Anatomy and Physiology Part 1

Directions for nursing CEU credit: Review the material in this module. Download the test form. Take the test. Fax or send the answer sheet with a form of payment. Passing score is 75%. You have 3 chances to take the test. Upon completion you will receive a CEU certificate. Critical Care ED is an approved provider of continuing education in nursing by the California Board of Nursing.

Module objectives: Upon completion of this module the learner will be able to:

Name three of the surfaces of the heart.
Define RAO and LAO.
Recite the pathway of blood movement through the heart.
Describe the interior of each heart chamber.
Recall 2 of the 3 main coronary arteries.
List the various portions of the heart fed by each coronary artery.
Discuss the locations of the various branches of the coronary venous system.
List the 3 layers of heart muscle.
Describe the role of arterial baroreceptors.
Discuss the role of various natriuretic peptides.
List the various types and roles of sympathetic nervous system receptors which influence the functioning of the heart.
Contrast the roles of sympathetic and parasympathetic stimulation in the heart.
Discuss the various phases of the cardiac cycle.
Define heart rate, preload, afterload, and contractility.
Discuss myocardial oxygen consumption factors.
Describe the impact of the Renin-Angiotensin-Aldosterone System on heart functioning.

Anatomy

I. Cardiac Anatomy

Surface and Sectional Anatomy

The heart is usually described as having five surfaces. These include:

anterior surface: this is the area toward the front of the body

posterior surface: the opposing surface to anterior; the area to the back

diaphragmatic or **inferior surface**: the area which sits on the diaphragm

superior surface: referred to in electrophysiology, the upper portion of the heart

lateral surface: there is a left and right 'lateral surface, found on each side. When one refers to a patient having a myocardial infarction, this is generally the left lateral. The

majority of myocardial infarctions occur on the left, and phrases like ‘inferior MI’, ‘lateral MI’ are referring to the left ventricle.

septal surface: this represents the area between the right and left sides of the heart.

Figure 1 below shows the frontal plane (also called coronal plane) view of the heart. The areas of the left lateral surface and the diaphragmatic or inferior surfaces are highlighted.

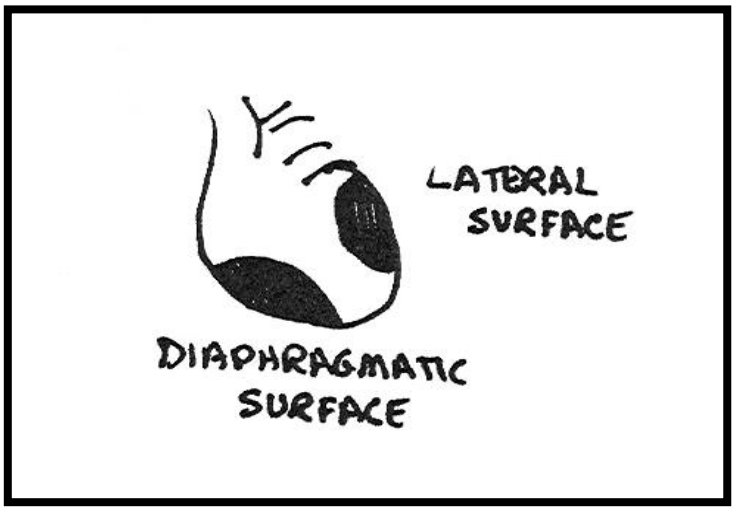

FIGURE 1: Frontal plane

Figure 2 is the sagittal plane or view of the heart. The sternum can be seen to the left of the heart and the spine to the right. The anterior, posterior, and diaphragmatic surfaces are highlighted here.

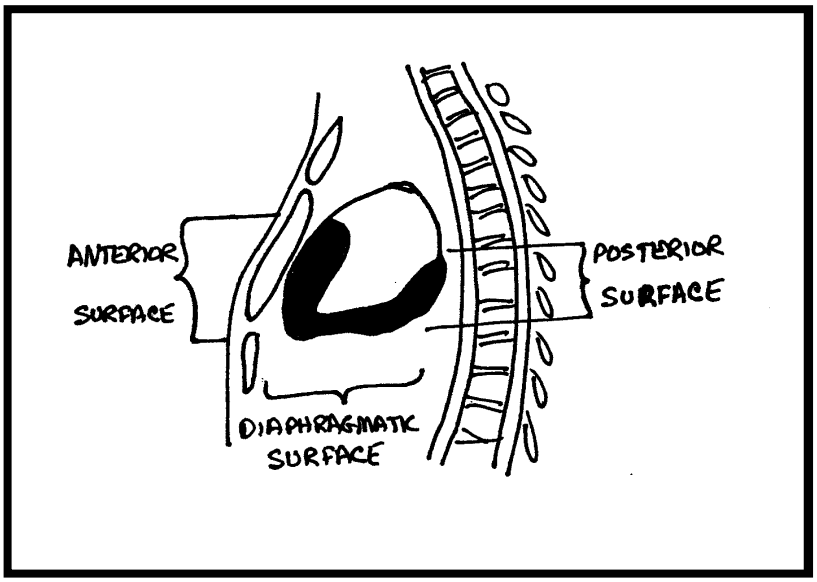

FIGURE 2: Sagittal plane

Figure 3 is the transverse plane or view. As you look down into the ventricles, notice the greater size and wall thickness on the left. It almost appears that the right ventricle was an afterthought that was attached to the left ventricle. In this transverse plane the posterior, lateral, and anterior surfaces are visible. In these figures however, note that the left ventricle exclusively is being labeled.

