REVISED
8:46 am, Jan 28, 2008

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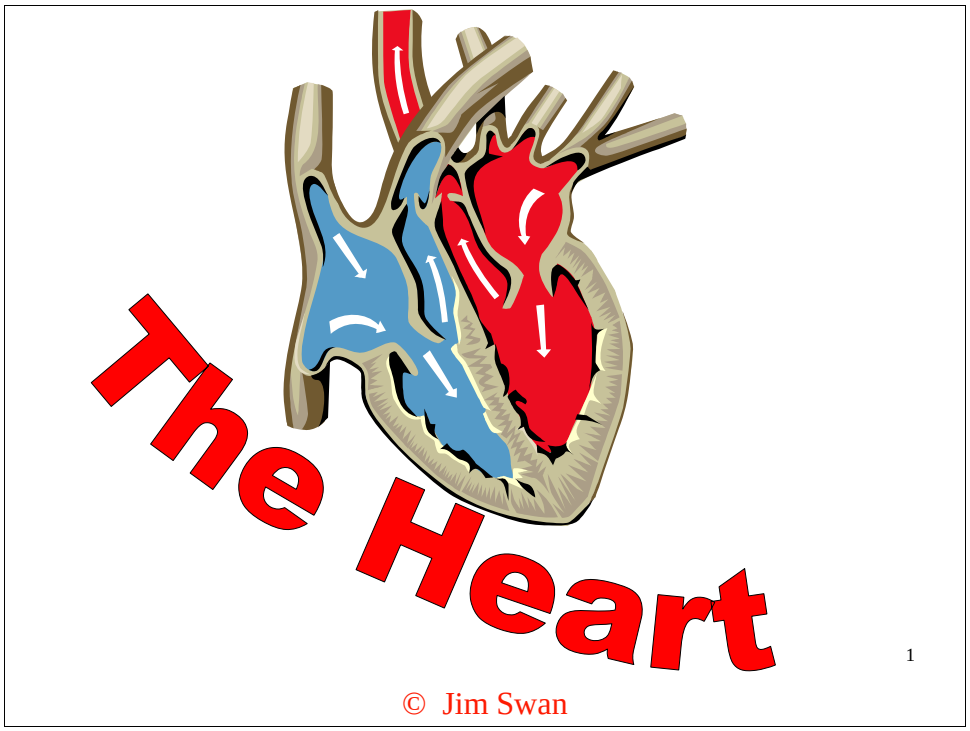

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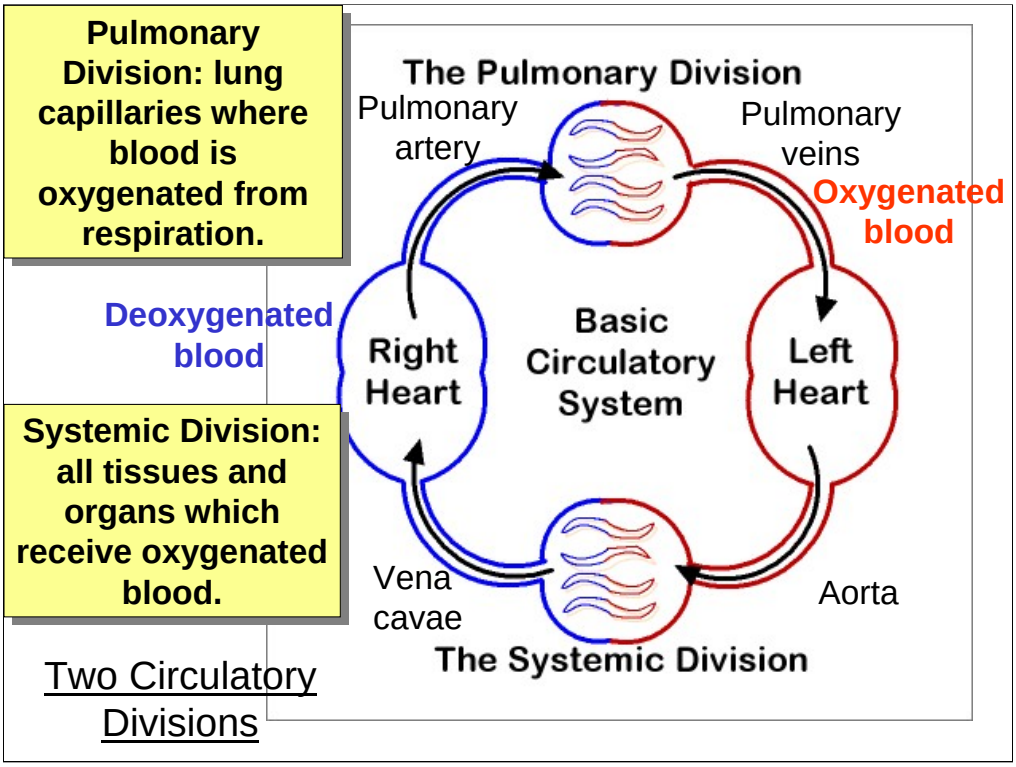
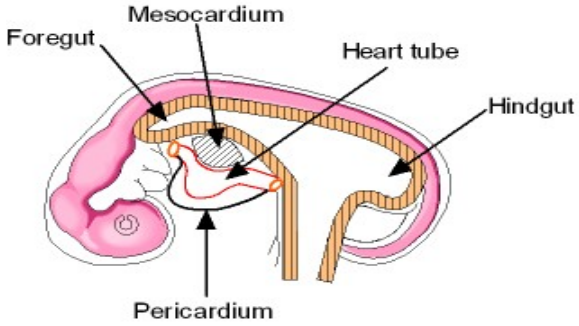
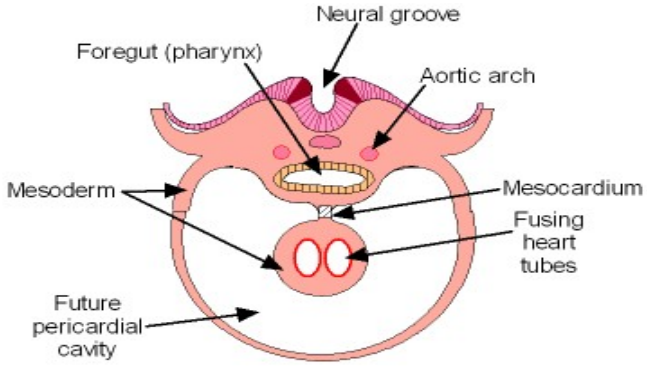


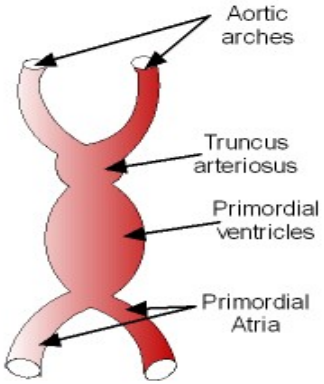
There are two divisions in the circulatory system: The **Pulmonary Division** - Lung capillaries serving structures where oxygen is obtained and carbon dioxide removed via respiration and not via the blood supply. The **Systemic Division**- All other organs and tissues where oxygen is provided by oxygenated blood in incoming arteries and carbon dioxide is carried away by outgoing veins. The heart is really two pumps: the right heart pumps de-oxygenated blood received from the systemic division to the lungs to become oxygenated, and the left heart pumps this oxygenated blood to the systemic division to deliver oxygen to the tissues and pick up carbon dioxide.



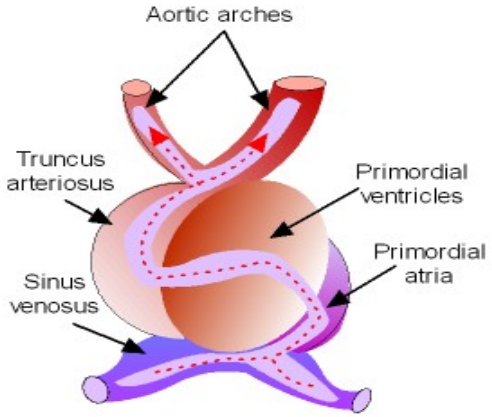
A) During the second week of embryonic development the heart consists of a pair of thin-walled muscular tubes located beneath the floor of the foregut (pharynx)



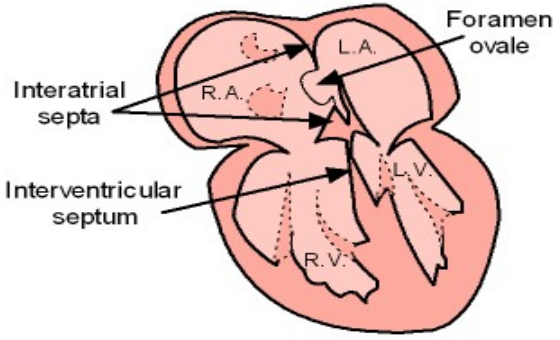
B) The mesoderm splits into a visceral and a parietal layer surrounding a space which will become the pericardial cavity.



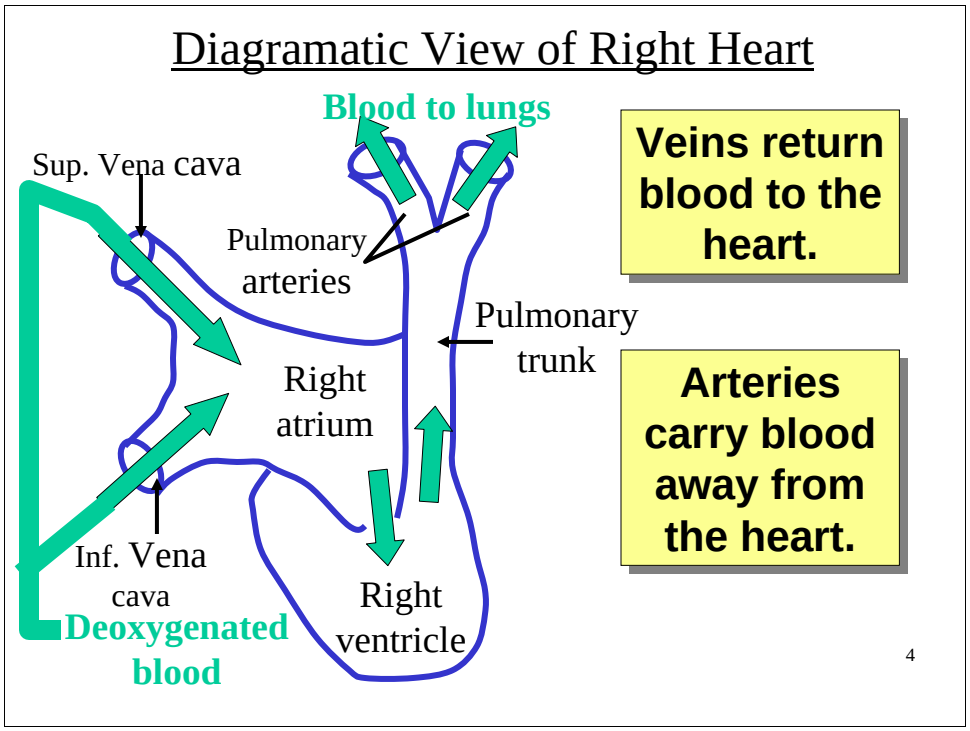
C) By the end of the third week the heart tubes have fused producing a single-chambered heart which is pumping blood through the truncus arteriosus to all parts of the body.



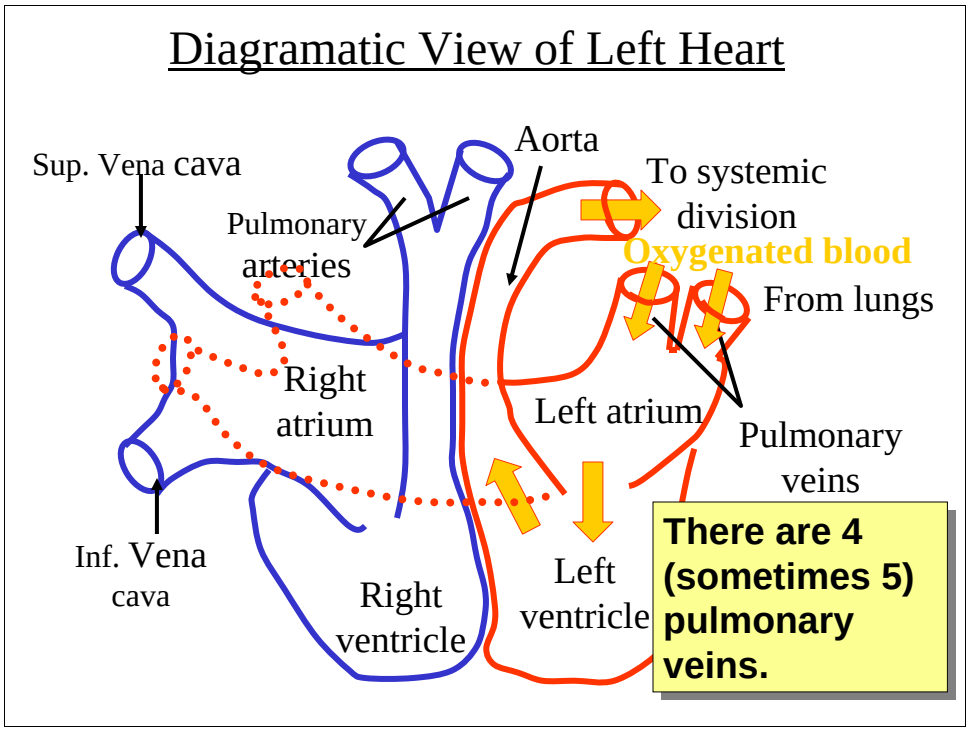
D) In Week 4 the heart elongates and curves back upon itself forming an S shape. The musculature of the chambers is beginning to differentiate them.



E) In week 5 the interatrial and interventricular septa begin to subdivide the heart into the four chambers. A gap between the two atria, called the **foramen ovale**, allows the blood to flow from right atrium to left atrium, bypassing the lungs. After birth this hole becomes the **fossa ovalis**.



The right heart is blue because it carries deoxygenated blood, receiving it from the systemic system and pumping it to the pulmonary system for oxygenation.



Left heart is red because it carries and pumps oxygenated blood. Blood enters from the pulmonary division and the left ventricle pumps it to the systemic division. Because the left atrium spreads behind the heart the right pulmonary veins are obscured in most anterior views (here shown by dotted lines). A pulmonary vein originates from each lobe of each lung, three on the right, two on the left, but usually two of the right pulmonary veins fuse before reaching the left atrium.

