circulatory shock too much of a transfer of fluid from vessels to tissues
  - As the tissue becomes swollen with fluid, oxygen delivery and waste removal are impaired leading to tissue necrosis
  - Pulmonary edema presents a threat of suffocation
  - cerebral edema can produce headaches, nausea, seizures, and coma





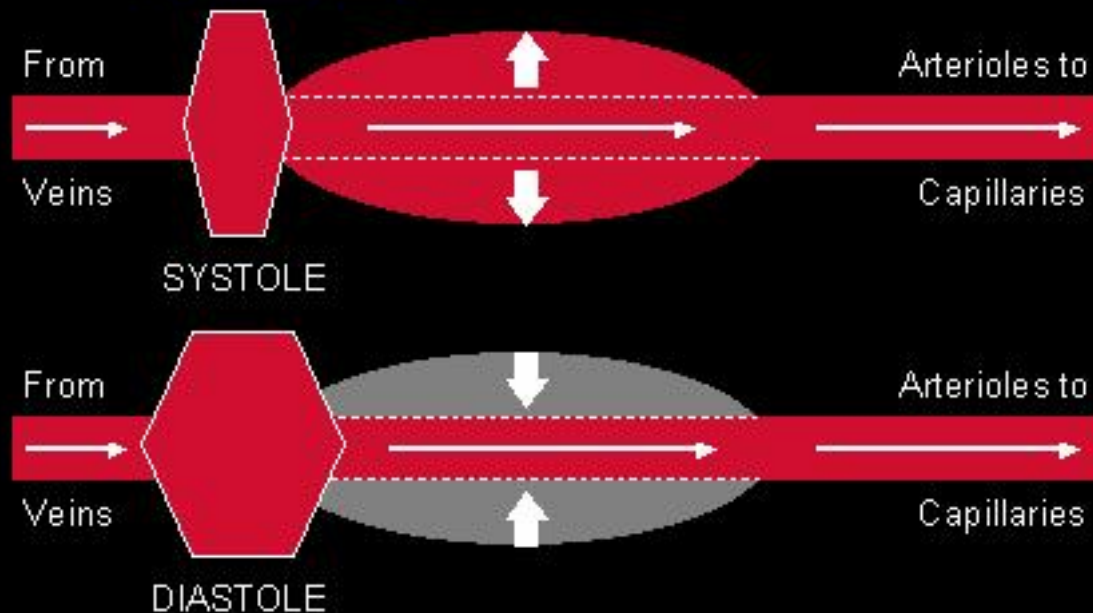


"pulse point"

