19. Cardiovascular System: Heart

I. Location of the Heart and the Pericardium

Location and position of the heart
The heart lies in the thoracic cavity directly posterior to the sternum. More specifically, the heart is enclosed in the pericardial cavity in the anterior portion of the mediastinum. The superior end of the heart, to which are attached the “great veins and arteries” of the heart, is called the base (which may be a bit confusing). The inferior tip of the heart is called the apex. The heart is offset slightly to the left of the midline, and it lies at an angle to the longitudinal axis of the body with the apex rotated slightly to the left.

Characteristics of the pericardium
The heart is covered by a set of membranes, called the pericardium. The pericardium has three layers (Fig. 19.6):
A. The outermost layer is called the fibrous pericardium. It is made of fibrous connective tissue. It is tough and keeps a constant shape as it anchors the heart in place.
B. The next layer in is called the parietal pericardium. It is directly attached to the fibrous pericardium. You should remember this and the next layer from the BIO 205 laboratory.
C. The innermost layer is called the visceral pericardium. This layer attaches directly to the heart and it is also considered the outermost layer of the heart.

Pericardial fluid fills the space between the parietal and visceral layers. It acts like motor oil, lubricating the heart and reducing friction as it pumps.

II. Heart Anatomy

Layers of the heart wall
The heart itself is constructed in three layers (Fig. 19.8b):
A. The outermost layer of the heart is the epicardium. This is the same structure as the visceral pericardium.
B. The myocardium is the middle layer. This is the thickest layer of tissue that makes up the heart, and it is made of cardiac muscle, blood vessels, and nerves.
C. The innermost layer is the endocardium, which is a thin layer of tissue that lines the chambers of the heart.

Heart chambers
The human heart has four chambers: the two smaller chambers are called atria, and they are located superiorly; the two larger chambers are called ventricles, and they are located inferiorly (Fig. 19.8a). Separating the ventricles is the interventricular septum. A layer of connective tissue separates and electrically insulates the atria from the ventricles.

Superior and inferior venae cavae bring blood to the right atrium from the superior and inferior portions of the body, respectively. Pulmonary veins bring blood to the left atrium from the lungs. Blood exits the right and left ventricles via the pulmonary trunk and aorta, respectively.
Heart valves
Blood from the right atrium is pumped into the right ventricle through the right atrioventricular (AV) valve. This valve is also known as the tricuspid valve, named for its three flaps (or cusps). When the right ventricle contracts, the tricuspid valve closes. Connective tissue fibers called the chordae tendineae prevent the valve from swinging back into the right atrium. From the right ventricle, blood is pumped through the pulmonary semilunar valve into the pulmonary trunk, which branches into left and right pulmonary arteries that travel separately to each lung.

Pulmonary veins lead from the lungs to the left atrium, which pumps blood through the left AV valve (also known as the bicuspid valve or mitral valve) into the left ventricle. When the left ventricle contracts, chordae tendineae prevent the left AV valve from swinging back into the left atrium. From the left ventricle blood is pumped through the aortic semilunar valve into the ascending aorta, the aortic arch, and the descending aorta.

I expect you to be familiar with the four chambers of the heart and the direction in which blood flows through the heart. Also, make sure you have a clear understanding of the functions of the valves (Fig. 19.10). How would blood flow if the valves did not exist?

Although we will look at blood vessels in more detail in the next chapter, we can see at this time that the circulatory system can be divided functionally into two separate circuits: a pulmonary circuit and a systemic circuit (Fig. 19.3). The pulmonary circuit involves the blood traveling to and from the lungs. Blood is pumped through the pulmonary circuit by the right side of the heart. The systemic circuit involves the blood traveling throughout the rest of the body. Blood is pumped through the systemic circuit by the left side of the heart.

Microscopic structure of cardiac muscle
In many ways, cardiac muscle cells are similar in structure and function to skeletal muscle fibers. For example, both have actin and myosin arranged into sarcomeres that give the tissue a striated appearance. Some differences you should be aware of are as follows:

Whereas skeletal muscle fibers are very long and cylindrical, cardiac muscle cells tend to be short (hence they are not called fibers), and they branch.

Structurally, skeletal muscle fibers are isolated from each other by their membranes. Although groups of skeletal muscle fibers may work together in a motor unit, each is innervated directly by a motor neuron and fires its own action potential. Cardiac muscle cells are structurally linked by structures called intercalated discs (Fig. 19.11). These discs contain gap junctions that allow action potentials from cell to cell. The term functional syncytium is used to describe the fact that the branches and gap junctions link the entire myocardium into a single coordinated unit.