Heart Valves Produce One-way Blood Flow

Atrioventricular (AV) valves – prevent backflow of blood into the atria when the ventricles contract.

bicuspid valve – between left atrium and left ventricle

tricuspid valve – between the right atrium and right ventricle

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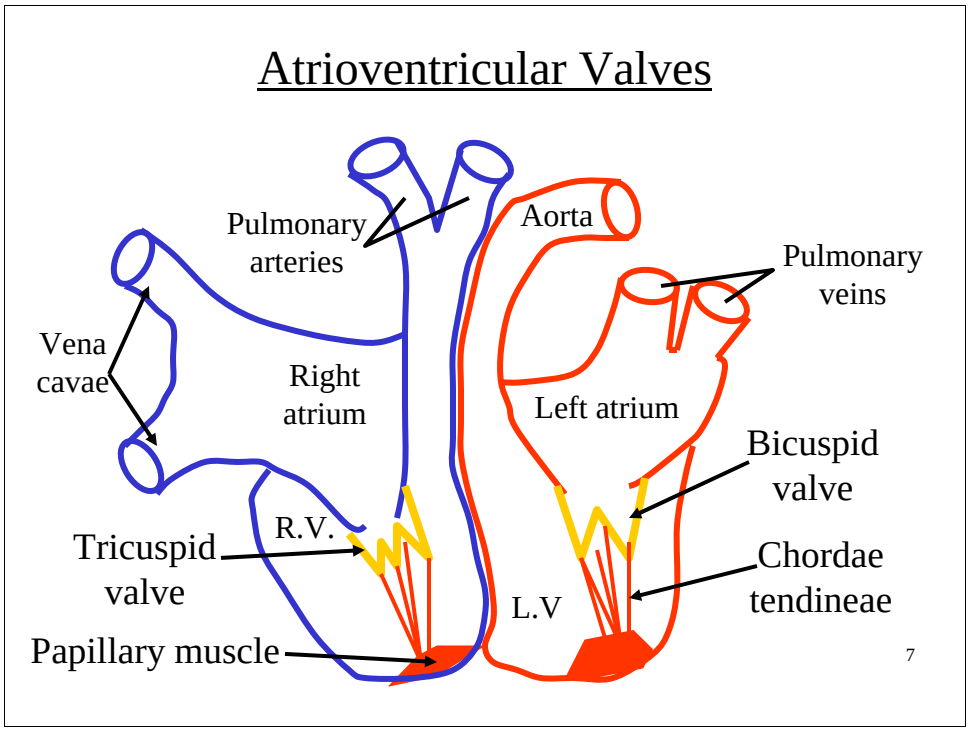
The heart pumps by squeezing, compressing and pressurizing the blood which then flows down the pressure gradient. The heart's valves force the blood to go in one direction and prevent (when working properly) backward flow.



The atrioventricular valves are found at the openings between each atrium and ventricle. They are held in place by **chordae tendineae**, which keep them from pushing inside-out. The chordae tendineae attach to muscular extensions from the ventricular wall called **papillary muscles**.

Action of the Atrioventricular Valves

Ventricle relaxes.
AV valve opens.
Blood flows through valve, filling ventricle.

AV valve closed by pressure of blood when ventricle begins contraction, blood forced into artery.

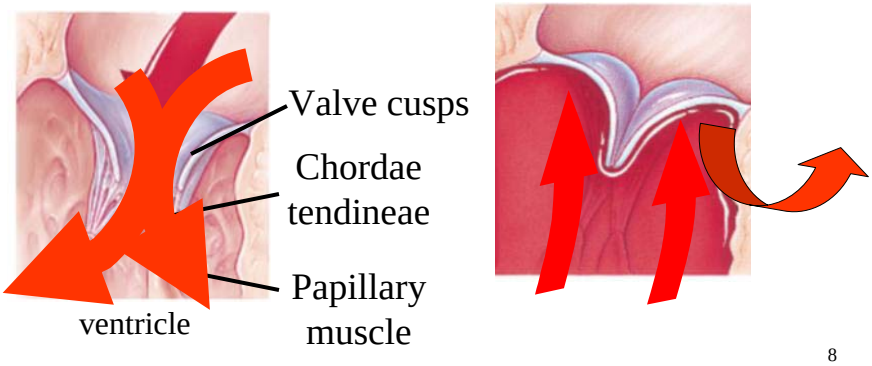


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Figure 19.9

Atrioventricular valves work in capturing blood as a parachute captures air. As blood fills the valves they press closely against one another blocking the pathway back to the atrium. The chordae tendineae act like the parachute ropes to prevent spilling of blood, or leakage. AV closing makes a “slapping” sound heard as the first heart sound. (the “lubb” in “lubb-dubb”)

Semilunar Valves

– prevent backflow of blood into the ventricles when the ventricles relax.

aortic valve – at entrance to aorta

pulmonary valve – at entrance to pulmonary trunk

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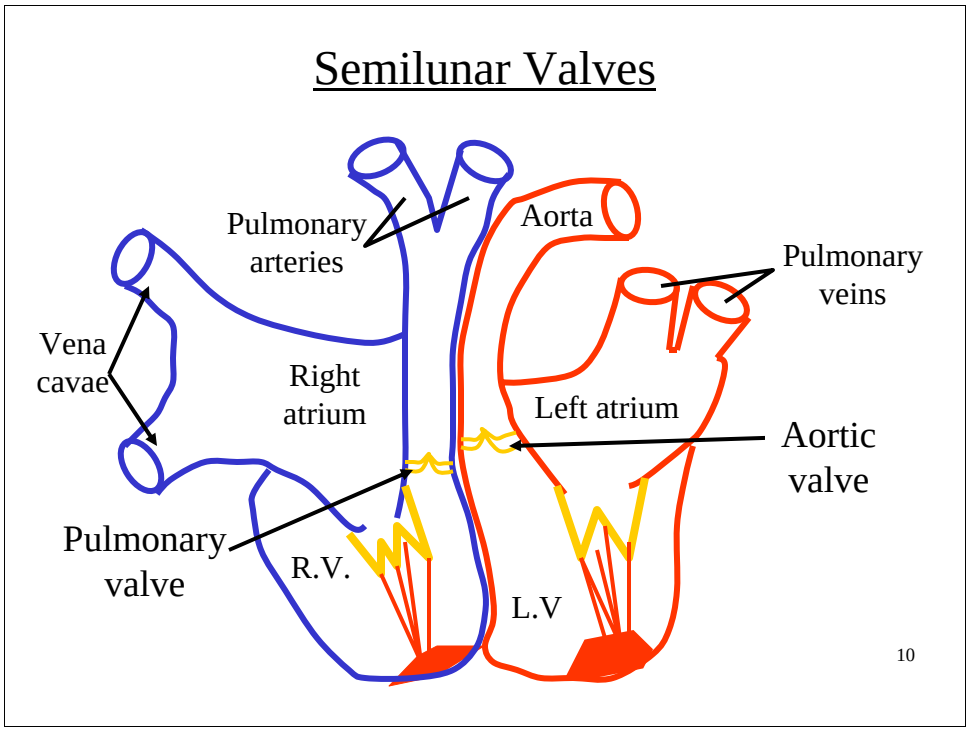
The semilunar valves also have cusps, which catch blood as it flows back toward the ventricle during ventricular systole.

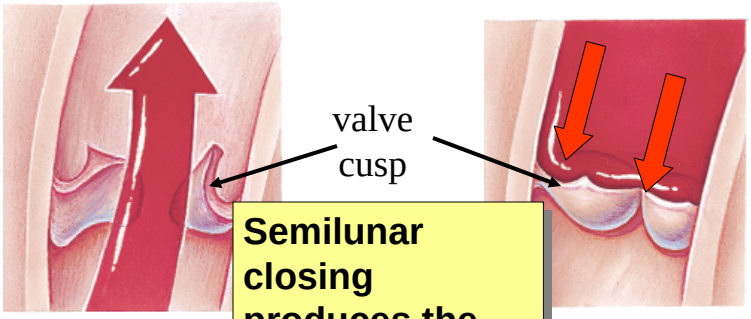


Location of the semilunar valves is at the entrance to both arteries, the pulmonary and aorta.

Action of the Semilunar Valves

Ventricle contracting, valve open, blood flows into arteries.

Ventricle relaxes, backflow of blood closes valve.



valve cusp

ventricle

ventricle

Semilunar closing produces the second heart sound.

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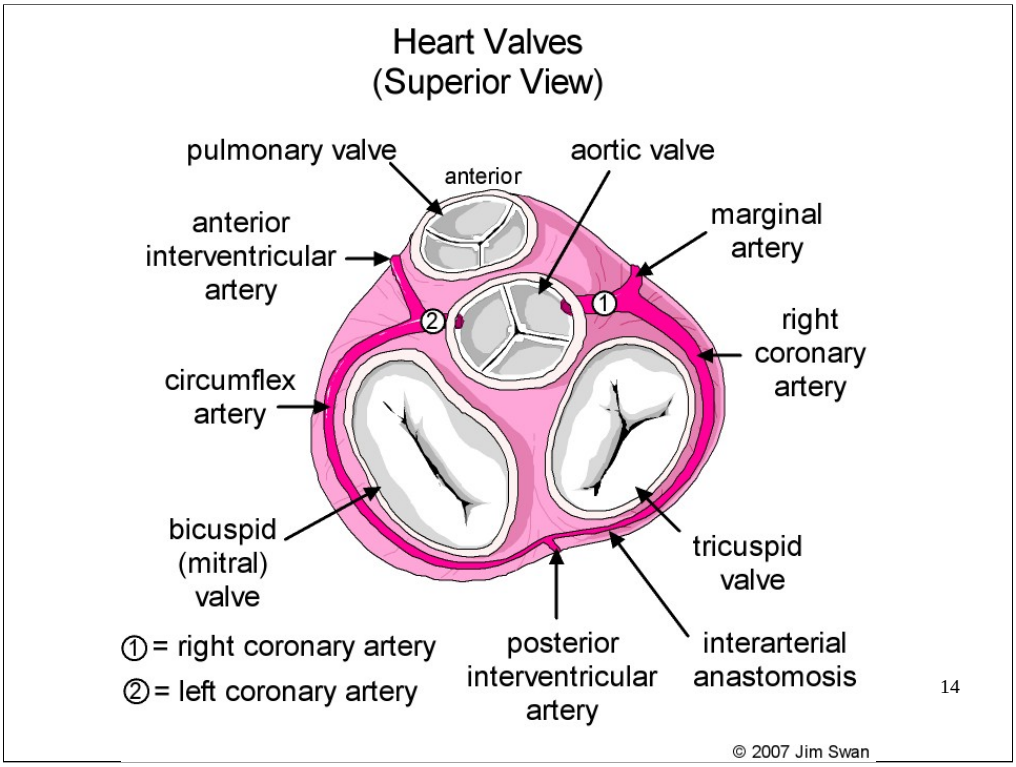
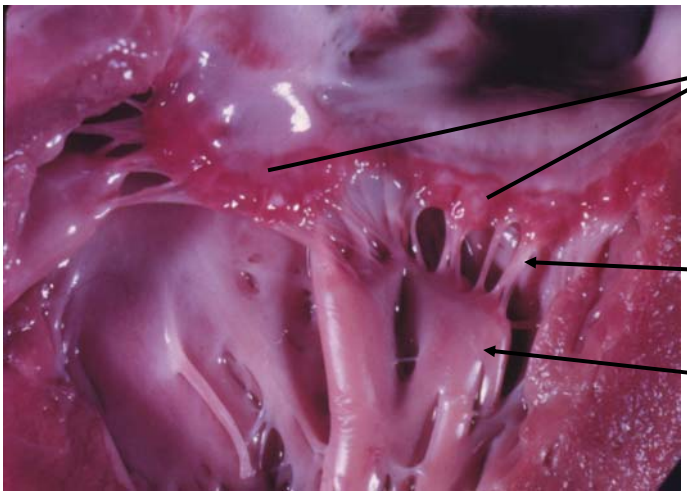
Semilunar valves contain three cusps each. When the ventricles contract these valves open as blood rushes through, and the cusps are pushed back against the arterial wall. When the ventricles relax blood is pulled back toward the ventricles, filling the cusps, and closing the semilunar valves.



Valves are shown from above. Note the origin of the coronary arteries behind the aortic semilunar valves. Also note the anastomosis between them which produces collateral circulation. Slight gaps can allow small amounts of blood to leak through, creating sound known as a heart murmur. Most murmurs are insignificant, but if the leakage is significant blood flow will be impaired and heart valve replacement will be necessary.

AV Valve



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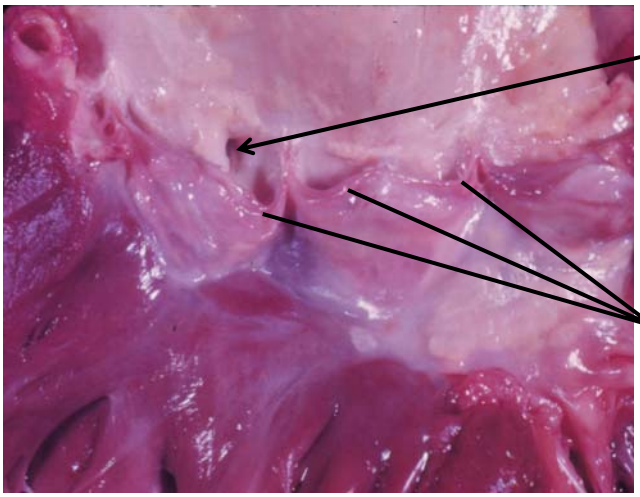
Here you see the cusps of the bicuspid (mitral) valve with their chordae tendineae and attach papillary muscles.

The Aortic Semilunar Valve



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Behind two of the semilunar valve cusps are the openings into the coronary arteries. These arteries will, therefore, fill when ventricular diastole fills the cusps.

Terms:

stenosis - constriction and narrowing of a valve, usually caused by disease.
prolapse - a stretched valve resulting in incomplete closure and producing leakage.
heart murmur - the sound produced by a leaking valve. Most murmurs are inconsequential but some indicate severe inadequacy of the valve.
regurgitation - the backflow of blood due to an incompetent (ineffective) valve.

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All of these conditions make valves inefficient, and can lead to cardiac congestion and ultimately heart failure.

Assignment

Trace the blood flow through the heart listing in order all vessels, chambers, and valves.

Start with deoxygenated blood arriving from the systemic division, and end with oxygenated blood entering the systemic division.

Approximately 15 items.

We will go over the flow in the next class.

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This is a study assignment which will help you to conceptualize blood flow through the heart. Specify at each point whether the blood is oxygenated or deoxygenated.