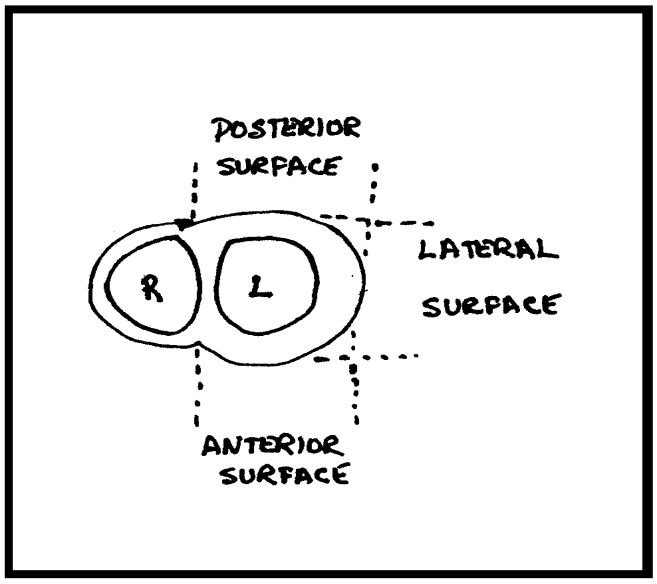

FIGURE 3: Transverse plane

Figure 4 labels the various surfaces of the heart as seen from the right anterior oblique (RAO) view. The word 'base' or 'basal' refers to the 'top' of the heart and the word 'apex' or 'apical' refers to the bottom of the heart.

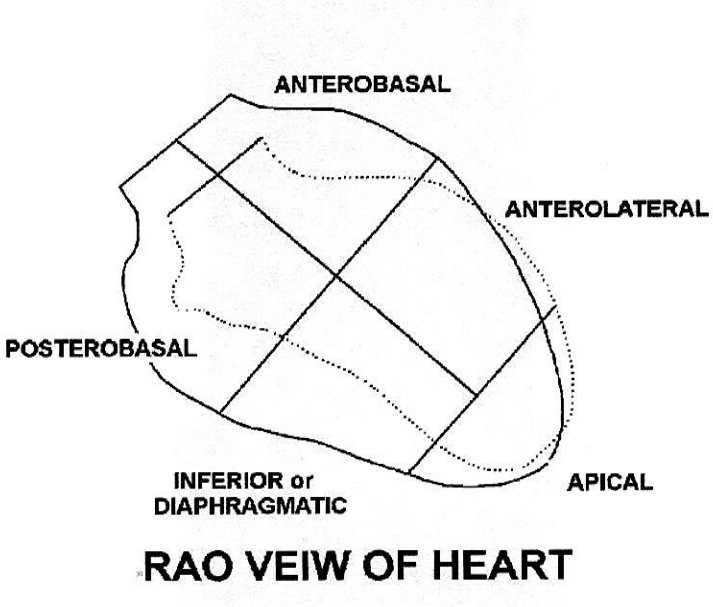

FIGURE 4: RAO view of the heart

Figure 5 is the left anterior oblique or 'LAO' view of the heart. It can be seen that the left ventricle (LV) overshadows the right ventricle (RV) in this view.

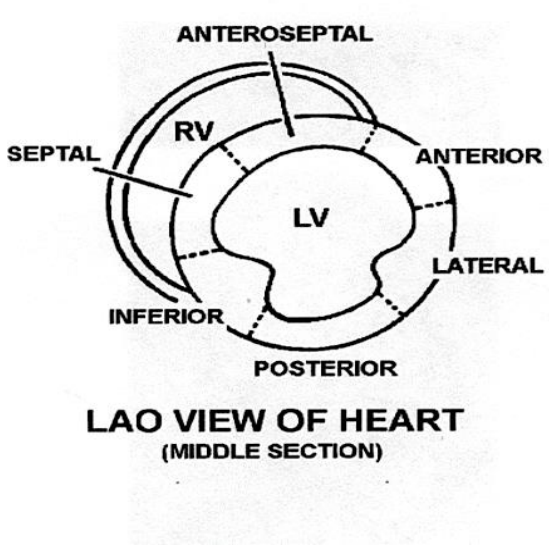

FIGURE 5: LAO view of the heart

Heart Function

The heart has four chambers and functions as a double pump. The **right** and **left atria** serve as primers for the ventricles. The actual pumping job is performed by the ventricles.

The **right atrium (RA)** receives un-, de-oxygenated, or venous blood from the body through the **superior** and **inferior vena cavae (SVC and IVC).** The SVC receives blood from the upper portions of the body while the IVC brings blood back to the heart from the lower portions of the body. The venous blood from the heart muscle itself returns to the RA through the **coronary sinus (CS).**

The RA contracts and sends this blood through the tricuspid valve toward the **right ventricle (RV)**. The RV then pumps this venous blood to the lungs for oxygenation through the **pulmonic valve** and to the **pulmonary artery (PA).**

The **left atrium (LA)** receives the oxygenated or arterial blood from the **pulmonary veins (PV).** Blood passes from the LA to the **left ventricle (LV)** through the mitral valve. The left ventricle pumps the oxygenated blood out the **aortic valve** to the **aorta** and on to the systemic circulation.

There are four heart valves. Two separate atria from ventricles (the **mitral** and **tricuspid**). These are inflow tract valves and are often referred to as **AV valves**.

Two valves separate the ventricles from the great vessels. On the right the pulmonic valve separates the right ventricle from the pulmonary artery. On the left the aortic valve separates the left ventricle from the aorta.

The valves are cusps that open with pressure on the convex side (downstream) and close with pressure on the concave side (upstream). The mitral valve has two cusps; all other valves have three.

Figure 6 traces the flow of blood throughout the heart.