The action potential of a skeletal muscle fiber has a very short refractory period (about 1-2 ms). This contributes to the ability of a skeletal muscle fiber to enter a state of tetanus. Cardiac muscle cells have long refractory periods (about 250 ms). This normally prevents a cardiac
muscle cell from entering a state of tetanus. Why would you not want cardiac muscle to enter tetanus?

III. Coronary Vessels: Blood Supply of the Heart Wall

Coronary arteries
We all know the heart pumps blood for all of the body, but we often overlook the fact that the heart needs its own supply of blood. Because the heart works continuously, cardiac muscle cells require a constant supply of oxygen and nutrients. Branching off the aorta (just as it leaves the heart) are the coronary arteries, which supply the heart itself with blood (Fig. 19.13).

The right coronary artery supplies blood to the right atrium, portions of both ventricles, and portions of the heart’s electrical conduction system. The left coronary artery supplies blood to the left atrium, portions of both ventricles, and the interventricular septum. Near the apex of the heart, branches of the right and left coronary arteries merge at junctions called anastomoses. Because the blood vessels merge, there is collateral circulation of the blood flow. This means that blood can reach much of the heart via different pathways. This collateral circulation may allow blood to flow to the entire heart even when an artery is blocked.

A heart attack typically results from blockage of a coronary artery, which cuts off the supply of oxygen to part of the heart.

Coronary veins
Cardiac veins bring deoxygenated blood from capillaries of the heart to the coronary sinus, which dumps blood into the right atrium.

IV. Anatomic Structures Controlling Heart Activity

Beating of the heart is a process that results from cooperative efforts of two types of cardiac muscle fibers: Contractile cells make up the bulk of the myocardium, and they are specialized to contract. Given the exceptions noted above, these cells function in a manner similar to skeletal muscle cells. Autorhythmic cells make up the conducting system of the heart and control electrical activity of the heart. Unlike skeletal muscle cells and contractile cells, autorhythmic cells have unstable resting potentials that spontaneously move toward threshold and lead to action potentials. This property is known as automaticity. Thus, autorhythmic cells will generate action potentials on their own. Hitting threshold leads to the opening of many voltage-gated Ca^{++} channels that allow an influx of Ca^{++}. Thus the action potential of an autorhythmic cell is due to influx of calcium rather than sodium.

The heart’s conduction system
The rate at which the heart beats is determined by both intrinsic and extrinsic control mechanisms. Intrinsic control is related to the property of automaticity described previously. Basically, the heart tends to beat at its own rhythm. However, the rate at which the heart beats
can be affected by extrinsic (external) factors. First we will examine the intrinsic control.

Cells of the conducting system cannot maintain stable resting potentials. After repolarization, the membrane potential inevitably drifts toward threshold and depolarization. The rate of spontaneous depolarization varies in different regions of the conducting system, but it is fastest in the **sinoatrial (SA) node** (Figs. 19.14 and 19.17). Therefore, depolarization of the SA node sets the pace for the heart, and the SA node is referred to as the **pacemaker** of the heart. When fibers of the sinoatrial node contract, the electrical impulse spreads across both atria and causes the atria to contract.

The layer of connective tissue between the atria and ventricles prevents the direct spread of this impulse from the atria to the ventricles. A structure called the **atrioventricular (AV) node** carries the impulse to the ventricles, but at a slower speed (Figs. 19.14 and 19.17). This creates a delay between excitation of the atria and excitation of the ventricles. The **atrioventricular bundle** (or **bundle of His**) and **Purkinje fibers** then rapidly spread the impulse through the ventricles and cause the ventricles to contract. Why is it important to have a delay between excitation of the atria and excitation of the ventricles?

In lab, you will learn how the electrical activity of the heart can be monitored with an instrument called an **electrocardiograph** (Fig. 19.20). Abnormal function of the heart’s conduction system can result in a number of serious clinical problems, generally characterized as **arrhythmias**. **Fibrillation** is the term used to describe rapid, uncoordinated contractions of cardiac muscle fibers. How does fibrillation affect the heart’s ability to pump blood?