1) Deoxygenated blood from systemic division

2) Superior and inferior vena cavae

3) Right atrium

4) Tricuspid valve

5) Right ventricle

6) Pulmonary valve

7) Pulmonary arteries

8) Lungs

9) Pulmonary veins

10) Left atrium

11) Bicuspid valve

12) Left ventricle

13) Aortic valve

14) Aorta

15) Oxygenated blood to systemic division

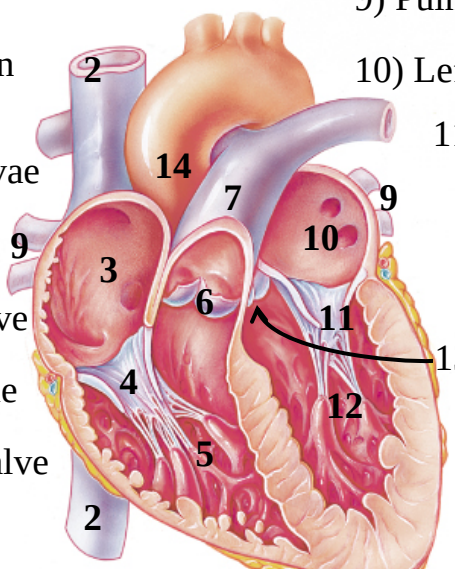
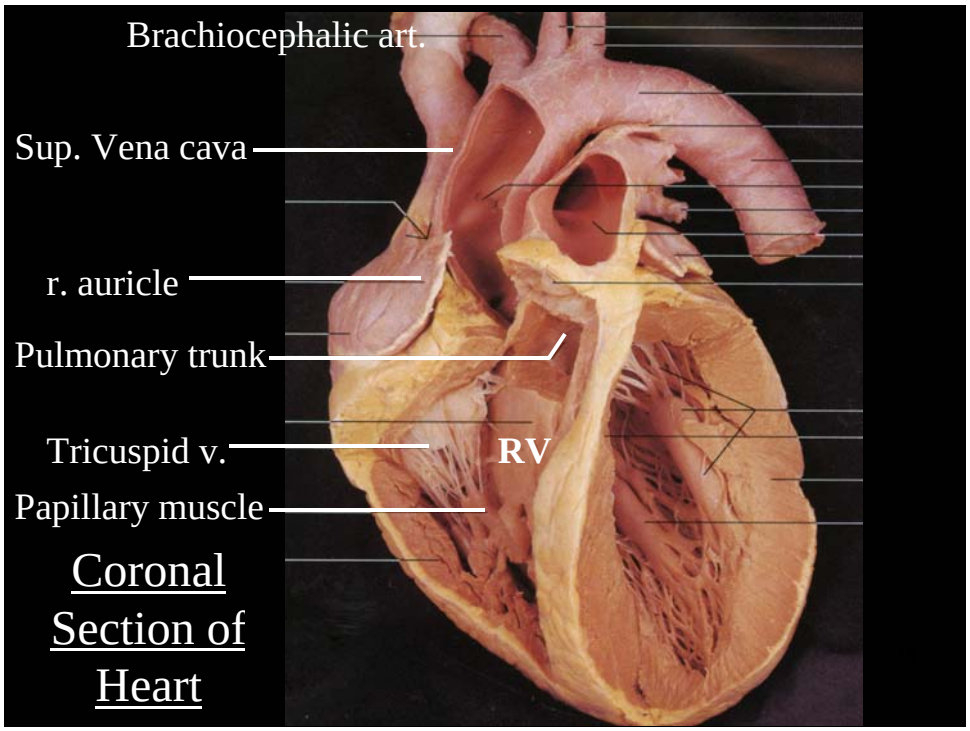
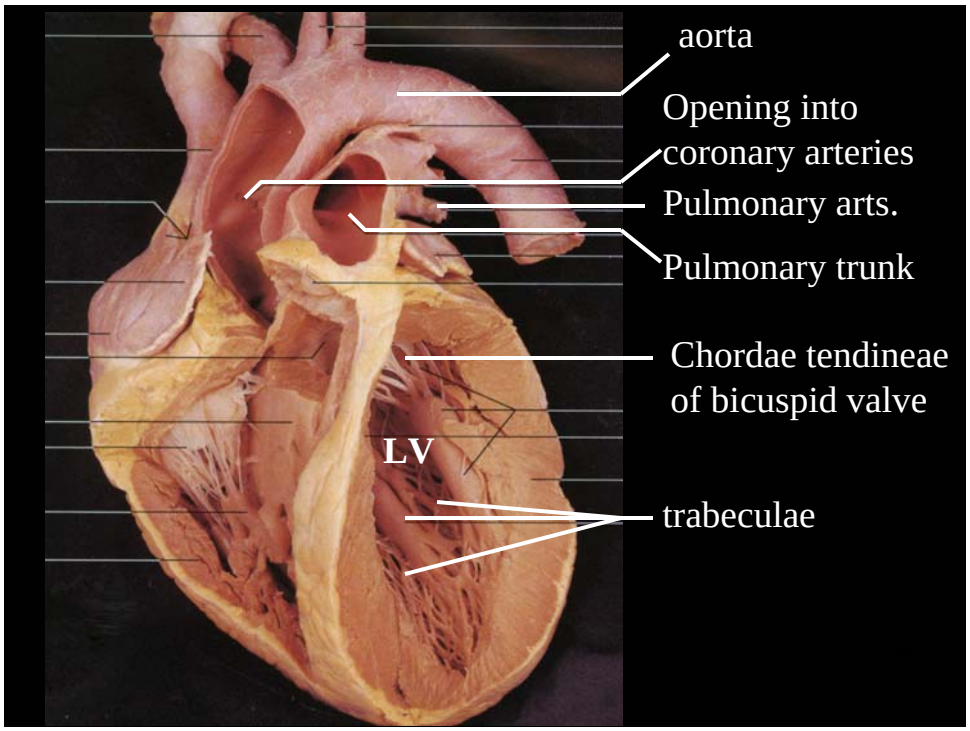


Figure 18.4 e

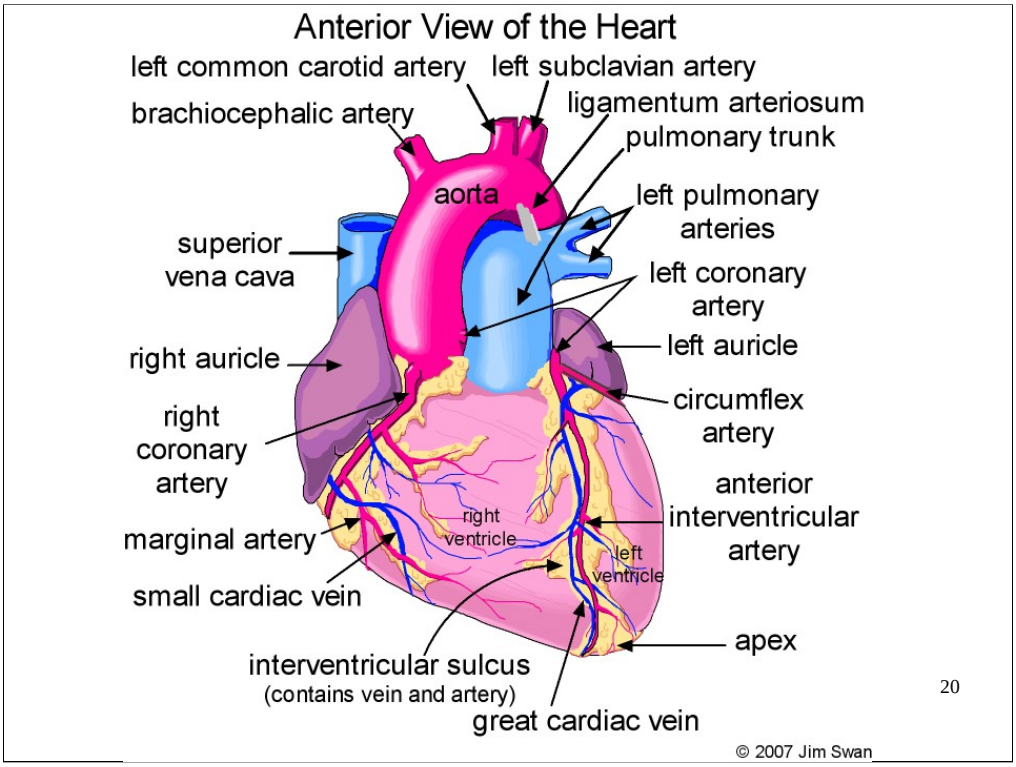
Here is the scheme for the assignment from the previous slide. If you did it yourself, excellent. If you didn't, then just do it.



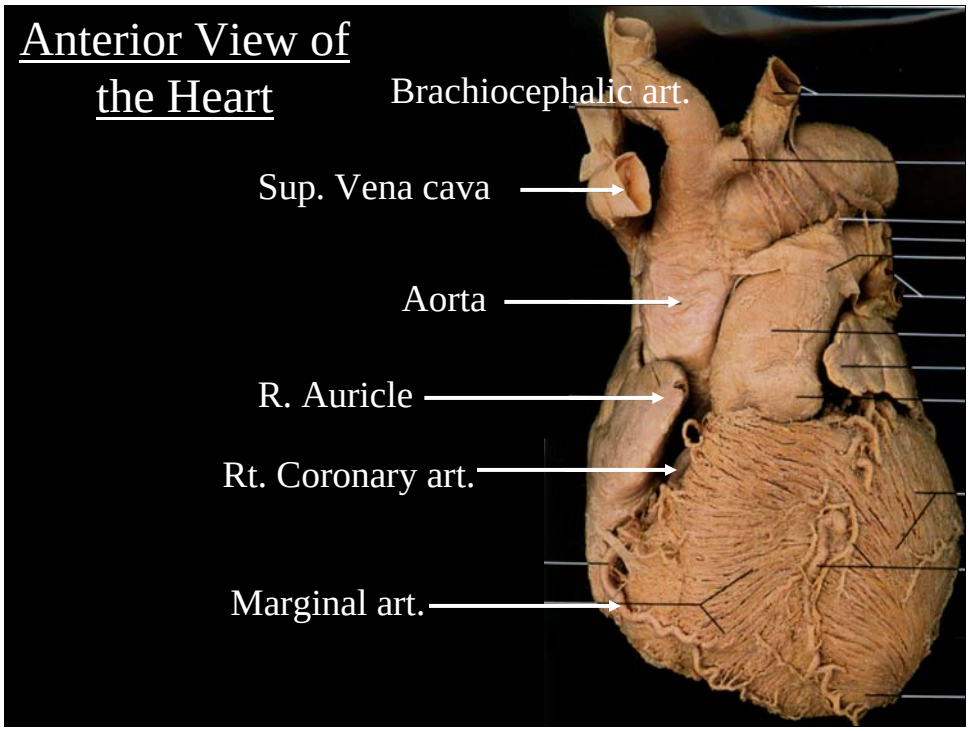
In this actual cadaver dissection note the differences between the left and the right chambers.



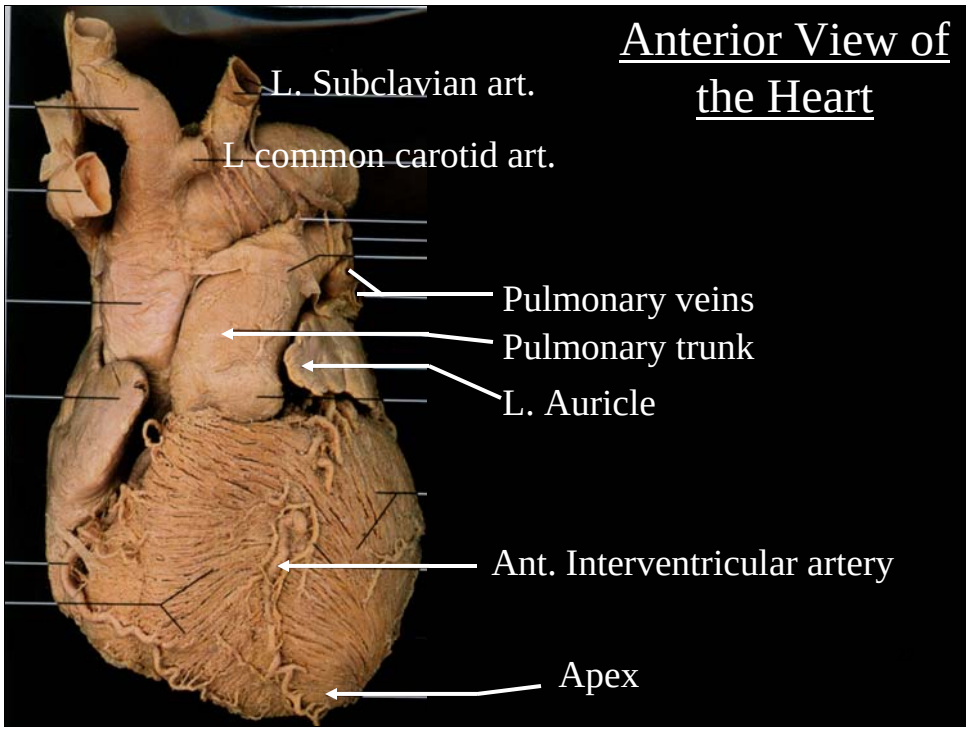
Note the greater thickness of the left ventricular myocardium. The **trabeculae** form a network for the ventricular myocardium which contain the **purkinje fibers**.



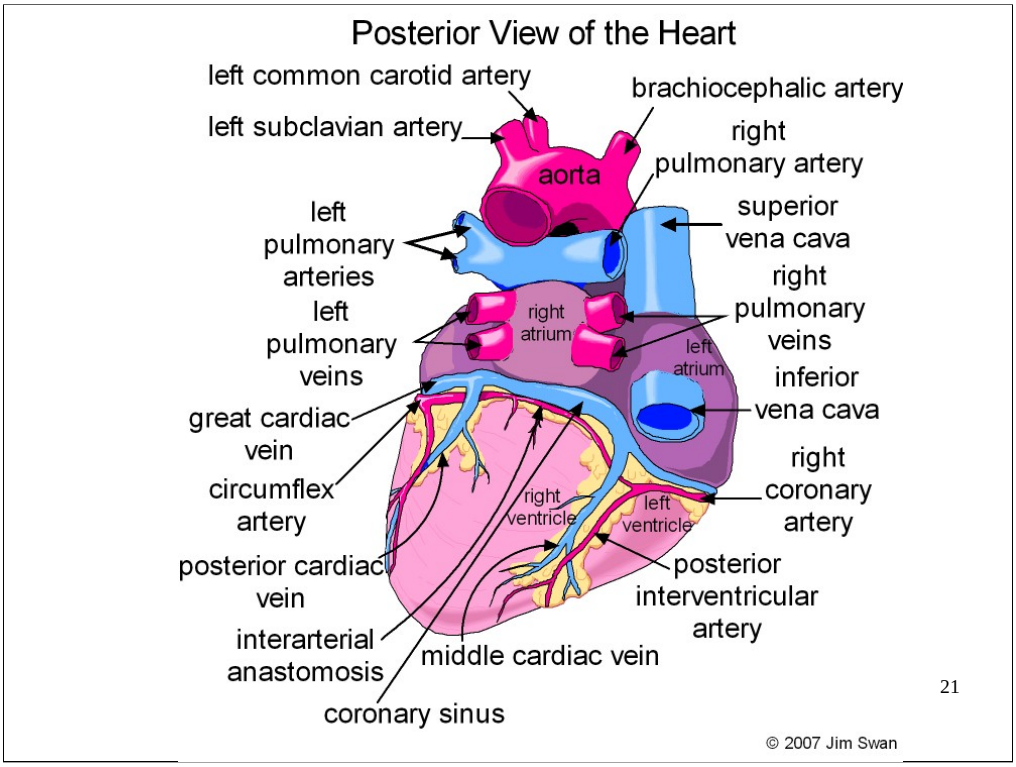
Note that the coronary arteries and veins run through the sulci or grooves between the atria and ventricles and between the ventricles and one another.