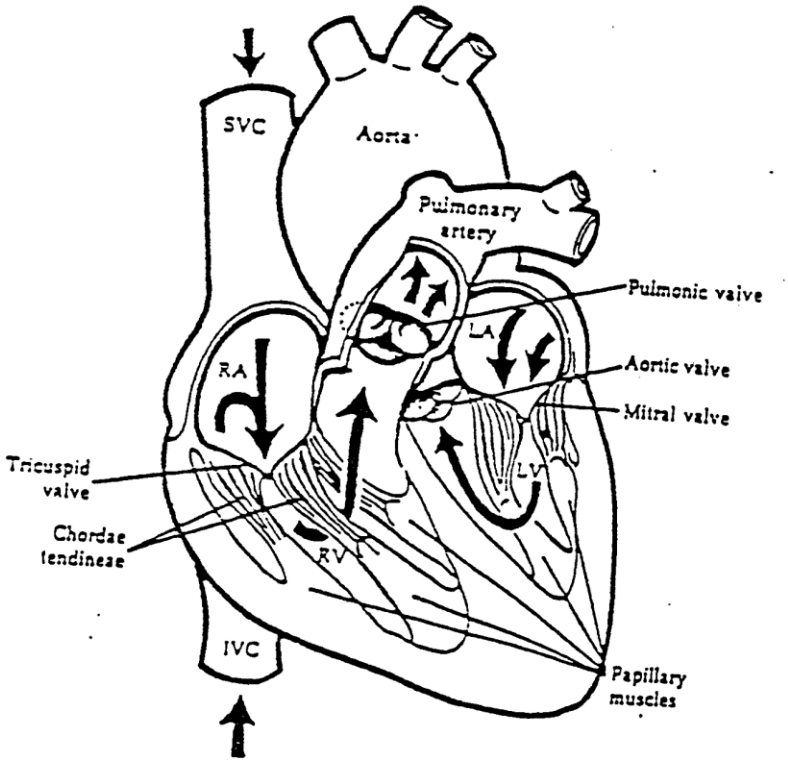

FIGURE 6: Movement of blood through the heart

Structure

The heart is found in the **mediastinum** or central area of the chest along with the pericardium and the great vessels. The base of the heart is actually the top (not what one would logically assume), and this is where the atria are and where the great vessels are found. The apex of the heart is the tip of the left ventricle, or where it seems to come to a point.

Self-Study Questions:

1. What are the various surfaces of the heart?
2. What surfaces of the heart are visible with each plane?
3. What do RAO and LAO stand for?

4. What portions of the heart are visible with RAO and LAO?
5. Trace the flow of blood through the heart.
6. Where is the base of the heart and where is the apex?

Chambers

Right atrium

Deoxygenated blood returns to the right atrium from the SVC, IVC, and coronary sinus. This blood passes through the right atrium, through the tricuspid valve and into the right ventricle. The walls or the RA are relatively thin. Specific features of the right atrium include the **crista terminalis** or terminal crest. This is the area believed to be responsible for initiation of inappropriate sinus tachycardia or 'crystal' tachycardias. This area is a vertical muscular ridge. There are also fine muscular ridges called **pectinate muscles** that are at right angles to the terminal crest.

The **fossa ovalis** is the area in the right atrium that had been the foramen ovale prior to birth. It is through the foramen ovale that oxygenated blood passed from the right to left atrium. In the adult heart, the fossa ovalis becomes a shallow depressed area. It is through this thin membrane area that septal puncture is generally performed.

The opening of the coronary sinus is also found in the right atrium. The valve for the coronary sinus is called the **thebesian valve**. Figure 7 is a cartoon-like drawing showing the many openings or fossae to be found in the right atrium.

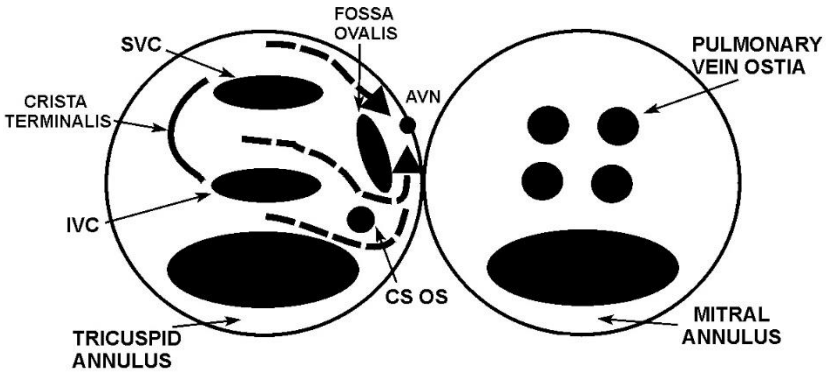

FIGURE 7: Atrial floor plan

Right ventricle

The right ventricle receives the unoxygenated blood from the right atrium through the tricuspid valve. When ventricular contraction occurs, the blood is forced through the pulmonic valve, into the pulmonary artery and on to the lungs for oxygenation.

The right ventricle has a ridged muscular portion. These muscles are called **trabeculae carnae**. The smooth-walled superior portion of the right ventricle is called the **infundibulum**.

Left atrium

Blood arrives in the left atrium from the pulmonary veins. The pulmonary veins have gained great prominence in the EP world due to their role in atrial fibrillation initiation. There are four pulmonary veins, right superior and inferior and left superior and inferior. Their abbreviations are **RSPV**, **RIPV, LSPV**, and **LIPV**. The left atrium has some very smooth and some trabeculated areas. Blood passes from the left atrium through the mitral valve and to the left ventricle.

The **left atrial appendage** (LAA) is an area of the left atrium in which blood flow is quite stagnant, allowing the blood in this area to form clots more easily. This is particularly true during atrial fibrillation, when the atrium does not contract as forcefully.This may lead to strokes and other embolic problems.

Ligament of Marshall is an epicardial vestigial fold which may be found in what was the embryonic left SVC. This area contains nerves, a vein, and muscle tracts. There are sympathetic nerve trunks here in addition to parasympathetic innervation. In a study by Kurotabi, a significant increase in the incidence of PV foci in LSPV was found in those with a well developed vein of Marshall.

Left Ventricle

The left ventricle as stated earlier is much thicker than the right, at least 2-3 times as thick. The left ventricle must be stronger in order to pump blood out against the peripheral vascular resistance of the entire body. Oxygenated blood from the left atrium passes through the mitral valve and into the left ventricle. The left ventricle pumps this blood through the aortic valve, into the aorta, and to the body.

There are more trabeculations on the left compared to the right; however those on the left are more delicate.

Septum

The septum separates the right and left ventricles and is thus generally called the interventricular septum. There is both a muscular and a membranous component to the septum. The muscular portion seems more like a left ventricular structure and actually looks as if it is bulging into the right ventricle. The membranous septum separates the right atrium from the left ventricular component which leads to the aortic outflow tract.