**Innervation of the heart**
Although the heart has its own intrinsic rhythm, the heart rate can be adjusted extrinsically by control centers in the medulla (Fig. 19.14). The **cardioacceleratory center** of the medulla sends signals to the heart via sympathetic pathways in spinal nerves T1-T5; these signals accelerate the heart rate, and they also increase contractility (the force with which the heart contracts). What neurotransmitter is released onto the heart when the cardioacceleratory center is active?

The **cardioinhibitory center** of the medulla sends signals to the heart via parasympathetic pathways in the vagus nerves; these signals slow the heart rate. What neurotransmitter is released onto the heart when the cardioinhibitory center is active?

**V. The Cardiac Cycle**

The period from the beginning of one heartbeat to the beginning of the next is called the **cardiac cycle** (Fig. 19.21). Contraction of a portion of the heart makes up a phase of the cardiac cycle called **systole**. Systole is followed by relaxation, called **diastole**. One key to understanding the cardiac cycle is understanding that blood will tend to flow from an area of high pressure to an area of low pressure.
Events of the cardiac cycle
Because the beating of a heart is a cyclic process, we can start our description anywhere. We will begin our description at a point where all four chambers are in diastole, and the atria are about to begin contraction . . .

1. Atrial systole. The ventricles have already been filling with blood passively while the heart was relaxed. At the point when the ventricles are about 70% full and pressure has begun to build in the ventricles, the atria contract. Contraction of the atria is called atrial systole, and it lasts about 100 ms. This pumps additional blood into the ventricles as pressure generated in the atria exceeds pressure in the ventricles. The quantity of blood in each ventricle is maximal after atrial systole, and the volume of blood in the ventricles at this time is referred to as the end-diastolic volume (EDV). You should note that the atria do not do much in the way of filling the ventricles, they are already mostly full by the time the atria contract!

2. Early ventricular systole. As the atria relax after atrial systole, the ventricles begin to contract in a process called ventricular systole. As pressure in the ventricles rises, the AV valves shut and for a time all valves of the heart are closed. This phase is referred to as isovolumetric contraction.

3. Late ventricular systole. Eventually, the pressure inside the ventricles rises sufficiently to open the semilunar valves, and blood is ejected into the pulmonary trunk and the aorta. The ventricles eject a quantity of blood referred to as the stroke volume (SV). SV is usually equal to about 60% of the EDV, and this percentage is referred to as the ejection fraction. The volume of blood in the heart at the end of ventricular systole is the end-systolic volume (ESV).

4. Early ventricular diastole. Contraction of the ventricles is followed by ventricular diastole. As the ventricles begin to relax, pressure in the ventricles drops rapidly. At this point, pressure in the aorta exceeds that in the ventricles, and the semilunar valves close. For a brief period, all four valves are again closed at the same time. This phase is referred to as isovolumetric relaxation. The ventricles relax, but the volume does not change.

5. Late ventricular diastole. As pressure in the ventricles drops further, the AV valves open and blood flows passively from the atria into the ventricles. This will bring us back to step 1.

Examine Fig. 19.22 and see how much of this you can put together.

VI. Cardiac Output

Introduction to cardiac output
The volume of blood pumped per unit of time is the cardiac output (CO) of the heart:

\[
CO \text{ (ml/min)} = HR \text{ (beat/min)} \times SV \text{ (ml/beat)}
\]

where HR is the heart rate. Changes in either HR or SV will affect cardiac output.
Variables that influence cardiac output
Stroke volume is the difference between the EDV and the ESV:

\[ SV = EDV - ESV \]

Changes in either EDV or ESV will affect SV.

The EDV is affected largely by two factors:

A. **Filling time** is the duration of ventricular diastole. The longer the filling time, the greater the EDV.

B. **Venous return** is the rate of flow back to the heart from the body. The greater the rate of venous return, the greater the EDV.

The ESV is affected largely by three factors:

A. **Preload** is the degree to which the heart is stretched during ventricular diastole. The greater the stretching during preload, the greater the force with which the cardiac muscle will contract and the greater the SV will be. This principle was first described as the **Frank-Starling principle**. Increased preload leads to decreased ESV.

B. **Contractility** is the force produced during contraction independent of preload. Various factors may either increase contractility or decrease contractility. For example, the sympathetic nervous system increases contractility via the release of norepinephrine. Increased contractility leads to decreased ESV.

C. **Afterload** is the amount of force the ventricles must produce to eject blood through the semilunar valves. The most common cause of an increase in afterload is obstruction of the blood vessels. This can be particularly harmful if there has been damage to the myocardium. An increase in the afterload increases the ESV.