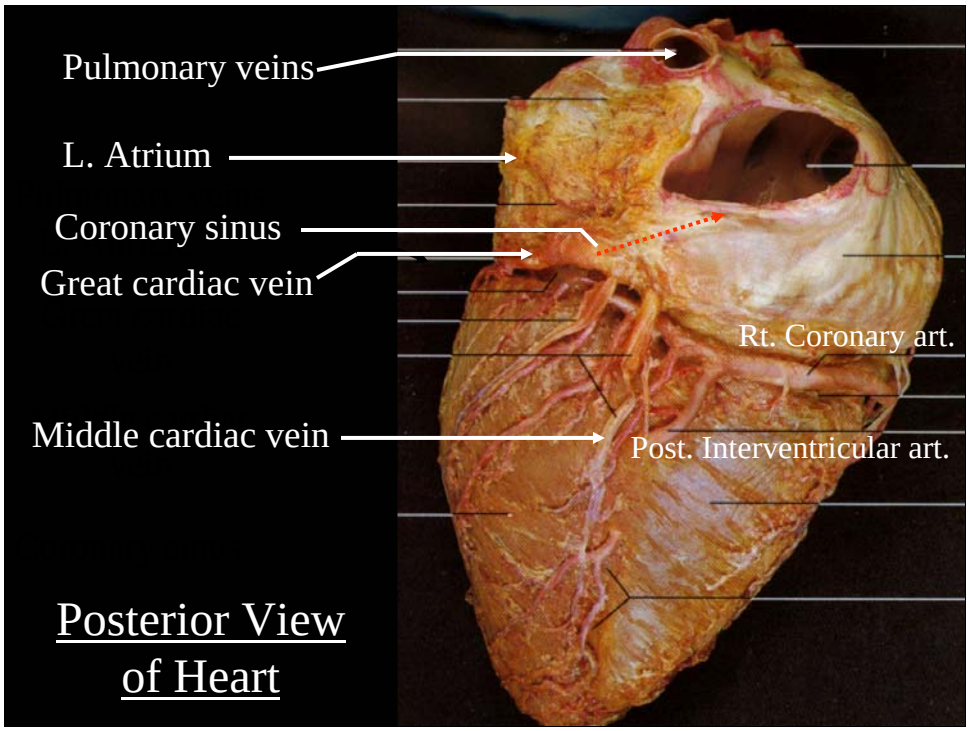
The **visceral pericardium** (**epicardium**) has been removed exposing the surface of the heart muscle (**myocardium**)



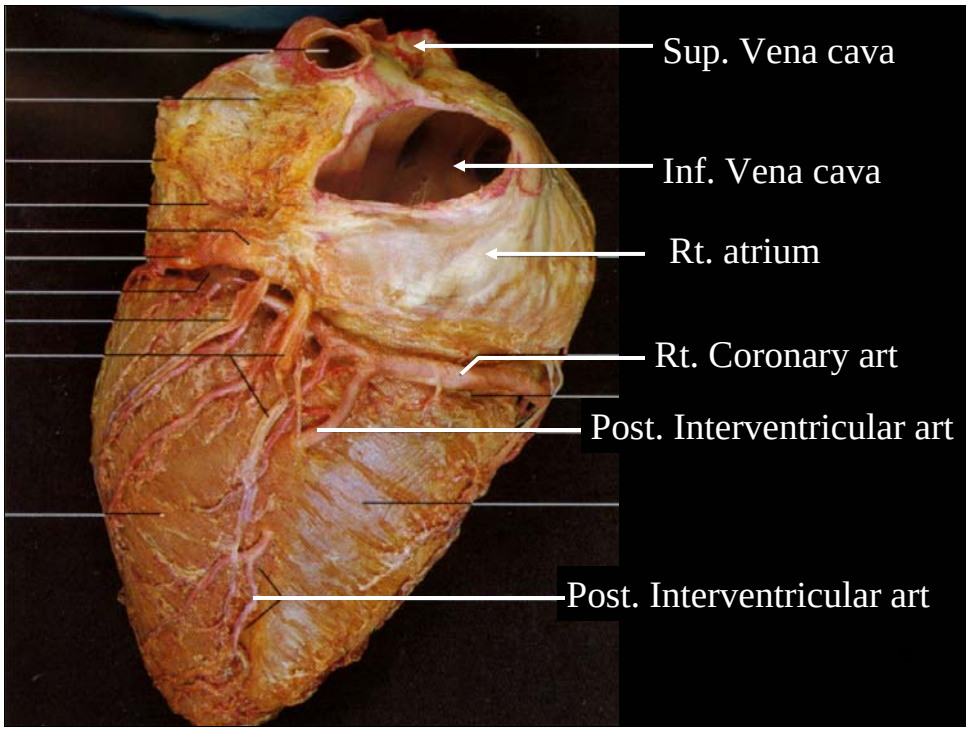
Removal of the visceral pericardium and its fatty tissue exposes the coronary arteries and veins, normally embedded in the pericardial tissue. Note that some of these descend into the ventricular muscle.



All coronary veins ultimately drain into the great coronary vein and coronary sinus which enters the right atrium. The coronary veins are the only systemic veins which don't enter the superior or inferior vena cava.



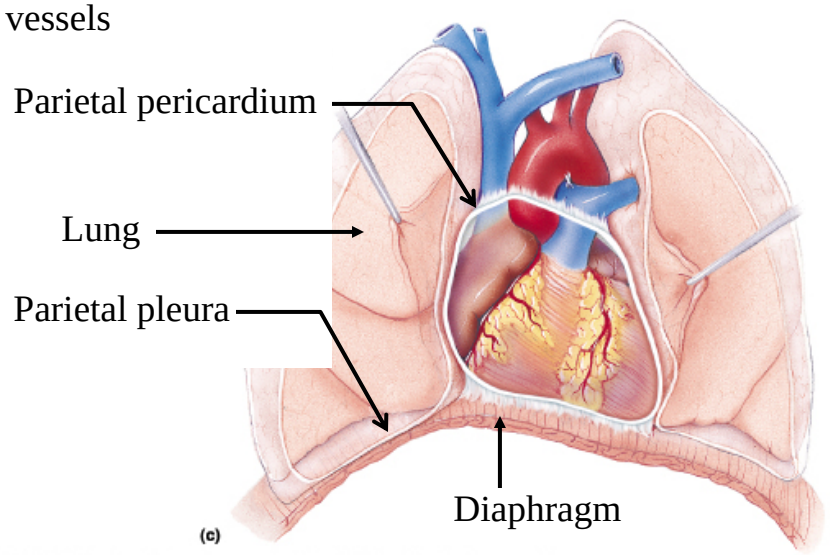
Coronary and other veins are distinguished from their corresponding arteries by being flatter and thinner. Arteries and veins run alongside one another in the grooves (sulci) of the heart.



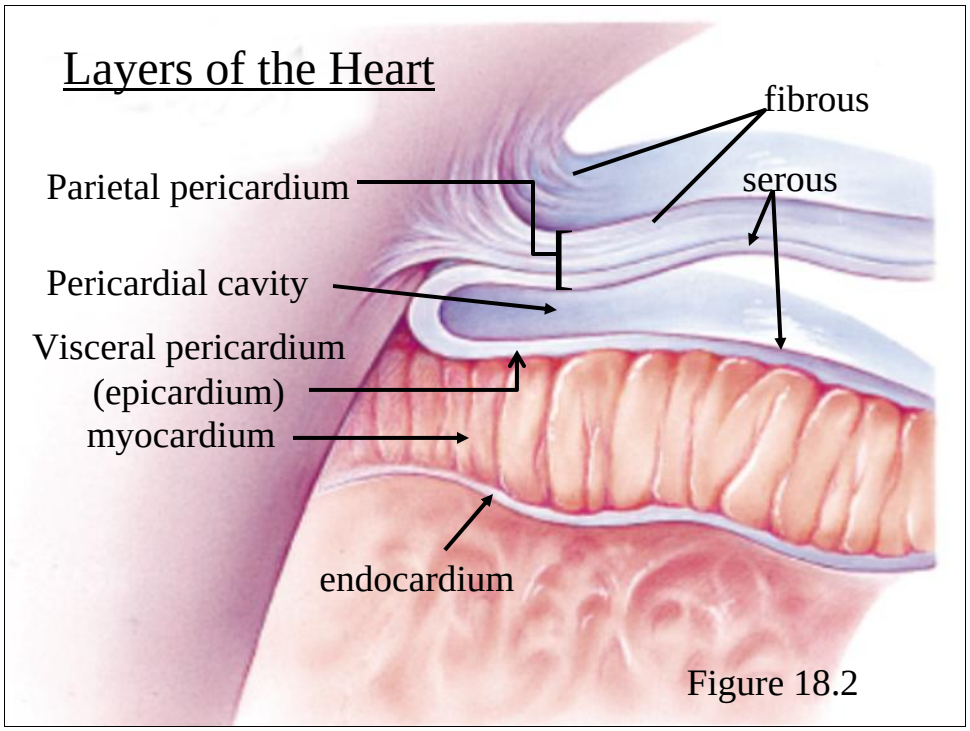
Note the thinness of the atrial wall, and how the **posterior interventricular artery** descends into the ventricular myocardium. The arteries anastomose to provide collateral circulation to the myocardium.

Figure 18.1 Heart, Anterior Exposure

Mediastinum – portion of thorax containing heart and great vessels



The heart is located in the **mediastinum**, a section of the thoracic cavity. It is surrounded by a double-layered membrane, the pericardium. The outer layer of the pericardium, the **parietal** layer, attaches to surrounding structures including the large vessels and the diaphragm. The inner layer, the **visceral** pericardium (see next slide) attaches to the surface of the heart. Between these layers is serous fluid, which lubricates the membranes and prevents tearing and abrasion when the heart beats and moves due to body movement.



The pericardium is a double layered membrane with serous membranes lying next to one another. The serous fluid produced serves to lubricate the heart against tearing and abrasion. The endocardium is a smooth endothelial lining throughout the entire cardiovascular system.

The Cardiac Cycle

- the sequence of events in one complete heartbeat.

Terms:

systole - the contraction phase; unless otherwise specified refers to left ventricle, but each chamber has its own systole.

diastole - the relaxation phase; unless otherwise specified refers to left ventricle, but each chamber has its own diastole.

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First we consider the basic steps of the cardiac cycle, then we will examine each step in more detail.

Major Events in The Cardiac Cycle

1) **quiescent period** - period when all chambers are at rest and filling. 70% of ventricular filling occurs during this period. The AV valves are open, the semilunar valves are closed.

2) **atrial systole** - pushes the last 30% of blood into the ventricle.

3) **atrial diastole** - atria begin filling.

This occurs nearly simultaneously with the next event…

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4) **ventricular systole** – ventricles contract, first closing the AV valves and causing the first heart sound…

then the semilunar valves open permitting ventricular ejection of blood into the arteries.

5) **ventricular diastole** - As the ventricles relax the semilunar valves close first producing the second heart sound, then…

the AV valves open allowing ventricular filling.

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Cardiac Output

Minute Volume = Heart Rate X Stroke Volume

Heart rate, HR at rest = 65 to 85 bpm (widest range usually quoted)

Each heartbeat at rest takes about .8 sec. of which .4 sec. is quiescent period.

Stroke volume, **SV** at rest = 60 to 70 ml.

C.O. at rest = 70 bpm X 70 ml/beat = 4900 ml/min/vent.

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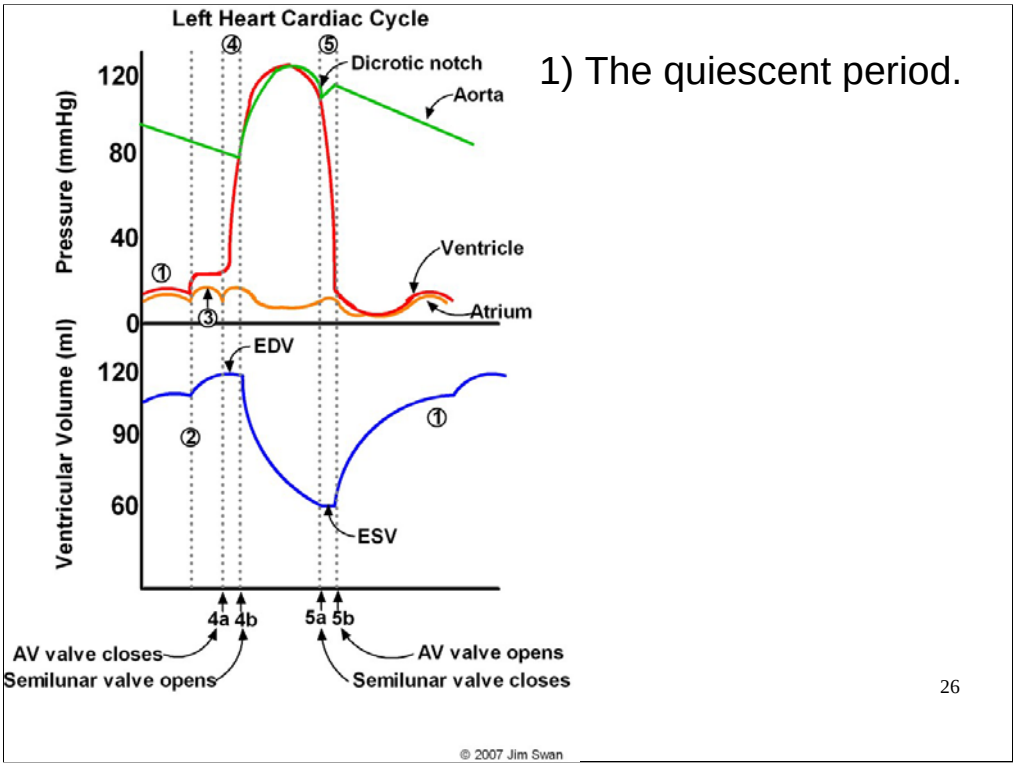
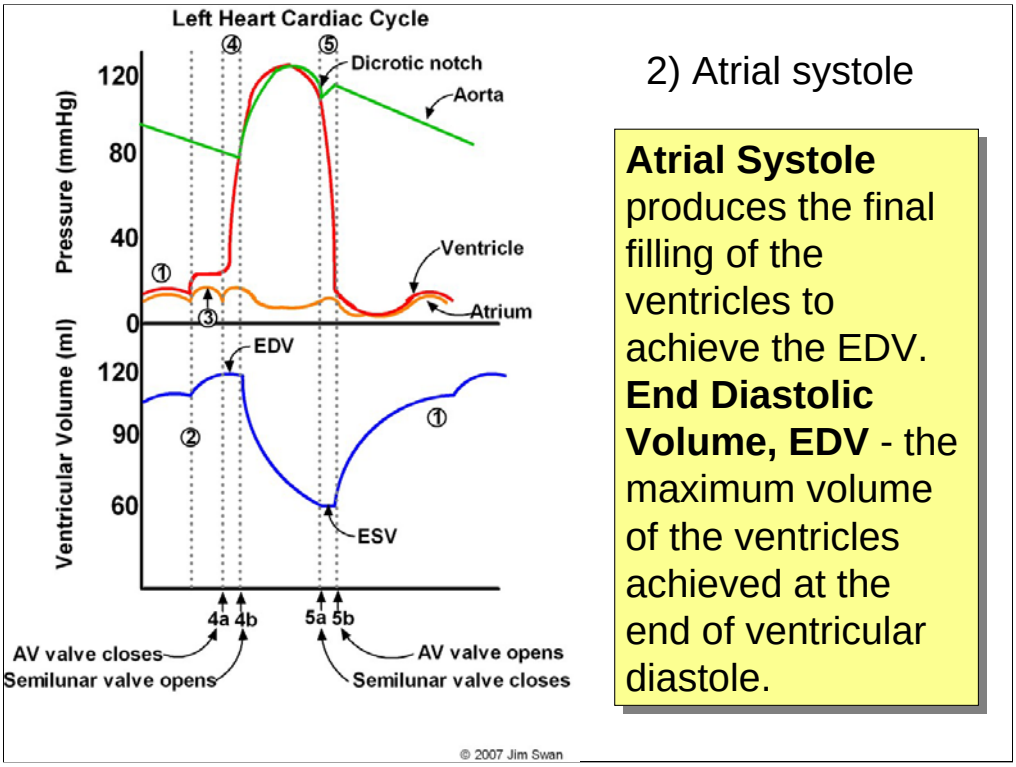
The heart can increase both rate and volume with exercise. Rate increase is limited due to necessity of minimum ventricular diastolic period for filling. Upper limit is usually put at about 220 bpm. Maximum heart rate calculations are usually below 200. Target heart rates for anaerobic threshold are about 85 to 95% of maximum.