Heart Valves

There are four heart valves. Two separate the atria from the ventricles (**mitral** and **tricuspid**). These are inflow tract valves and are often referred to as AV valves. The valves are cusps that open and close with pressure on the convex side (downstream) and close with pressure on the concave side (upstream). The tricuspid valve has an anterior, septal, and posterior leaflet, with each having a **papillary muscle** and a **chordae tendinae** or tendon to connect them. The mitral valve has an anterior and posterior cusp in addition to several accessory cusps. There are anterior and posterior papillary muscles that are attached to these valve cusps and chordae tendinae that connect the muscles to their respective cusps.

Two valves separate the ventricles from the great vessels. On the right side the **pulmonic** valve separates the right ventricle from the pulmonary artery. On the left the **aortic** valve separates the left ventricle from the aorta. The **aortic** valve, also called a semilunar valve, has a right, left, and posterior cusp. This valve prevents the backflow of blood into the left ventricle after the left ventricle has contracted.

Figures 6 and 8 show the location of the heart valves.

Heart Sounds

The heart sounds that we hear when listening to the chest with a stethoscope are the sounds of the heart valves opening and closing. The **S1** sound, the first heart sound, occurs due to closure of the mitral and tricuspid valves, while the 2nd heart sound, **S2** occurs during closure of the aortic and pulmonic valves. **Murmurs** occur when blood has difficulty passing through a valve. These problems may occur due to a valve being stenotic or closing incompletely, or due to valve incompetence, with a resulting turbulence and backward flow.

Figure 8 is a cartoon drawing of the chambers of the heart and the valve locations. It helps put the relationship of these structures in perspective and helps one to imagine chamber contractions and valve closures in a different way.

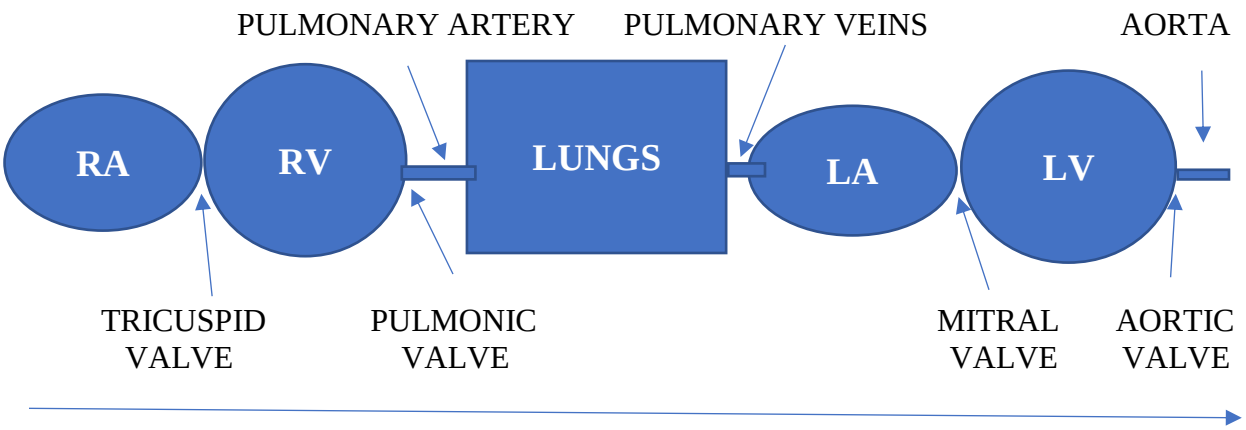

FIGURE 8: Cartoon of blood flow through the heart and lungs

II. Coronary Arteries

The first branching of the aorta is the coronary arteries, the right and the left main. The left main subsequently branches into the **left anterior descending** (LAD) and the **left circumflex** (LCX).

The **LAD** delivers blood to most of the anterior wall of the left ventricle, the anterior portion of the interventricular septum, the anterior wall of the right ventricle and the anterior papillary muscle of the left ventricle. An arterial blockage at the origin of this vessel would thus put all of these structures in jeopardy.

The **LCX** delivers blood to the lateral wall of the left ventricle and left atrium, the posterior wall of the left ventricle, a portion of the inferior wall of the left ventricle, to the sinus node in 45% of the population and to the AV node in 10% of the population.

The **right coronary artery** (RCA) brings blood to the right atrium and ventricle, to the posterior wall of the left ventricle, to the interventricular septum and the inferior portion of the left ventricle. In addition the RCA feeds the sinus node in 55% of the population and the AV node in 90% of the population.

Figure 9 shows the artery locations and labels some of their branches. It should be noted that the RCA and LCX both bring blood to the posterior wall of the left ventricle.

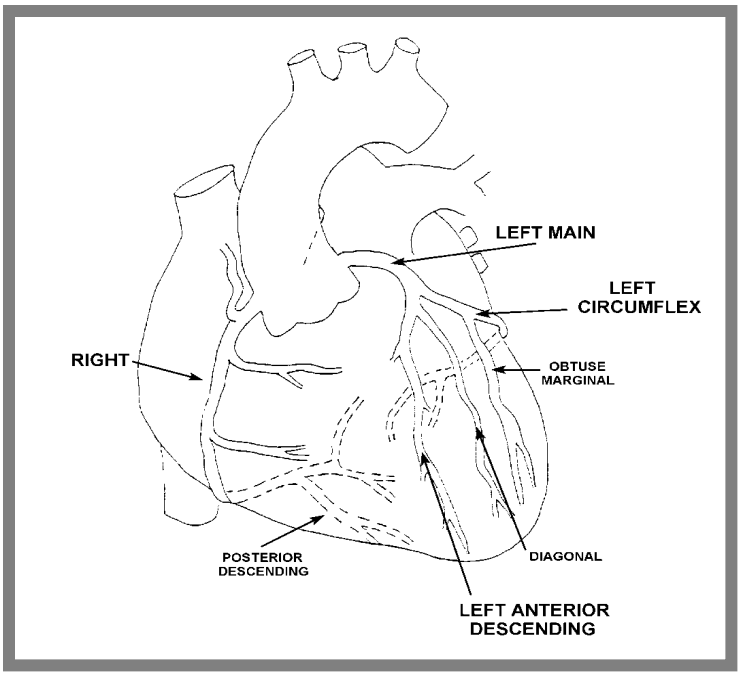

Figure 9: Location of the coronary arteries

❖Left Anterior Descending
- Left muscle mass (most of anterior wall of left ventricle)
- Anterior portion of interventricular septum
- Anterior wall of right ventricle
- Anterior papillary muscle of left ventricle