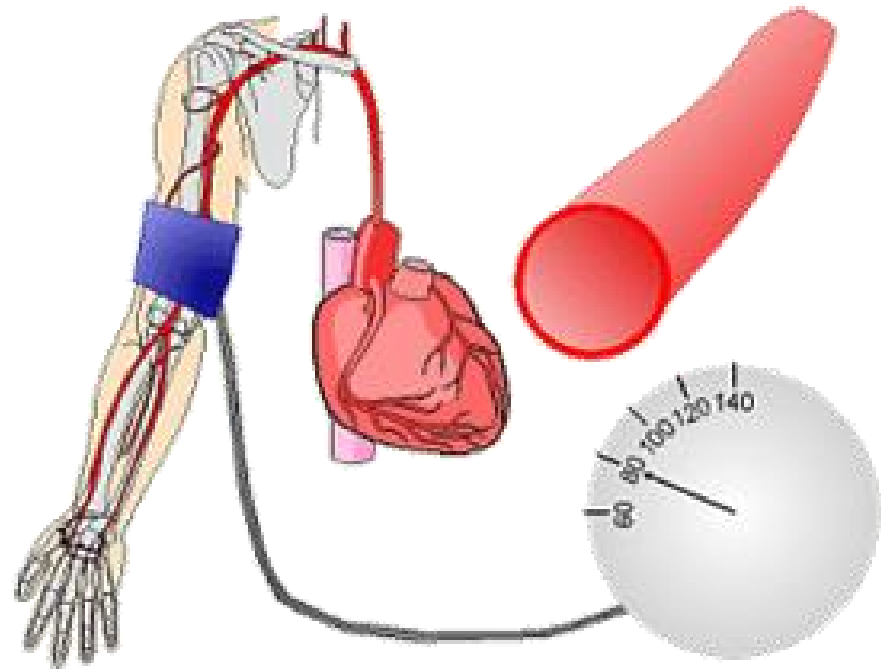
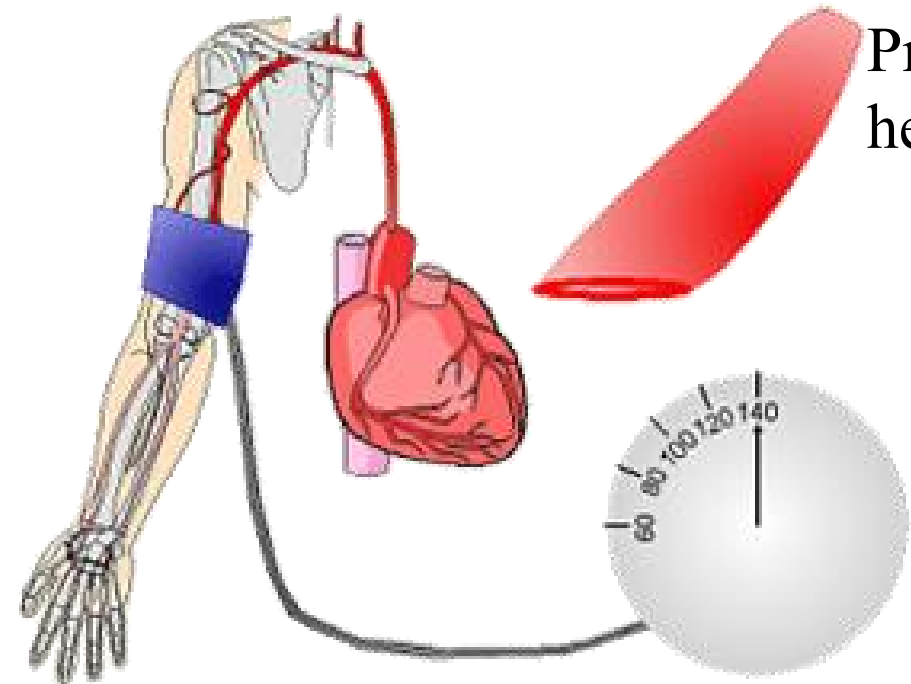


## Arteries

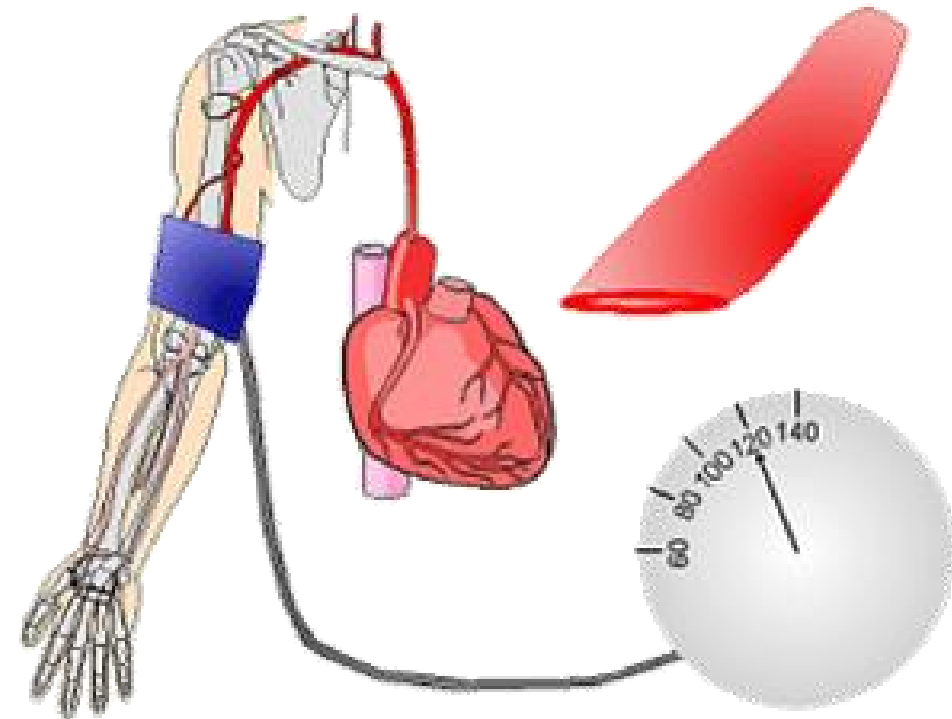
- ◆ Low resistance, rapid transit passageways
- ◆ Muscle & elastic connective tissue in walls
  - *elastic recoil*