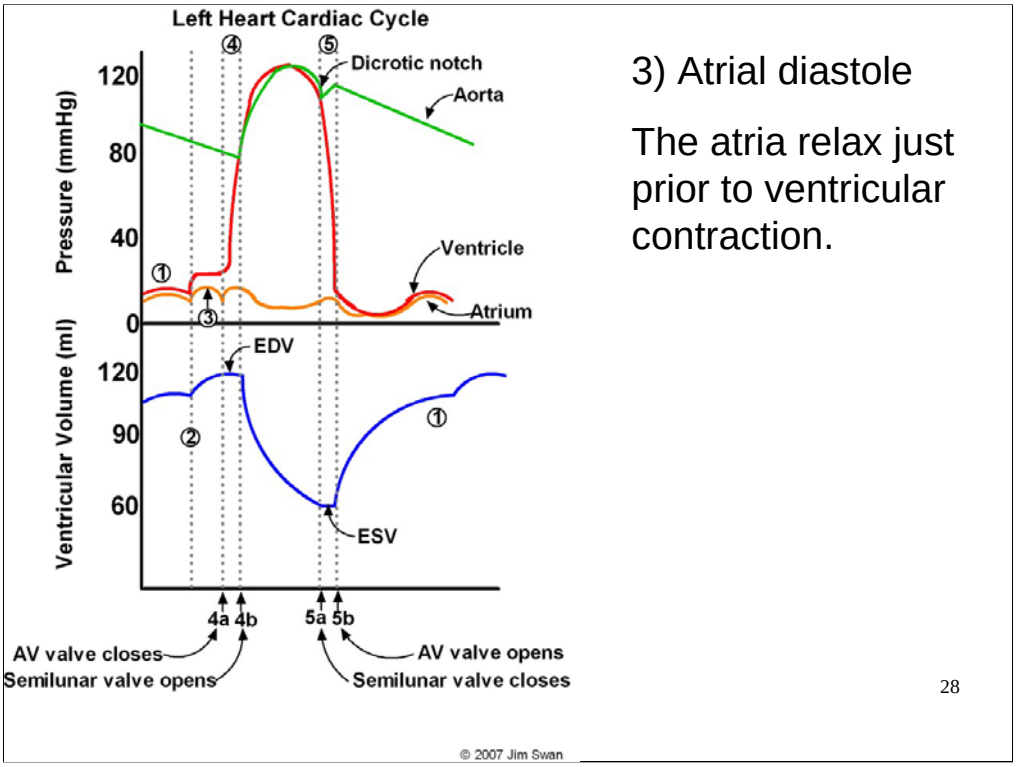
During the Quiescent Period - all chambers relaxed and filling - the AV valves are open and the semilunar valves are closed.



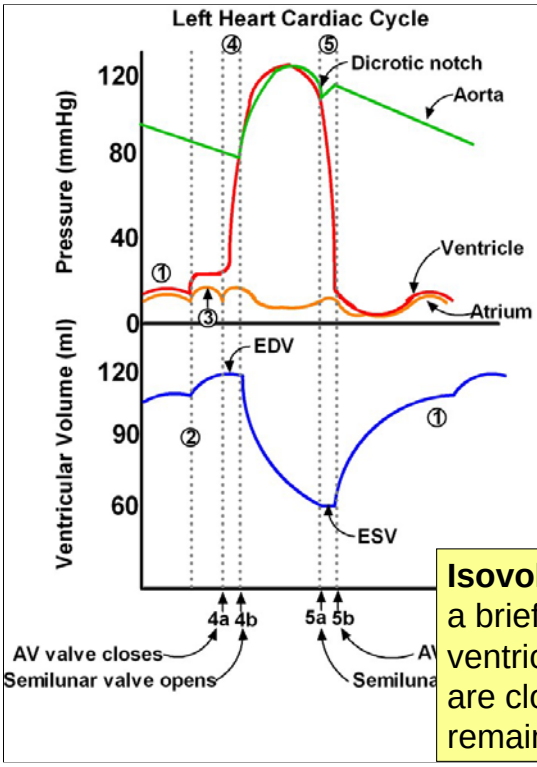
2) Atrial systole

Atrial Systole produces the final filling of the ventricles to achieve the EDV. **End Diastolic Volume, EDV** - the maximum volume of the ventricles achieved at the end of ventricular diastole.

An increase is seen in the atrial pressure curve. Atrial contraction produces the final filling of the ventricles, producing an increase in ventricular volume. The maximum volume of the ventricle seen as a result is the End Diastolic Volume, or EDV. Why does ventricular pressure also show an increase?



The atria relax just before the ventricles begin to contract. This helps to pull the AV valves closed.



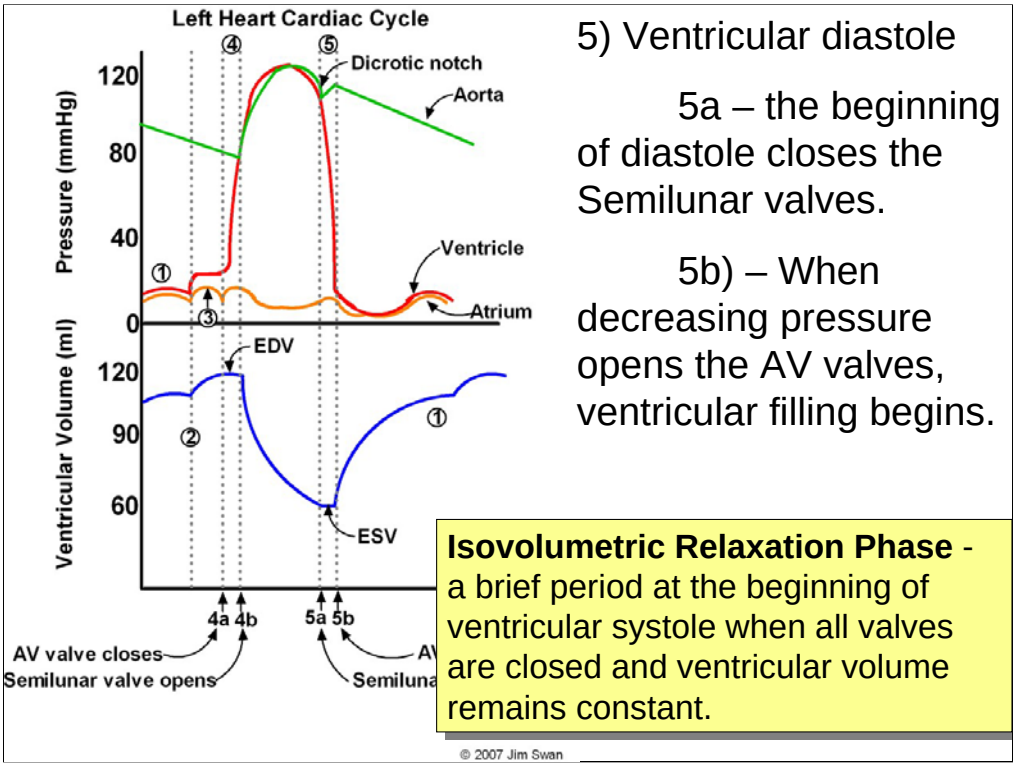
4) Ventricular systole

4a – the beginning of systole closes the AV valves.

4b) – When increasing pressure opens the semilunar valves, ejection of blood occurs from the ventricles.

Isovolumetric Contraction Phase - a brief period at the beginning of ventricular systole when all valves are closed and ventricular volume remains constant.

Ventricular systole occurs in two parts: a) pressure increases to close the AV valves; b) pressure increase opens the semilunar valves producing the ejection phase. The period of time between a and b is called the isovolumetric contraction phase.



5) Ventricular diastole

5a – the beginning of diastole closes the Semilunar valves.

5b) – When decreasing pressure opens the AV valves, ventricular filling begins.

Isovolumetric Relaxation Phase - a brief period at the beginning of ventricular systole when all valves are closed and ventricular volume remains constant.

Ventricular diastole occurs in two parts: a) pressure decreases to close the semilunar valves; b) pressure continues to decrease, opening the AV valves producing the ejection phase. The period of time between a and b is called the isovolumetric relaxation phase. The closing of the semilunar valves produces a pressure wave, known as the dicrotic notch, seen on a pulse wave .

Additional Terms

SV / EDV = Ejection Fraction

Normally around 50% at rest and will increase during strenuous exercise in a healthy heart. Well trained athletes may have ejection fractions approaching 70% in the most strenuous exercise.

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Preload - This is the pressure at the end of ventricular diastole, the beginning of ventricular systole. It is roughly proportional to the End Diastolic Volume (EDV), i.e. as the EDV increases so does the preload of the heart.

Afterload - This is the pressure at the end of ventricular systole and is related to the resistance to blood flow. It is roughly proportional to the End Systolic Volume (ESV). When the peripheral resistance increases so does the ESV and the afterload of the heart.

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The difference between preload and afterload is a measure of the heart's efficiency.

$$\text{Blood Flow} \sim \frac{\Delta \text{ blood pressure}}{\text{Resistance to blood flow}}$$

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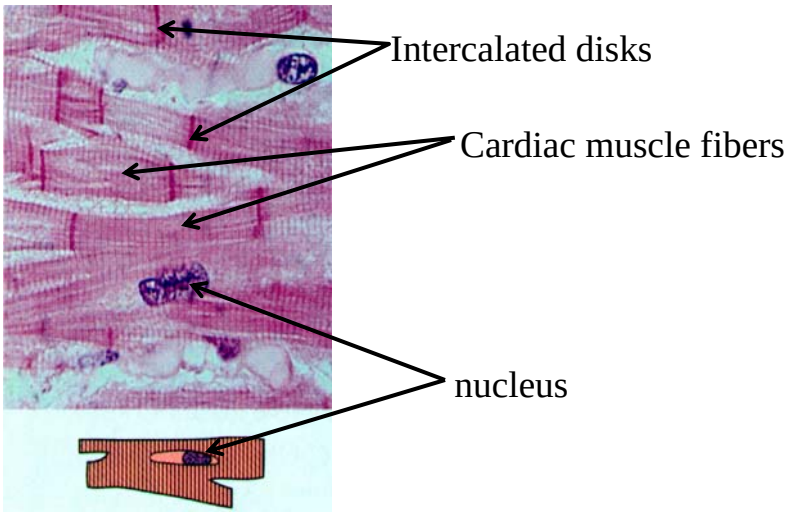
Blood flow is directly proportional to the pressure gradient over a section of a blood vessel, and inversely proportional to the resistance to flow. Resistance is produced by friction along the vascular wall, and is increased with vasoconstriction, atherosclerosis, and hypertension.

Cardiac Muscle Structure



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Cardiac muscle cells are faintly striated, branching, mononucleated cells, which connect by means of intercalated disks to form a functional network. Intercalated disks are anchoring structures containing gap junctions which allow ions and electrical impulses to pass between cells. The action potential travels through all cells connected together forming a functional syncytium in which cells function as a unit.

Cardiac Muscle Characteristics

branched - connects to other cells through intercalated disks to form a network called a syncytium.

intercalated disks - gap junction intercellular connections which allow the impulse to pass to all cells connected to form the syncytium.

syncytium - a connected network of cells which function as a unit.

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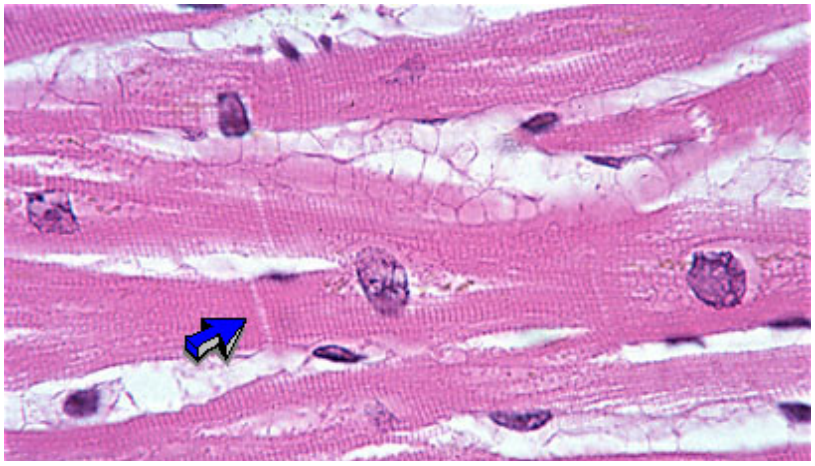
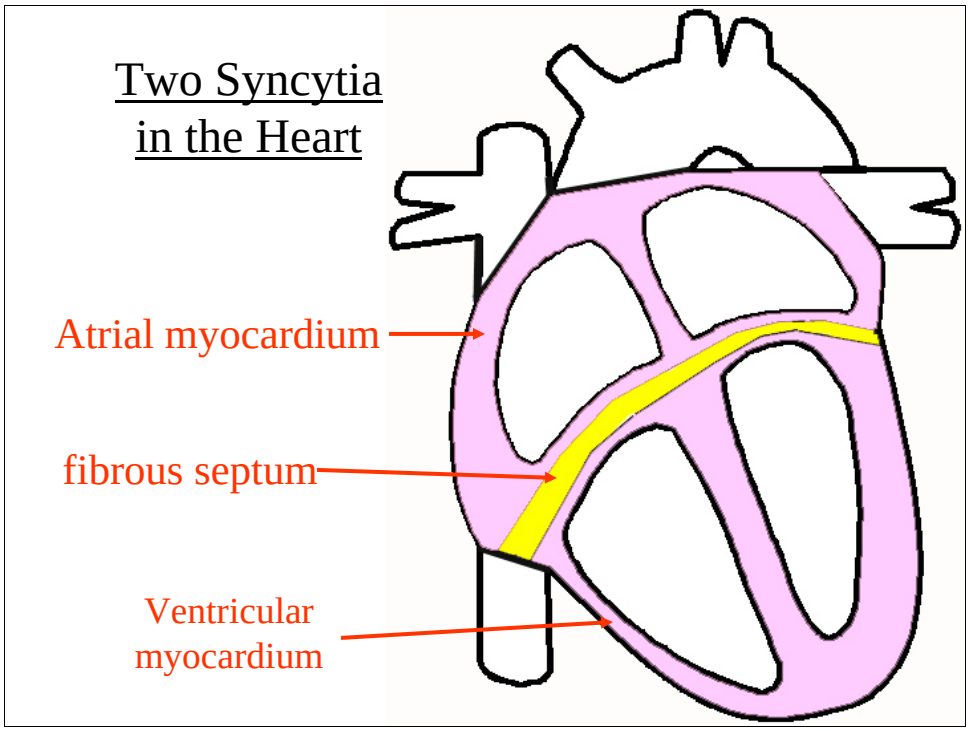
Another Example of Cardiac Muscle



Intercalated disk (blue arrow)

Note the branching nature of the tissue, with cells connecting to other cells by means of intercalated disks. Note the faint striations, an indication of the similarity of cell structure to that of skeletal muscle.



The heart has two syncytia (pleural of syncytium), the atrial myocardium is one, the ventricular myocardium is the other. They are separated from one another by a **fibrous septum**. An impulse cannot pass from one to another directly, but must travel through the heart's conduction system.

Electrical Characteristics of Cardiac Muscle

A small fraction of cardiac muscle fibers have **myogenicity** and **autorhythmicity**.

Myogenicity is the property of spontaneous depolarization and impulse generation.

Autorhythmicity - the natural rhythm of spontaneous depolarization.

Contractility - like skeletal muscle, most cardiac muscle cells respond to stimuli by contracting. Autorhytmic cells have minimal contractility.