❖Left Circumflex
- Lateral aspect of left ventricle and left atrium
- Posterior wall of left ventricle
- Part of inferior wall
- Sinus node in 45% of population
- AV node in 10% of population

❖Right Coronary Artery
- Right atrium and ventricle
- Posterior portions of left ventricle
- Interventricular septum
- Inferior portion of left ventricle
- Sinus node in 55% of population
- AV node in 90% of population

Table 1. AREAS OF THE HEART MUSCLE FED BY CORONARY ARTERIES

III. **Venous Return**

Venous return from the heart muscle occurs through cardiac veins which parallel the coronary arteries. The middle cardiac vein lies next to the posterior descending coronary artery. The small cardiac vein parallels the right coronary artery. And the great cardiac vein lies next to the anterior descending branch of the left coronary artery.

The **coronary sinus** is the final conduit for the venous blood of the heart. The coronary sinus is located on the posterior wall in the AV groove. The coronary sinus has become important in electrophysiology studies as a window to information about the electrical activity of the left side of the heart without actually entering the left side. A catheter may be advanced through the coronary sinus os and to the left side to use during atrial fibrillation, left atrial tachycardia, left-sided WPW and left-sided VT cases. Pacing catheters are also placed through the coronary sinus to achieve bi-ventricular pacing.

IV. Histology

Heart walls

The heart muscle is composed of three layers. The outer layer is the **epicardium**. The epicardium is a thin serous membrane which is actually a part of the pericardium. The middle portion of muscle is the **myocardium,** and the layer which is in contact with the blood flowing through the heart is the **endocardium.** The endocardium is composed of endothelial tissue, which also covers the heart valves and is the type of tissue which forms the inner lining of the vessels which connect to the heart.

Fibrous tissue separates the atria and ventricles and provides the framework for the cardiac valves. This tissue is also known as the **cardiac skeleton**.

Pericardial Sac

The heart is contained within the **pericardium,** a double-walled, membranous sac which consists of an external layer of dense fibrous tissue and an inner serous layer that surrounds the heart directly. The base of the pericardium is attached to the central tendon of the diaphragm. A thin serous liquid is found in the cavity, which helps to eliminate friction during contraction.

Cardiac Muscle

The basic functional unit of cardiac muscle fiber or myocyte is the sarcomere. The sarcomere contains two groups of protein filaments, actin and myosin, which overlap. Intercalated discs are found scattered throughout the myocyte membrane and contain three components having both structural and functional attributes: desmosomes, responsible for anchoring the sarcomere to the cell walls; adherens junctions, helping to maintain cell-to-cell contact; and gap junctions, providing a low resistance connection to allow excitation to more easily pass from one cell to the next.

Self-Study Questions:

1. Where do you find the crista terminalis?
2. Where are the pectinate muscles?
3. Discuss the significance of the fossa ovalis.
4. What is the infundibulum?
5. What is the RSPV?
6. Discuss the significance of the left atrial appendage.
7. Why is the left ventricular wall thicker than the right?
8. Name the AV valves.
9. Closure of which 2 valves creates the S2 sound?
10. The RCA delivers blood to which parts of the heart?
11. Discuss the significance of the coronary sinus in electrophysiology.
12. Where are the coronary veins found?
13. Name the three layers of the heart muscle.

Hemodynamics

I. Blood Pressure

Systolic blood pressure is the arterial pressure measured during ventricular contraction. Diastolic pressure is the pressure obtained during the resting phase. A variety of factors are responsible for determining systemic blood pressure values. One of the more obvious is the actual volume of blood which is in the circulation. We know that loss of blood causes blood pressure to drop, and an excessive amount of blood or fluid will cause hypertension.

The vascular system can also affect the blood pressure measurement. A common example is hypertension, where lack of elasticity in the vessel walls leads to a higher pressure. Medications can also cause either vascular constriction or dilation, and lead to blood pressure changes.

II. Cardiac Output

Cardiac output is basically the product of heart rate multiplied by stroke volume. It is the volume of blood ejected by the ventricle in one minute. The heart of the normal resting adult will pump 4-8 liters of blood per minute. Cardiac output volume is determined by all of the events which occur during the cardiac cycle (translated into '**preload**'), contractile force of the heart muscle (**contractility**), the vascular resistance and volume workload against which the heart must 'push' (**afterload**), and the **heart rate**.

For clinical purposes, c**ardiac index (CI)** is the parameter that is often used instead to estimate the volume of blood delivered. Cardiac index factors the body surface area of the individual into

the equation. Thus, CI = CO/BSA. BSA is obtained by multiplying height times weight. The normal CI is 2.4-4 liters/minute/M^2.

There are 4 distinct periods during the cardiac cycle, two related to atrial activity, and two to ventricular activity. When taken together, there is overlapping of atrial and ventricular activity. Figure 10 Illustrates the various cycles and displays events that are going on concurrently.

Atrial systole is the period of time, as seen by the electrocardiogram, from about the middle of the P wave to the middle of the QRS. This is the period of atrial contraction. Atrial systole occurs during the latter portion of ventricular diastole or resting phase. The squeezing or contraction of the atria ideally insures that the maximal amount of blood is delivered to the ventricles. This period is associated with the 'atrial kick', this act of atria contraction providing 20% of the volume the ventricle will have received. Atrial kick is lost when patients go into atrial fibrillation, and their atria 'quiver' rather than contract. Following atrial systole, the A-V valves close, producing the first heart sound, or S1. This closure helps to prevent backflow of blood into the atria as the ventricles contract.

The period of **atrial diastole**, the relaxation phase for the atria, begins with the ventricular contractile phase or isovolumic contraction. During this time the RA receives blood from the IVC, SVC and through the coronary sinus (CS). Meanwhile on the left oxygenated blood is returning from the lungs to the LA from the pulmonary veins. Atrial diastole is actually occurring for about 3/4th of the entire cardiac cycle.