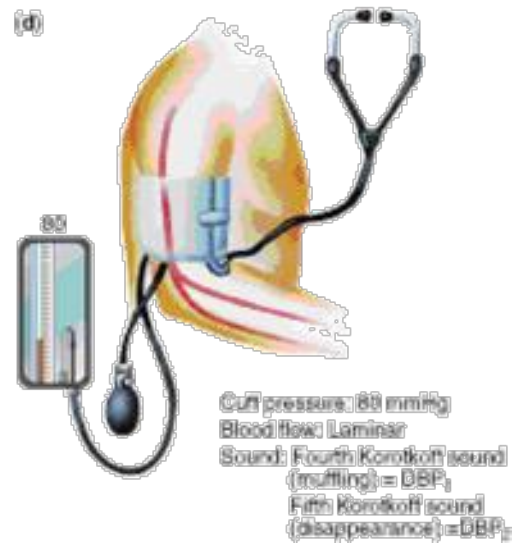
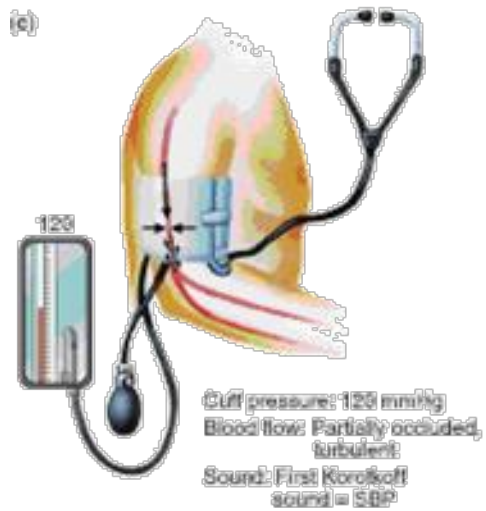
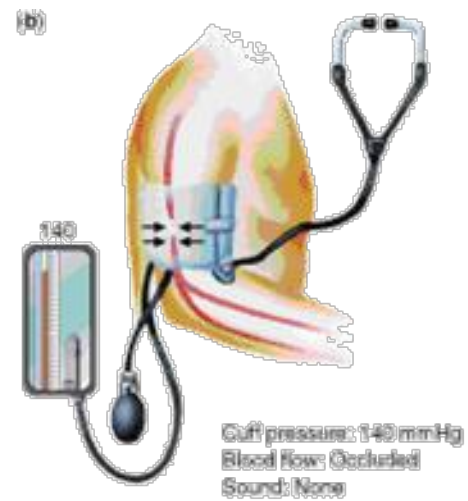
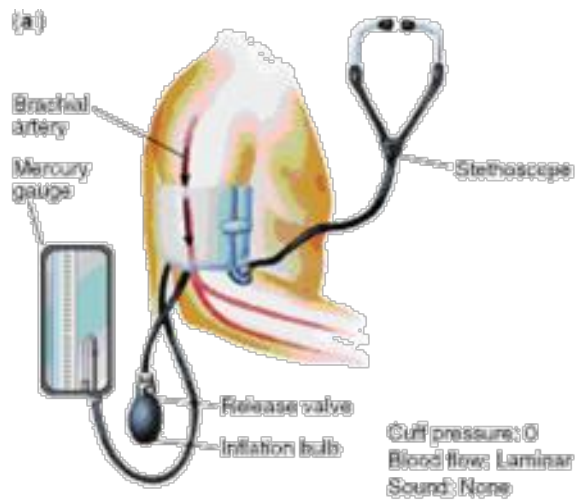
Pressure of cuff > than  
heart systolic press



Heart pressure > cuff (both  
systolic and diastolic)