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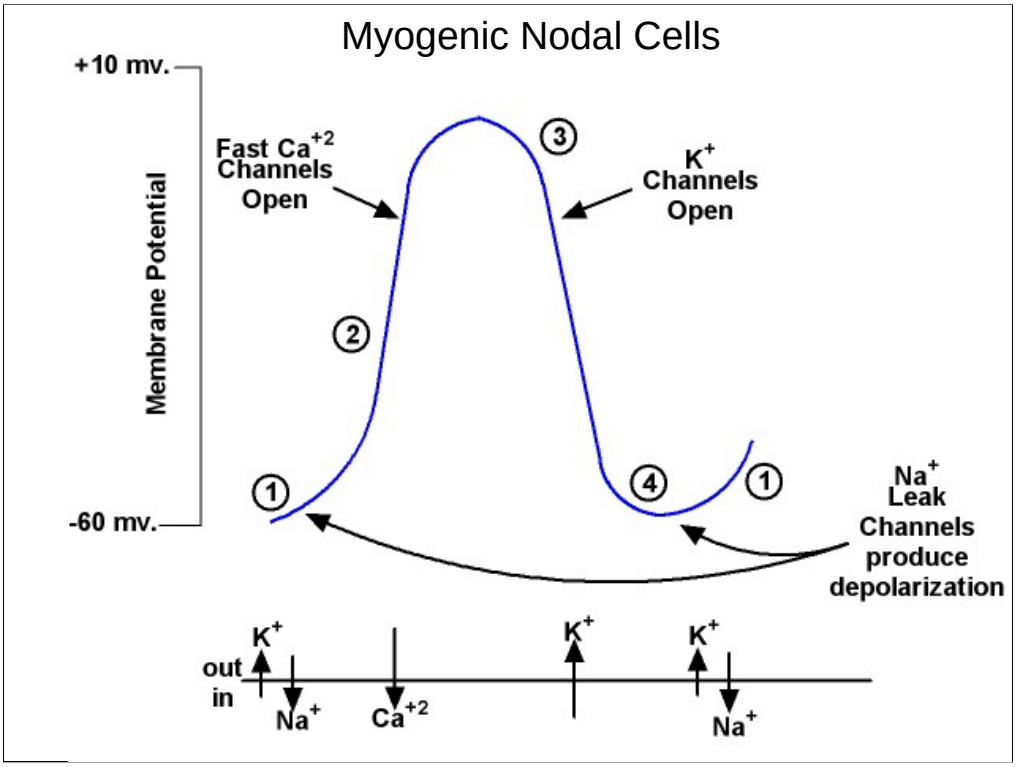
Unlike skeletal muscle, cardiac muscle contains two types of cells, the myogenic cells, and the contractile cells.



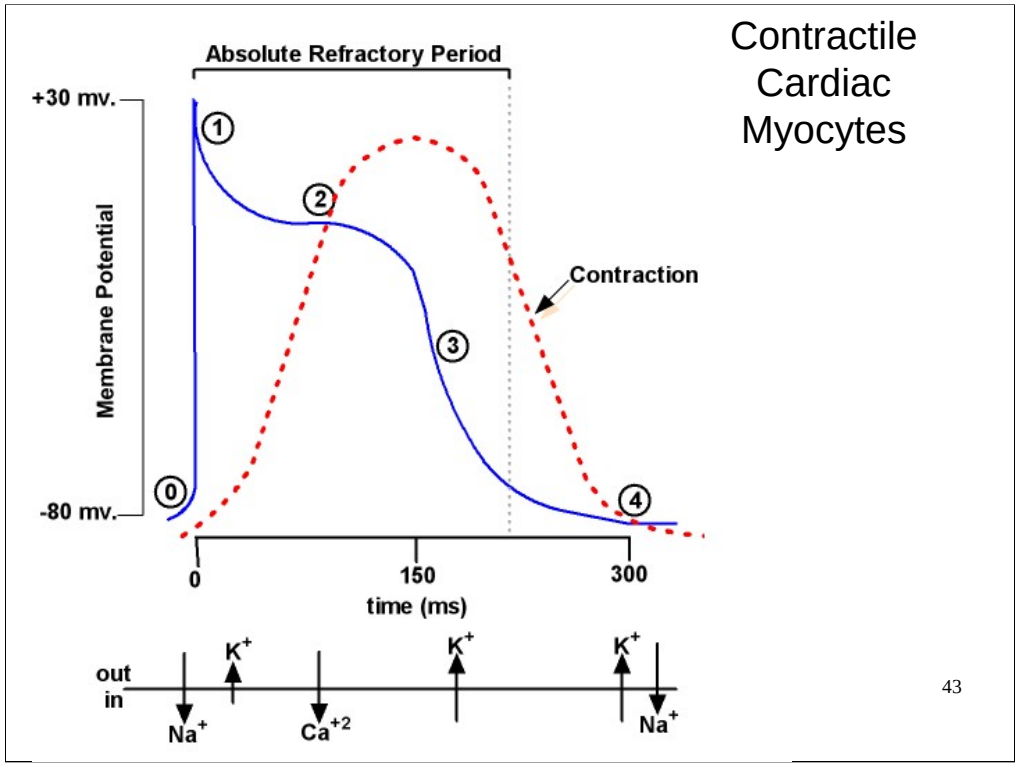
1)The slow sodium-calcium channels are leaky and cause the spontaneous depolarization to threshold for action potential generation. The fastest of these cells, those in the **SA node**, act as the heart's pacemaker.

2) Threshold depolarization causes the fast Ca+2 channels to open producing a spike of depolarization.

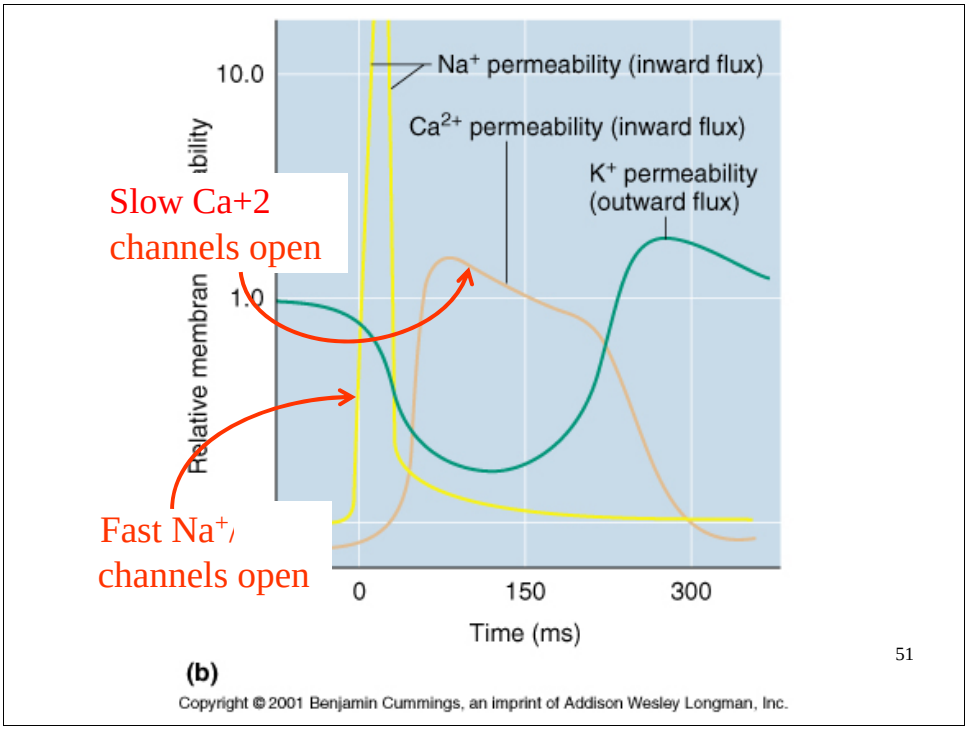
3) The Fast Ca+2 channels close and the K+ channels open, producing repolarization.

4) The potential reaches the resting level as the K+ channels close. Leaky Na+ channels take over again and spontaneous depolarization continues.

The autorhythmic cells have very little contractility. Their threshold can be affected by neurotransmitters and drugs.



In contractile cardiac myocytes, the fast Na+ channels respond to depolarization passing through the syncytium (0) to produce the spike, just as in skeletal muscle cells. K+ channels open to begin repolarization (1), but the slow Ca+2 channels open to prevent repolarization and prolong depolarization, producing a plateau (2). As these channels close and depolarization ends, repolarization occurs due to K+ leaving the cell. This continues until the resting potential is reached (4). Note that the prolonged depolarization facilitates the slow wave of contraction and prevents new stimuli from affecting the cells during this time.



Opening of the fast Na^+ channels produces the spike seen at the beginning of the myocardial action potential. Opening of the slow Ca+ channels produces the plateau which keeps the myocardial cells depolarized. Opening of the K^+ channels allows for repolarization.

Skeletal Muscle vs. Cardiac Muscle

polarization maintained by the Na^+/K^+ pump until a stimulus causes ion gates (channels) to open:	polarization maintained by the Na^+/Ca^{++} pump. In myogenic cells ion gates leak producing spontaneous depolarization.
fast Na^+ channels open to produce the depolarization spike associated with an action potential	fast Na^+ channels open to produce a spike at the beginning of the action potential.
short refractory period as K^+ channels open to re-establish membrane polarity	Slow Ca^{++} channels open to produce plateau depolarization This produces a long refractory period which ends as potassium channels open to produce repolarization.

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A comparison of skeletal and cardiac muscle illustrates the basis for the observations of the **depolarization plateau** and excedingly long **refractory period** in cardiac muscle.

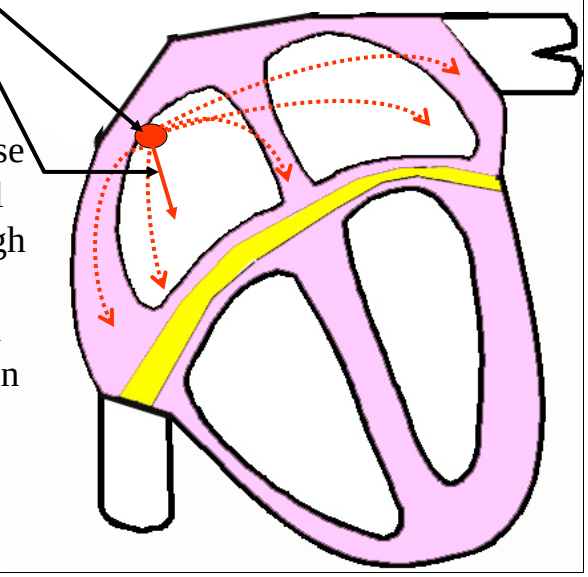
Cardiac Conduction System

See also Figure 19.14

SA Node

Internodal pathway

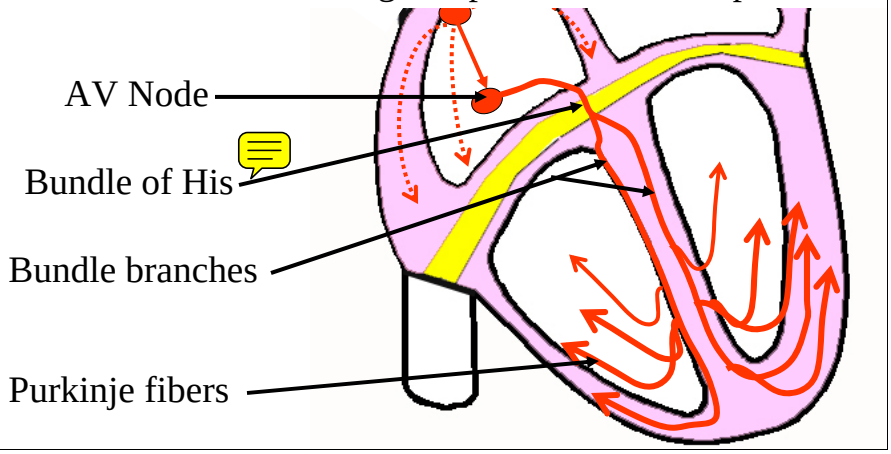
1) SA node depolarizes and the resulting impulse spreads across the atrial myocardium and through the internodal fibers to the AV node. The atrial myocardium contracts in response, a physical event.



The fastest autorhythmic area is called the **SA node** (sino-atrial node) and it acts as the heart's "pacemaker". The action potential produced by its depolarization spreads across the atrial myocardium which contracts in response.

All components of the conduction system are autorhythmic areas composed of fibrous myocardial cells whose primary function is electrical conduction, not contractility. E.g. these are muscle cells which act as nerve cells.

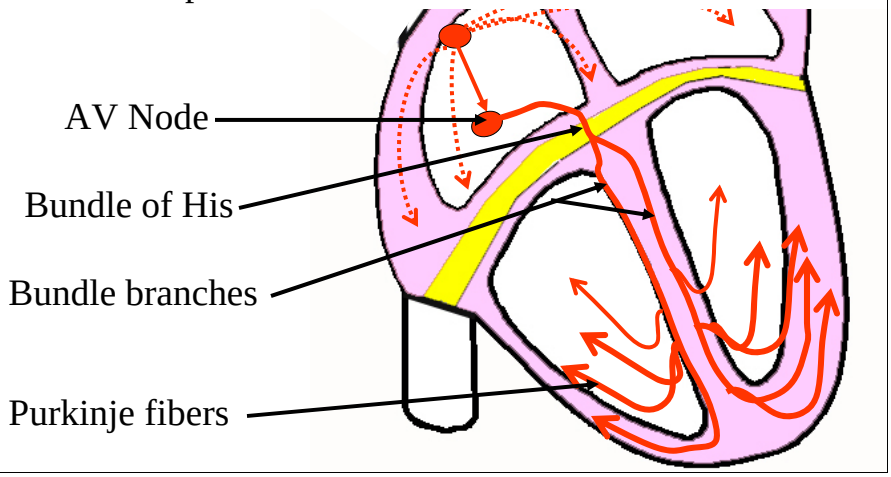
2) AV node picks up the impulse and transfers it to the AV Bundle (Bundle of His). This produces a .1 second delay in the cardiac cycle. It takes approximately .03 sec from SA node depolarization to the impulse reaching the AV node, and .13 seconds for the impulse to get through the AV node and reach the Bundle of His. Also during this period the atria repolarize.



The **AV node** is located in the floor of the right atrium, near the interatrial septum. The **Bundle of His** passes through the fibrous atrioventricular septum into the interarterial septum where it divides into **bundle braches**. There are two primary bundle branches, the left and right and many smaller branches and distributing fibers, the **Purkinje fibers**.

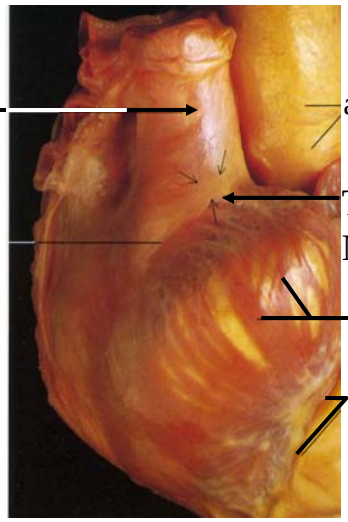
3) From the AV node the impulse travels through the bundle branches and through the Purkinje fibers to the ventricular myocardium, causing ventricular depolarization and ventricular contraction, a physical event.

4) Ventricular repolarization occurs.



The physical events, shown in red type, are not visible on an EKG. The electrical events, occurring as the impulse passes through the conduction system, produce the EKG.