Ventricular systole or the **isovolumic contractile phase**, is the time period when ventricular contraction occurs. During this phase the pressure in the ventricles rises and the AV valves close in order to prevent backflow. Ventricular ejection sends blood out the aortic and pulmonic valves into the pulmonary artery and aorta. On figure 10 this is the period of greatest recorded pressure. Notice that the QRS of the EKG is inscribed just prior to this event (electrical event precedes the mechanical event).

Ventricular diastole is the period also known as **isovolumic relaxation**. During this period there is no change in ventricular volume. In addition, the aortic and pulmonic valves close, preventing backflow into the ventricles. This closure of the aortic and pulmonic valves is responsible for the production of the 2nd heart sound. Following this period rapid ventricular inflow resumes. As the atria were in the process of filling during atrial diastole or relaxation phase, atrial pressures began to exceed ventricular pressures. This increased atrial pressure causes the AV valves to open and blood to enter into the ventricles. Atrial contraction then occurs, causing further filling of the ventricles (remember the atrial kick). It is important to note that it is during the period of ventricular diastole that the coronary arteries and the heart muscle itself actually receive blood. Coronary artery perfusion does not occur during ventricular contraction as the vessels would be involved in the extreme 'squeezing' motion and unable to be filled.

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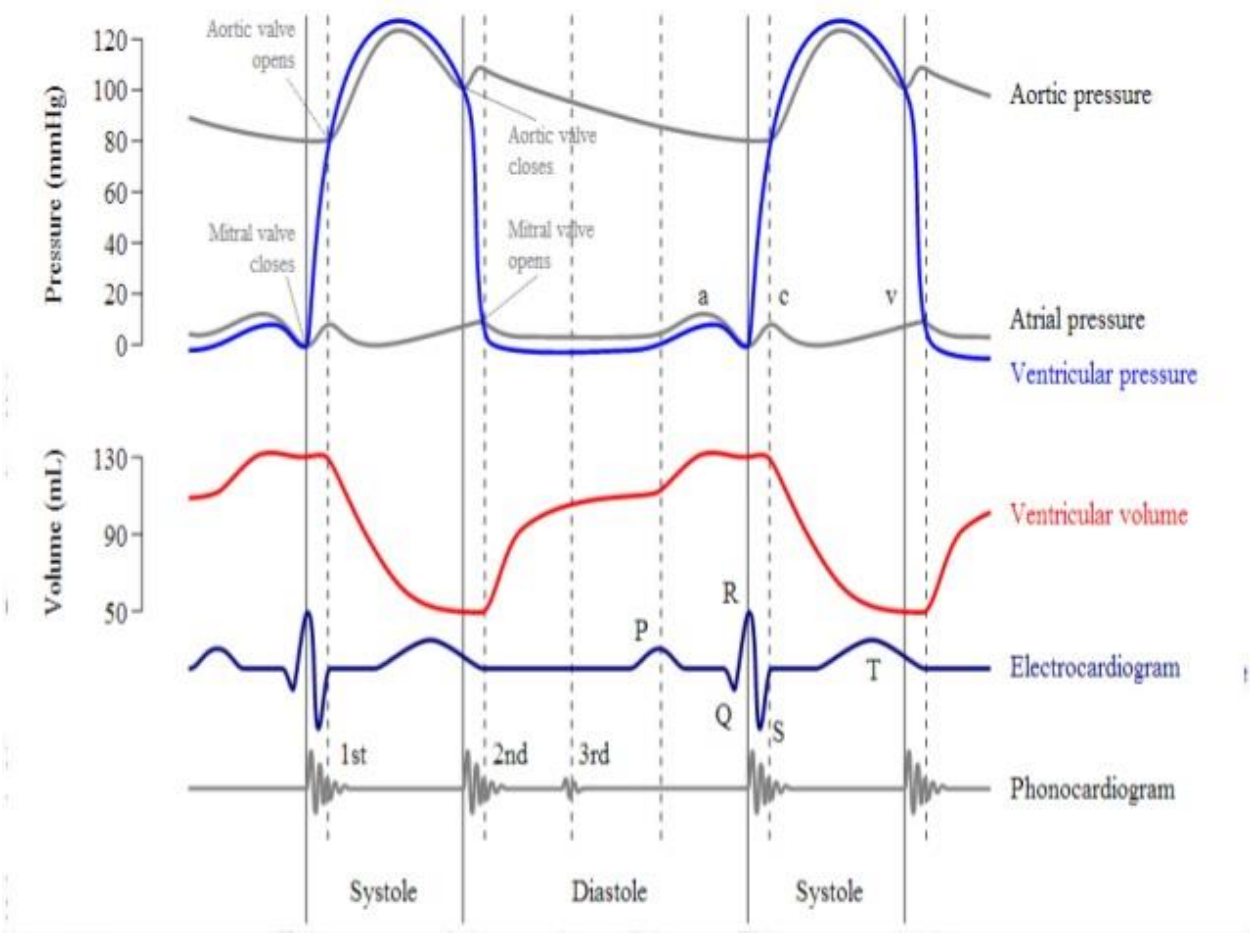

FIGURE 10: The cardiac cycle

III. Heart rate, stroke volume, preload, afterload, contractility, and myocardial oxygen consumption

Normally the heart can vary its cardiac output up to 5-6x the resting level, depending on one's age and physical condition. **Two methods** by which the heart regulates cardiac output in response to stress or disease is via **heart rate** and **stroke volume**.

The most effective method is through changes in **heart rate**. The heart is able to double or triple cardiac output in the healthy person by an increase of rate to 170 or 180. However, with disease a rate of even 120 may be harmful because of the increased oxygen demand and decreased time for diastolic coronary artery filling which occur.

Stroke volume is the amount of blood ejected with one contraction. Changes in **stroke volume** occur through the influence of three factors: **preload**, **afterload**, and **contractility**. **Preload** is

actually ventricular end-diastolic volume. The Frank-Starling law describes the principle for this factor. Preload is the initial stretch of myocardial fibers at end-diastole. It is determined by the diastolic filling pressure, total blood volume, distribution of blood volume, and atrial systole.