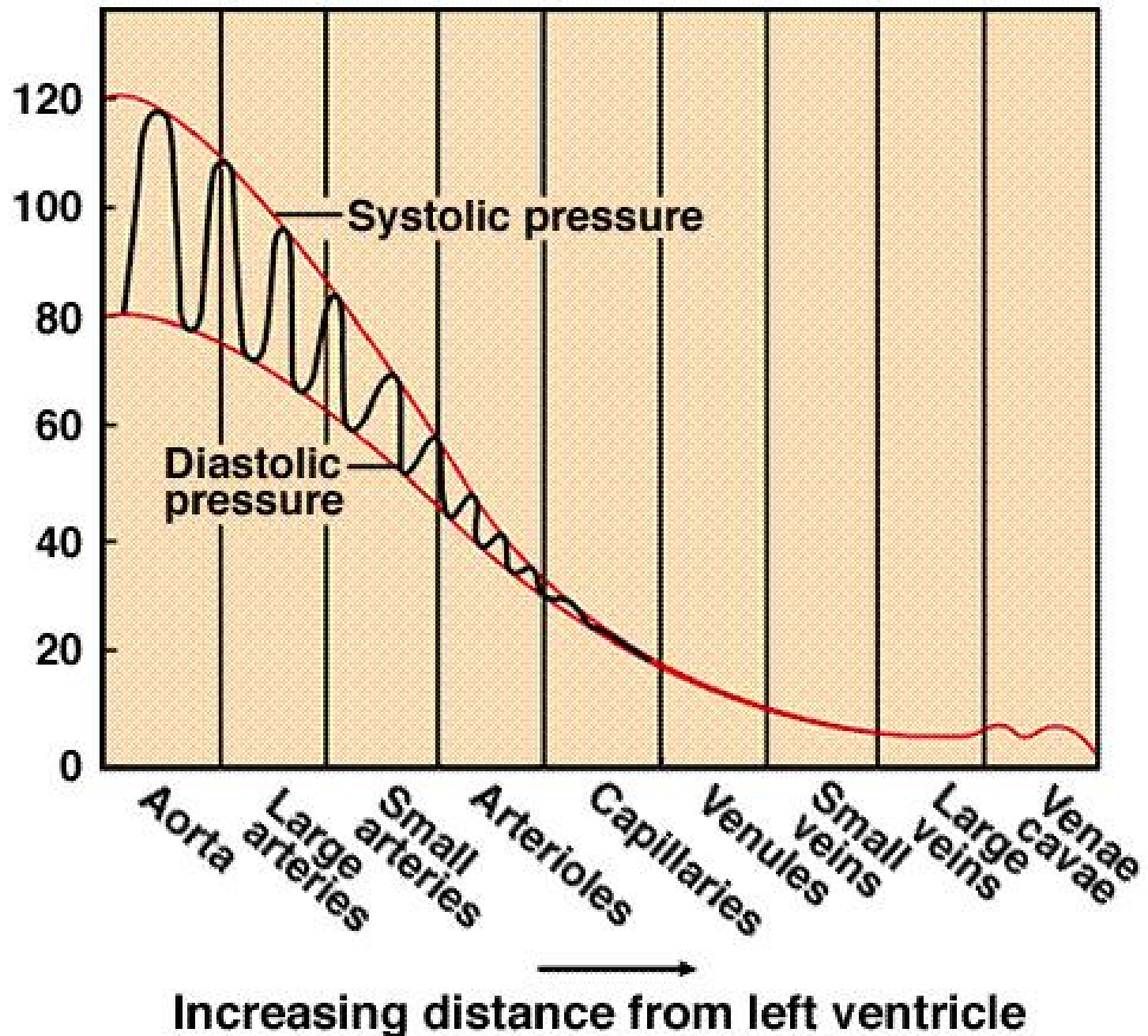


Heart pressure > cuff but cuff  
> diastolic pressure





# Blood Pressure and Distance



# CARDIOVASCULAR ANIMATIONS

## Conditions Procedures

- Atherosclerosis
- Angiography
- Heart Attack
- Angioplasty with Stent
- Stroke
- Coronary Artery Bypass Graft
- Abdominal Aortic Aneurysm
- Valve Replacement