The SA Node



The SA Node consists of small, modified muscle cells which generate the electrical signal that initiates the heartbeat

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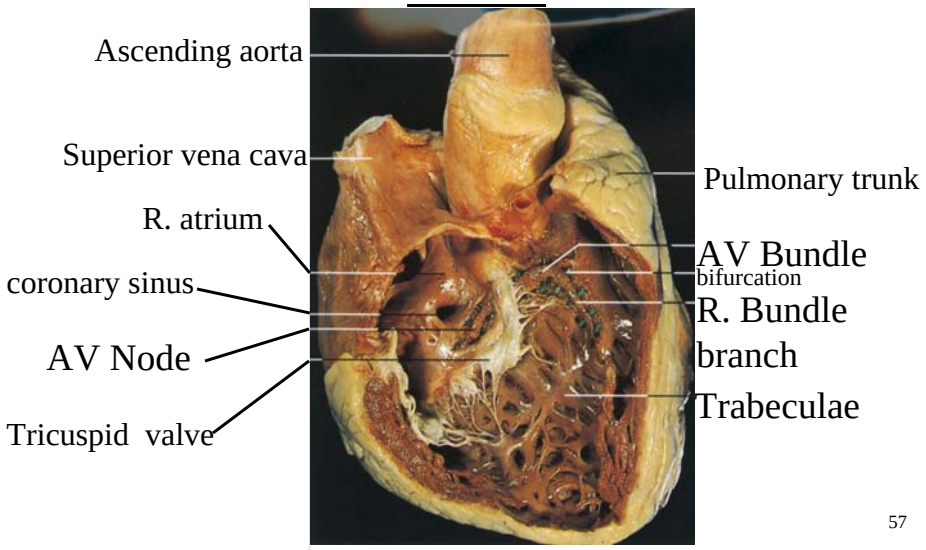
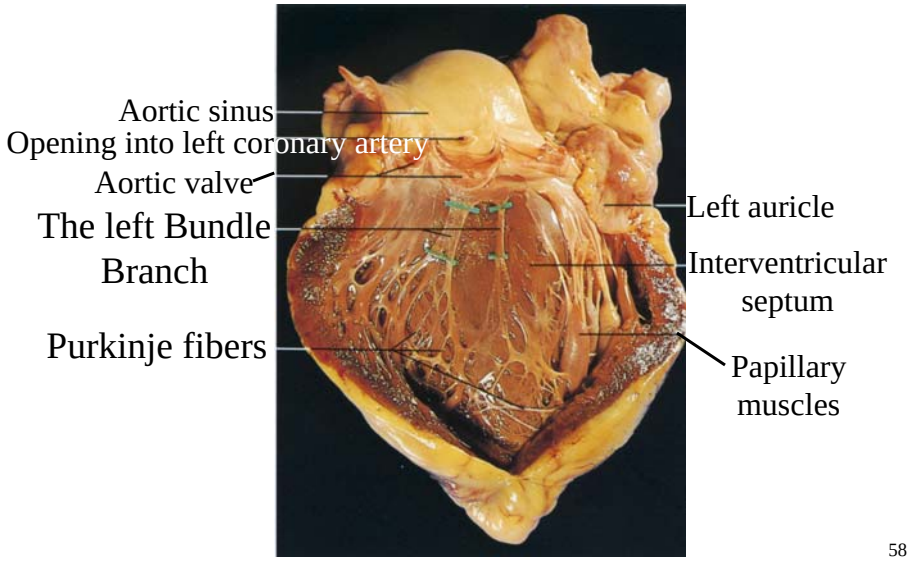
In this expanded right atrium you can see how thin the atrial wall actually is.

The AV Node and Right AV Bundle Branch

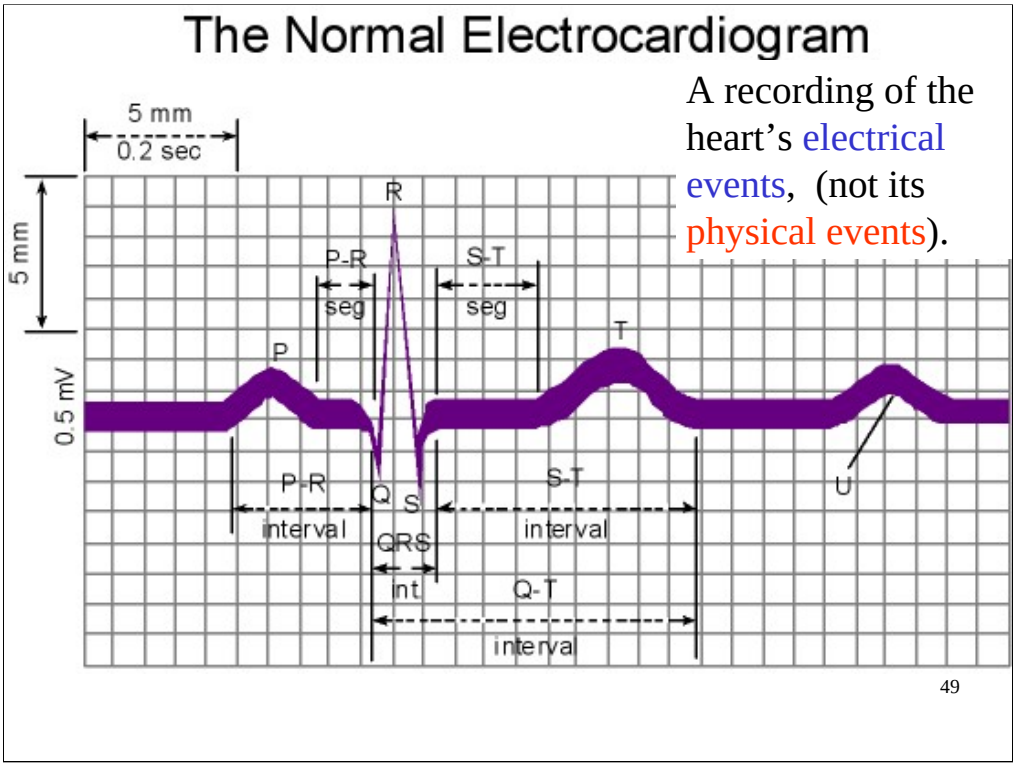


The AV node leads to the **Bundle Branches** (**Bundles of His**) which further branch repeatedly as they lead to Purkinje fibers in the ventricular muscle.

AV Bundle Branch, Left Side



Note the branches of the left side which lead to the **Purkinje fibers**. These fibers are seen as the **trabeculae** lining the ventricles.



P wave – corresponds to (1) – electrical events in the atria: SA node to AV node.

QRS complex – corresponds to (2,3): electrical events from AV node to Bundles to Purkinje fibers. Includes atrial repolarization

T wave – corresponds to (4): ventricular repolarization

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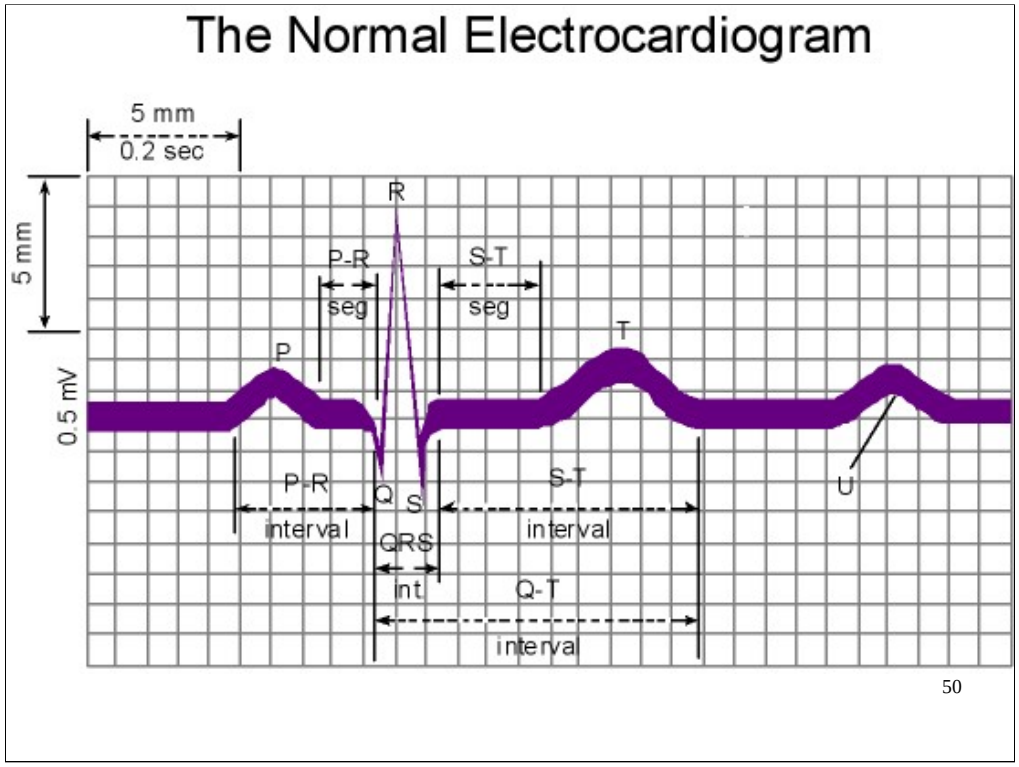
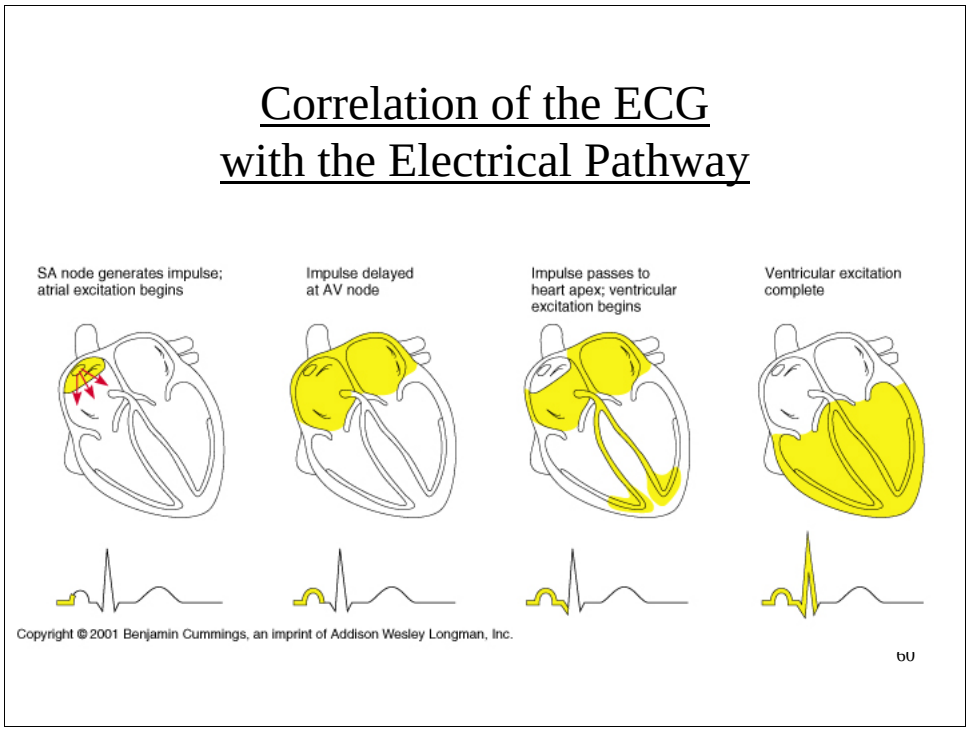


The ECG can be used to diagnose abnormalities in the functioning of the heart's components and conduction system. Several

easily recognized abnormalities are shown in Figure 19.18.



The electrical impulse spreads downward from the SA node through the atrial myocardium, and the contraction spreads in the same way, pushing blood into the ventricles. Ventricular electrical activity spreads from the apex upward, and the resulting ventricular contraction pushes blood upward into the arteries.

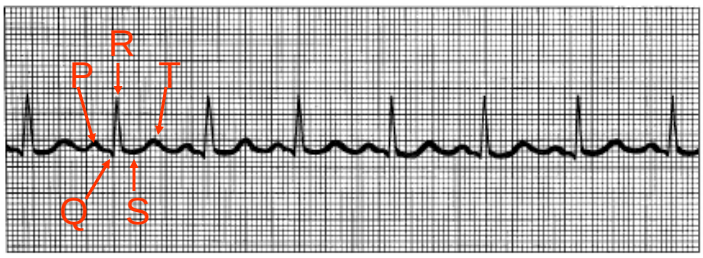
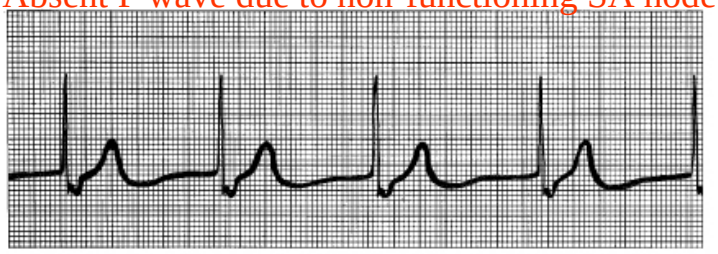


Figure 18.18

Normal ECG

(a)

Absent P wave due to non-functioning SA node



AV node becomes an ectopic pacemaker

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(b)

In tracing (b) there is no P wave because the SA node is not functioning. Without the SA node to initiate the beat, the next fastest area takes over, this being the AV node which has automaticity about half that of the SA node. An artificial pacemaker would be used to regulate the heart rate.

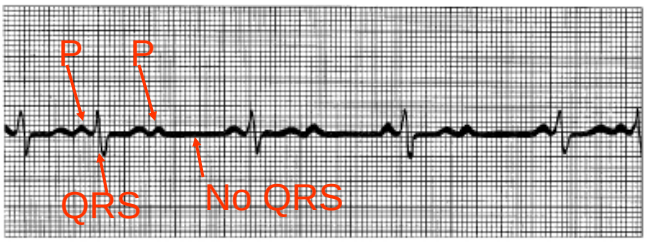
Artificial Pacemakers are used to compensate for damage to the SA node, Bundle branches or other parts of the heart's

conducting system. Some pacemakers stimulate only the ventricles, but more two chamber pacemakers are now being used

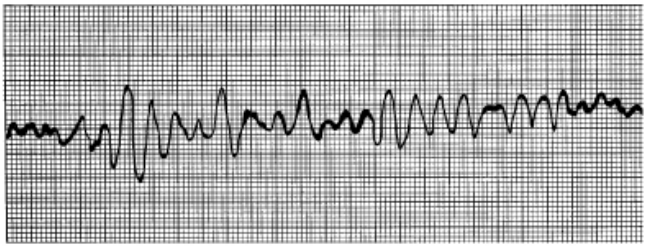
which stimulate both the atria and ventricles. These devices have complex sensing capability to sense the heart's own electrical

activity and coordinate the pacemaker's stimulation of the atria and ventricles. They can even respond to the increased

demand of exercise and compensate by increasing heart rate.



(c)



(d)

Figure 19.18

Partial bundle block

Ventricular fibrillation

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In a partial bundle block (trace c) the rate of impulse transmission through the AV node slows to about half its normal rate. This reduces the QRS frequency to every other beat. Sometimes partial bundle block responds to drugs, but others require an artificial pacemaker. Complete bundle block produces an ectopic pacemaker in the bundle branches and requires artificial pacing.

(d) Ventricular fibrillation is an electrical spasm in the ventricular myocardium and is lethal if not corrected quickly. A defibrillator is used to reset the myocardium by depolarizing it simultaneously. A regular rhythm can then be established. Implantable pacemaker-defibrillators can be used in patients who's hearts repeatedly suffer electrical spasms.