Ventricular **afterload** is the **systemic vascular resistance** and myocardial fiber shortening that occurs during systole. Think of factors such as presence of hypertension, aortic stenosis, and coarctation of the aorta, and how they would make it more difficult for forward flow to occur.

Contractility is the level or degree of ventricular performance at any given end-diastolic volume. Contractility depends on the contractile state of the heart. Drugs can alter contractility. Digoxin and epinephrine increase contractility by making more calcium available to the cells. Beta blockers and calcium channel blockers do the opposite, and decrease contractility. Contractility is usually decreased in heart failure.

The factors listed above all contribute to determination of the **myocardial oxygen consumption rate (MVO2)**, or amount of oxygen used through the cardiac cycle. Figure 11 shows the components that contribute to coronary blood supply and demand. As patients with cardiac disease are treated, many of these components are altered to re-establish balance. For example, with a myocardial infarction, attempts are made to increase blood flow through CABG, stenting, angioplasty, thrombolytic agents, and the use of nitroglycerine. At the same time, efforts may be being made to decrease factors to the right which increase the demand for blood.

Extreme heart rates that are found with tachycardias may compromise coronary blood flow, as inadequate time may be being allowed for chamber filling. As a result, chest pain may occur and consciousness impaired. Resumption of normal rates brings this back into balance.

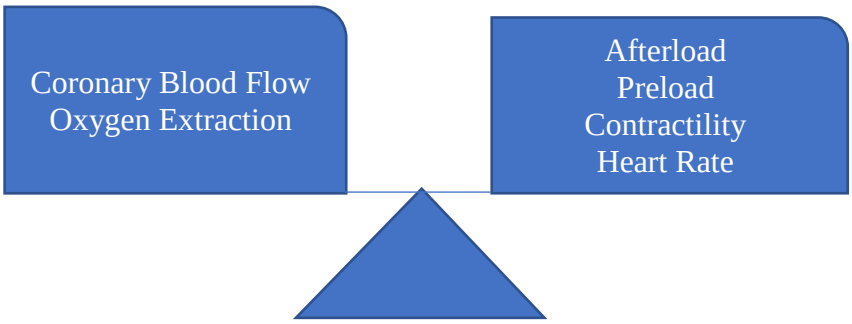

Figure 11: The balance of supply and demand.

IV. Autonomic Nervous System

Sympathetic Nervous System

Fibers of the **sympathetic nervous system** are present in the atria and the ventricles. The effect of sympathetic stimulation is to increase heart rate, increase conduction velocity, and increase contractility. Think **epinephrine** and **norepinephrine** (**neurotransmitter)** and think of the fight or flight response.

The specific cardiovascular effects of **adrenergic receptor activation** involve **alpha 1 and 2**, **beta 1 and 2**, and **dopaminergic** receptors.

Alpha 1 receptors are located in the heart and vascular smooth muscle, while alpha 2 receptors are found in just vascular smooth muscle.The effect exerted with alpha stimulation is to cause **increased systemic arterial and venous constriction (+++).**

Beta 1 receptors are located in the **SA** and **AV** nodes of the conduction system. The effects of Beta 1 stimulation include an increase in **heart rate** and **automaticity (++)**, and an increase in **contractility (+++)**.

Beta 2 receptors are located in the bronchial smooth muscle, blood vessels of the skeletal muscle, and the uterus. The effects of Beta 2 receptors include **systemic arterial and venous dilation (++)**, and to a lesser extent, **pulmonary artery dilation (+).**

Dopaminergic receptors include **DA 1** and **2**. DA1 is found in the renal, mesenteric and splanchnic vasculature, and DA2 in the peripheral vasculature. The effect of these receptors is to cause **systemic arterial dilation**.

RECEPTOR	LOCATION	EFFECTS
Alpha 1	Heart and vascular smooth muscle	Increased systemic arterial + and venous constriction +++
Alpha 2	Vascular smooth muscle	Increased systemic arterial + and venous constriction +++
Beta 1	SA and AV nodes	Increased heart rate and automaticity ++, and increase in contractility +++
Beta 2	Bronchial smooth muscle, blood vessels of the skeletal muscle, the uterus	Systemic arterial and venous dilation ++, and pulmonary artery dilation +
Dopaminergic (DA1)	Renal, mesenteric, and splanchnic vasculature	Systemic arterial dilation
Dopaminergic (DA2)	Peripheral vasculature	Systemic arterial dilation

Table 2. Summary of Adrenergic Receptor Activation

Parasympathetic Nervous System

Parasympathetic fibers are present in the atria and the ventricles, but exert their effect only in the atria. The effect of parasympathetic stimulation is to decrease heart rate, conduction velocity, and contractility. When we think of a vagal episode or vagotonia, parasympathetic stimulation is at work. This response is initiated with gagging, coughing, bearing down to give birth, having a bowel movement, and also can occur when groin pressure is applied during sheath removal in the cath lab. The parasympathetic effect on the vascular system is vasodilatation.

Self-Study Questions:

1. Discuss factors that control blood pressure.
2. Define systolic blood pressure.
3. Define diastolic blood pressure.
4. What is cardiac output?
5. What is the normal value for cardiac output?
6. What is cardiac index?
7. What are the four major periods during the cardiac cycle?
8. Describe the events that occur during atrial systole.
9. What occurs during atrial diastole?
10. What occurs during ventricular systole?
11. Define the isovolumic contractile phase.
12. What occurs during ventricular diastole?

13. Discuss the control of heart rate.
14. Define stroke volume.
15. Define preload, afterload, and contractility.
16. Discuss myocardial oxygen consumption rate and the various factors that influence this.
17. Differentiate the sympathetic nervous system effects on the heart from the parasympathetic.
18. List the sympathetic receptors that influence cardiac function.

V. Internal Homeostasis

There are ongoing efforts within the heart and vascular system to maintain homeostasis. This can be seen in a variety of situations and through a variety of mechanisms.