Other Cardiac Abnormalities

Atrial tachycardia, rapid heartbeat resulting from abnormal rhythm in the atria.

Atrial fibrillation is a short-circuiting electrical spasm in the atria.

Ventricular tachycardia is much more serious, resulting from severe ischemia and damage to the ventricular myocardium and often preceding ventricular fibrillation.

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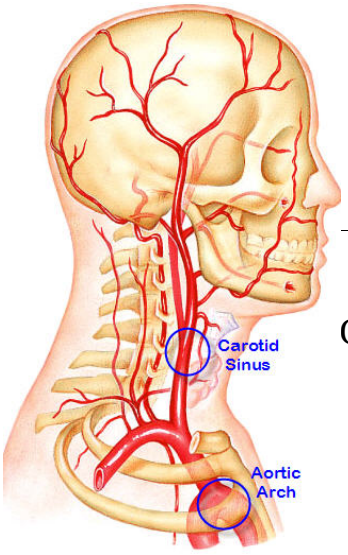
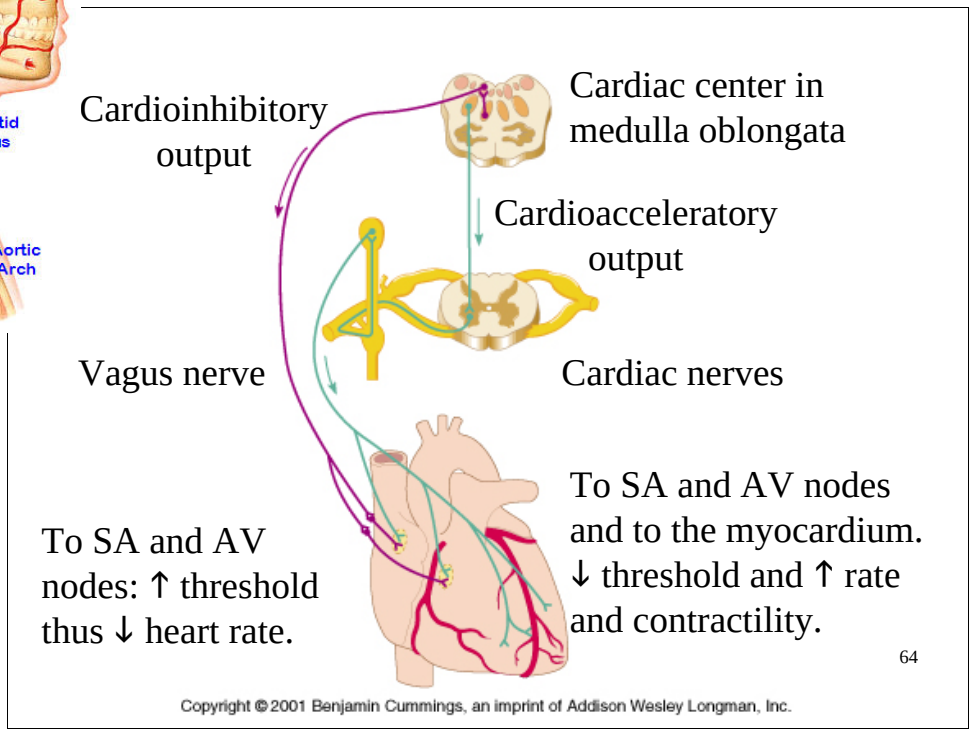
A typical form of atrial tachycardia (see paroxysmal atrial tachycardia also sinus tachycardia), involves the AV node and results in an inverted or obscured P wave. Atrial tachycardia tends to occur in young people and generally disappears with age without heart damage. A new surgical technique isolates and destroys the offending cells to restore normal rhythm.

Though more serious than atrial tachycardia, atrial fibrillation is rarely life-threatening. Since it interferes with the filling action of the atria, the heart's efficiency is reduced and may cause symptoms of weakness. In addition the sluggish blood flow in the atria may cause clots which can subsequently form emboli (clots which move and lodge in another location downstream).





The major control center for cardiac output is the Cardiac Center in the medulla of the brain. The medulla sends impulses to the heart through the parasympathetic (vagus nerve) and sympathetic (cardiac nerves) divisions. The vagus nerve innervates the SA and AV nodes and, by increasing the threshold for depolarization, reduces the heart rate. The vagus has no effect on contractility.

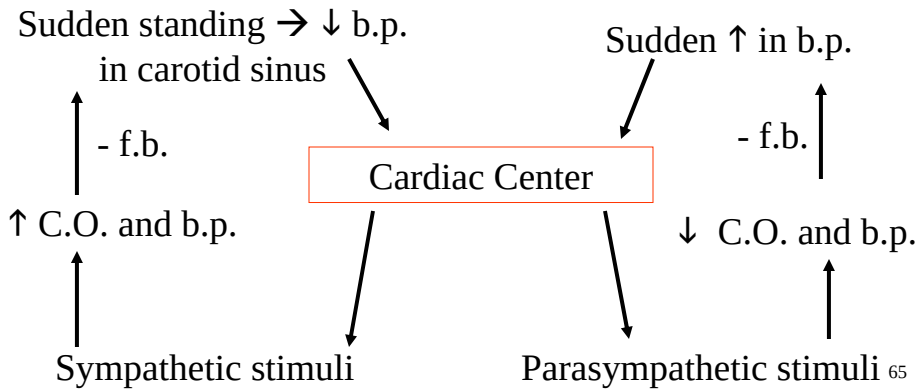
Sympathetic stimuli lead not only to the SA and AV nodes, but also to the myocardium itself. Its effect on the myocardium is to increase contractility, thus increasing the force and volume of contraction.

The cardioinhibitory center controls the heart at all times in the absence of stress, keeping the rate slow and steady.

The cardioacceleratory center mediates response to stress, "fight or flight", etc.

Inputs to Cardiac Center

Baroreceptor reflex– baroreceptors in the carotid and aortic sinuses maintain moment to moment pressure via control of cardiac output.



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The baroreceptor reflex does not respond to abnormally high or low pressures, at least not effectively. But rather the reflex keeps pressure within normal limits despite changes in body position, activity, etc. An example experienced by most everyone is the response to decreased pressure in the carotid sinus when one stands suddenly. At first light headedness occurs, sometimes even fainting. But the baroreceptor reflex kicks in and brings the pressure back up quickly. The opposite happens when pressure suddenly increases, but we are less aware of this phenomenon.

Higher brain center – the hypothalamus

- stimulates the cardiac center in response to exercise, emotions, fight or flight, etc.

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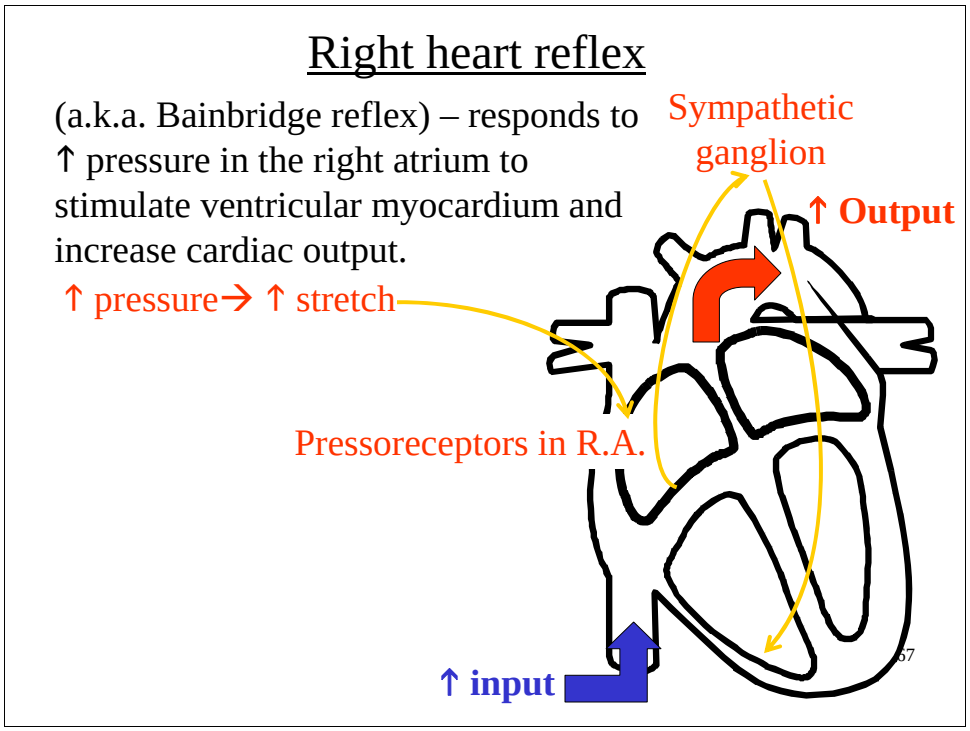
A major input to the cardiac center is the hypothalamus, which increases cardiac output when you get excited, go into “Fight or Flight” and exercise.



Right Heart Reflex - Pressoreceptors (stretch receptors) in the right atrium respond to stretch resulting from increased venous return. The reflex acts through a short neural circuit to stimulate the sympathetic nervous system resulting in increased rate and force of contraction. This regulates output to input. [*Venous return from the systemic division is related to exercise and enhanced by the skeletal muscular pump and semilunar valves in the large veins (See Figure 20.6). This mechanism also helps return lymph to the circulation for the same reasons.*]

The Frank-Starling Law

↑ stretch of myocardium → ↑ force of contraction

Myocardium behaves like skeletal muscle in its length-tension relationship.

Automatically compensates output to input.

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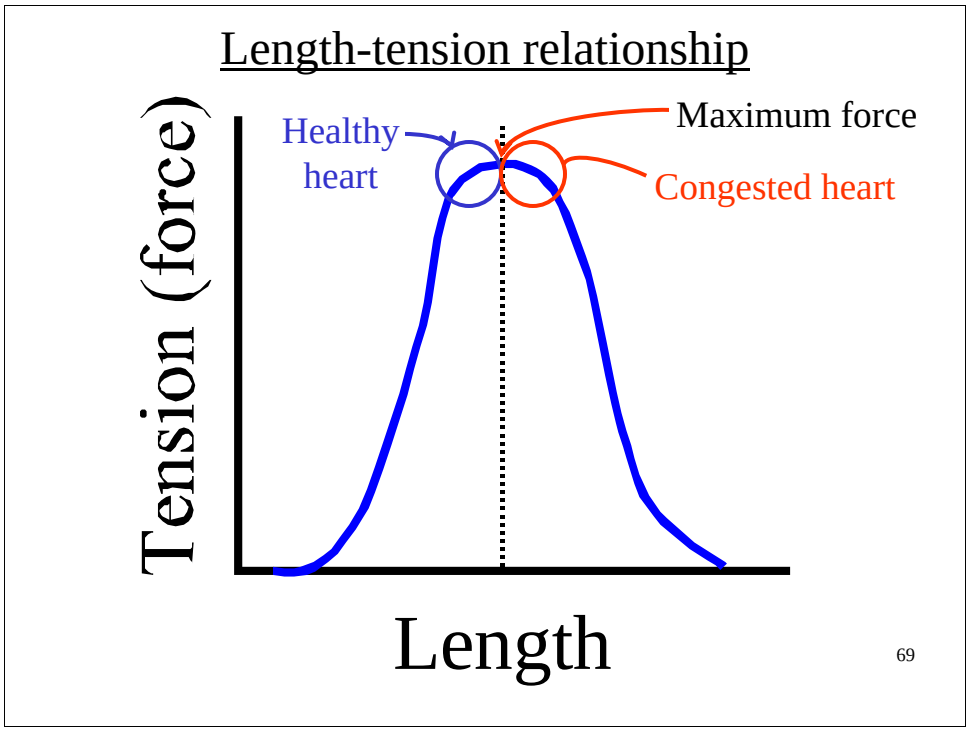
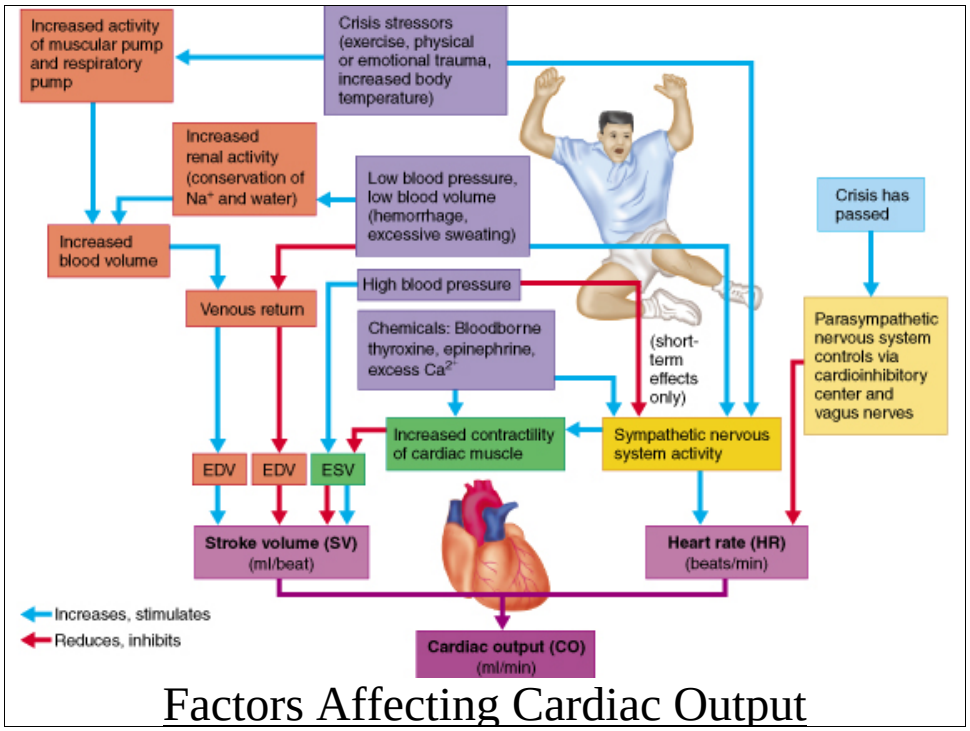
The **Frank-Starling Law** - (Starling's Law of the Heart) - Like skeletal muscle the myocardium has a length tension curve which results in an optimum level of stretch producing the maximum force of contraction. A healthy heart normally operates at a stretch less than this optimum level and when exercise causes increased venous return and increased stretch of the myocardium, the result is increased force of contraction to automatically pump the increased volume out of the heart. I.e. the heart automatically compensates its output to its input.



Like a skeletal muscle, the heart muscle achieves its greatest force at or near its resting length, in the middle of its range of contraction. When not stressed, the heart operates below this maximum level so that when increased demand is placed on it by increasing stretch from venous return, the heart will exhibit increased force to pump this blood out to the systemic division. In a congested heart the muscle is stretched beyond its ability to effectively respond. This leads to yet more stretch and less force in a feedback which leads to congestive heart failure.



Factors Affecting Cardiac Output

Review Figure 18.23, Factors affecting cardiac output. Be able to describe the effect of heart rate, stroke volume, ejection fraction, EDV, ESV, sympathetic stimulation, parasympathetic stimulation, loss of blood volume and pressure, increased blood volume and pressure, venous return, increased temperature, and other factors indicated in the figure.

Assignment:

Describe the effect of heart rate, stroke volume, ejection fraction, EDV, ESV, sympathetic stimulation, parasympathetic stimulation, loss of blood volume and pressure, increased blood volume and pressure, venous return, and increased temperature on cardiac output.