Heart rate is seen to increase or decrease, based on either an increase or a decrease in metabolic demands. During sleep, rate will decrease. In the athletically trained heart, stroke volume is increased and a normal or low rate is able to meet metabolic demands.

Heart rate increases when needs are great, as in fever, during times of exercise or stress, if contractility is decreased, or when a state of poor oxygenation exists.

The Frank-Starling law explains what we see with change in contractility. It states basically that the heart can stretch increasingly to handle any volume it receives…up to a point, to increase stroke volume.

Vascular tone

Arterioles have a role in regulating blood flow. One way this is achieved is through contributing resistance. Vascular smooth muscle is present in most of the arterial walls. Sympathetic stimulation plays a role via vasoconstriction in small arteries and arterioles. This effect causes pressure to rise, and through increasing the venous return to the heart, cardiac output is increased.

Baroreceptors

There are three types of **baroreceptors**: arterial, cardiopulmonary, and left ventricular. **Arterial baroreceptors** are responsible for the reflex control of the blood pressure. They are found in the adventitia of the carotid sinus (at bifurcation of carotid artery) and the aortic arch (between arch of aorta and bifurcation of subclavian artery). These receptors are mechanoreceptors: they respond to change in transmural pressure or stretch (ds/dt) of the vascular bed they are in. The carotid baroreceptors are sensitive to external compression or massage, which leads to decreased transmural pressure. These receptors are often referred to as pressoreceptors, but do not sense pressure; they actually sense change in stretch.

Cardiopulmonary receptors are low pressure baroreceptors that are located in the atria, ventricles, pulmonary veins and pulmonary arteries. A decrease in transmural pressure in the chamber or vessel results in a decrease in the rate of firing of these receptors. The receptors in ventricles are also sensitive to chemical stimuli. A response to a decrease in atrial or ventricular pressure leads to a reflex vasoconstriction of the skeletal muscle cutaneous resistance vessels, to increase BP.

Left ventricular baroreceptors are inhibitory or depressor receptors. These are located in the infero-posterior wall of the left ventricle. One of their actions is to modify the SA control of heart rate. These baroreceptors are thought to be responsible for some **syncope** through the **Bezold-Jarisch Reflex,** which is initiated in response to a forceful ventricular contraction on a relatively empty ventricle.

Baroreceptor Type	Location	Action
Arterial	Adventitia of carotid sinus Aortic arch	Responsible for reflex control of the blood pressure. Respond to change in transmural pressure or stretch of the vascular bed they are in.
Cardiopulmonary	Atria Ventricles Pulmonary veins Pulmonary arteries	Are low pressure baroreceptors. Decrease in transmural pressure in chamber or vessel results in a decrease in rate of firing of these receptors. Cause reflex vasoconstriction of skeletal muscle cutaneous resistance vessels, increasing BP.
Left Ventricular	Infero-posterior wall of left ventricle	Inhibitory or depressor receptors. Modify the SA control of heart rate. May be responsible for some syncope through the Bezold-Jarisch Reflex.

Table 3. Summary of Baroreceptor Actions

Chemoreceptors are found in the carotid body and aortic arch. They react to cellular oxygen deficiencies, increased CO2 levels, and pH. Based on their findings, the vasomotor center is stimulated and heart rate and blood pressure changes occur.

Natriuretic Peptides (NP) are noncontractile proteins that are released under certain abnormal physiologic events. They include **atrial natriurteic peptide (ANP), B-type natriuretic peptide (BNP, brain natriuretic peptide), C natriuretic peptide (CNP, C-Type),** and **urodilatin**

Stress or increase in cardiac chamber size leads to release of the natriuretic hormones. ANP originates in the atria and is released with atrial distension. BNP comes from the ventricular myocardium, and is released because of ventricular stimulation. A and B both cause vasodilatation, an increase in urinary sodium secretion, neurohormonal suppression, and anti-remodeling. B leads to increased diuresis, decreased renin and aldosterone, inhibition of the RAAS, and inhibition of sympathetic nervous activity.

CNP comes from the endothelium, and is released when there is shear stress in the endothelium. CNP causes vasodilation, decreased vascular smooth muscle growth, and decreased aldosterone levels

Natriuretic Peptides (NP)	**Location**	**Action**
Atrial natriuretic peptide (ANP)	atria	Released with atrial distension. Cause vasodilatation, increase in urinary sodium secretion, neurohormonal suppression, and anti-remodeling.
Brain natriuretic peptide (BNP)	Ventricular myocardium	Released because of ventricular stimulation. Cause vasodilatation, increase in urinary sodium secretion, neurohormonal suppression, and anti-remodeling. Leads to increased diuresis, decreased renin and aldosterone, inhibition of the RAAS, and inhibition of sympathetic nervous activity.
C natriuretic peptide (CNP)	Endothelium	Released when there is shear stress in the endothelium. Causes vasodilation, decreased vascular smooth muscle growth, and decreased aldosterone levels.

Table 4. Summary of Natriuretic Peptides

Renin-Angiotensin Aldosterone System (RAAS)

The **RAAS** system plays a major role in blood pressure and fluid regulation in the body. **Angiotensin II** is a product of **renin** production by the kidneys. Angiotensin II release occurs because of low blood pressure. Activation of **angiotensin I** receptors by angiotensin II leads to: vasoconstriction, sodium retention and water retention, increased **aldosterone** release, increased cellular growth, and increased sympathetic nervous activity. As a result, there is an increase in circulating blood volume and blood pressure rises.

Antidiuretic hormone (ADH) is **vasopressin**. The pituitary gland secretes ADH because the blood pressure is decreased; as a result, there is water retention and vasoconstriction.

Self-Study Questions:

1. What factor may increase or decrease heart rate?
2. Explain the Frank-Starling law.
3. Discuss the factors in vascular tone and blood pressure control.
4. Name three types of baroreceptors.
5. Where are baroreceptors located?
6. Describe the effects of each type of baroreceptor.
7. What are natriuretic peptides?
8. List three types natriuretic peptides.
9. What are the effects of the natriuretic peptides?
10. What is the role of chemoreceptors?
11. Discuss the RAAS system and the control of blood pressure.
12. Make a list of all factors that may increase or decrease blood pressure.

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