**CHAPTER 20**

**LECTURE OUTLINE**

1. **INTRODUCTION**
	1. The cardiovascular system consists of the blood, heart, and blood vessels.
	2. The heart is the pump that circulates the blood through an estimated 75,000 miles of blood vessels.
	3. The study of the normal heart and diseases associated with it is known as cardiology.
2. **ANATOMY OF THE HEART**
	1. **Location of the heart**
		1. The heart is situated between the lungs in the mediastinum with about two-thirds of its mass to the left of the midline (Figure 20.1).
		2. Because the heart lies between two rigid structures, the vertebral column and the sternum, external compression on the chest can be used to force blood out of the heart and into the circulation. (Clinical Connection)
	2. **Pericardium**
		1. The heart is enclosed and held in place by the pericardium.
			1. The pericardium consists of an outer fibrous pericardium and an inner serous pericardium (Figure 20.2a).
			2. The serous pericardium is composed of a parietal layer and a visceral layer.
			3. Between the parietal and visceral layers of the serous pericardium is the pericardial cavity, a potential space filled with pericardial fluid that reduces friction between the two membranes.
			4. An inflammation of the pericardium is known as pericarditis. Associated bleeding into the pericardial cavity compresses the heart (cardiac tamponade) and is potentially lethal (Clinical Connection).
	3. **Layers of the Heart Wall**
		1. The wall of the heart has three layers: epicardium, myocardium, and endocardium (Figure 20.2a).
		2. The epicardium consists of mesothelium and connective tissue, the myocardium is composed of cardiac muscle, and the endocardium consists of endothelium and connective tissue (Figure 20.2c).
		3. Inflammations of the myocardium and endocardium are myocarditis and endocarditis, respectively (Clinical Connection)
	4. **Chambers of the Heart**
		1. The chambers of the heart include two upper atria and two lower ventricles (Figure 20.3).
		2. On the surface of the heart are the auricles and sulci.
			1. The auricles are small pouches on the anterior surface of each atrium that slightly increase the capacity of each atrium.
			2. The sulci are grooves that contain blood vessels and fat and separate the chambers.
	5. **Right Atrium**
		1. The right atrium receives blood from the superior and inferior vena cava and the coronary sinus (Figure 20.4a).
		2. In the septum separating the right and left atria is an oval depression, the fossa ovalis, which is the remnant of the foramen ovale.
		3. Blood passes from the right atrium into the right ventricle through the tricuspid valve.
	6. **Right Ventricle**
		1. The right ventricle forms most of the anterior surface of the heart.
		2. Blood passes from the right ventricle to the pulmonary trunk via the pulmonary semilunar valve.
	7. **Left Atrium**
		1. The left atrium receives blood from the pulmonary veins.
		2. Blood passes from the left atrium to the left ventricle through the bicuspid (mitral) valve.
	8. **Left Ventricle**
		1. The left ventricle forms the apex of the heart.
		2. Blood passes from the left ventricle through the aortic semilunar valve into the aorta.
		3. During fetal life the ductus arteriosus shunts blood from the pulmonary trunk into the aorta. At birth the ductus arteriosus closes and becomes the ligamentum arteriosum.
	9. **Myocardial Thickness and Function**
		1. The thickness of the myocardium of the four chambers varies according to the function of each chamber.
		2. The atria walls are thin because they deliver blood to the ventricles.
		3. The ventricle walls are thicker because they pump blood greater distances (Figure 20.4a).
			1. The right ventricle walls are thinner than the left because they pump blood into the lungs, which are nearby and offer very little resistance to blood flow.
			2. The left ventricle walls are thicker because they pump blood through the body where the resistance to blood flow is greater.
	10. **Fibrous Skeleton of the Heart**
		1. The fibrous skeleton of the heart forms the foundation for which the heart valves attach, serves as points of insertion for cardiac muscle bundles, prevents overstretching of the valves as blood passes through them, and acts as an electrical insulator that prevents direct spread of action potentials from the atria to the ventricles (Figure 20.5).
3. **HEART VALVES AND CIRCULATION OF BLOOD**
	1. Valves open and close in response to pressure changes as the heart contracts and relaxes.
	2. **Operation of the atrioventricular valves**
		1. Atrioventricular (AV) valves prevent blood flow from the ventricles back into the atria (Figure 20.6a and c).
		2. Back flow is prevented by the contraction of papillary muscles tightening the chordae tendinae which prevent the valve cusps from everting.
	3. **Operation of the semilunar valves**
		1. The semilunar (SL) valves allow ejection of blood from the heart into arteries but prevent back flow of blood into the ventricles (Figure 20.6).
		2. Semilunar valves open when pressure in the ventricles exceeds the pressure in the arteries.
		3. Heart valve disorders affect blood flow through the heart.(Clinical Connection)
	4. **Systemic and Pulmonary Circulations**
		1. The left side of the heart is the pump for the systemic circulation. It pumps oxygenated blood from the lungs out into the vessels of the body.
		2. The right side of the heart is the pump for the pulmonary circulation. It receives deoxygenated blood from the body and sends it to the lungs for oxygenation.
		3. Figure 20.7 reviews the route of blood flow through the chambers and valves of the heart and the pulmonary and systemic circulations.
	5. **Coronary Circulation**
		1. The flow of blood through the many vessels that pierce the myocardium of the heart is called the coronary (cardiac) circulation; it delivers oxygenated blood and nutrients to and removes carbon dioxide and wastes from the myocardium (Figure 20.8b).
		2. The principal arteries, branching from the ascending aorta and carrying oxygenated blood, are the right and left coronary arteries.
		3. Deoxygenated blood returns to the right atrium primarily via the principal vein, the coronary sinus.
		4. When blockage of a coronary artery deprives the heart muscle of oxygen, reperfusion may damage the tissue further. This damage is due to free radicals. Drugs that lessen reperfusion damage after a heart attack are being developed.
		5. Low levels of oxygen carried to the myocardium may result in weakened heart cells (or myocardial ischemia), and if the damage is permanent then this is called myocardial infarction (MI) or heart attack (Clinical connection)
4. **CARDIAC MUSCLE AND THE CARDIAC CONDUCTION SYSTEM**
	1. **Histology of Cardiac Muscle**
		1. Compared to skeletal muscle fibers, cardiac muscle fibers are shorter in length, larger in diameter, and squarish rather than circular in transverse section (Figure 20.9). They also exhibit branching.
		2. Cardiac muscles have the same arrangement of actin and myosin, and the same bands, zones, and Z discs as skeletal muscles.
		3. They do have less sarcoplasmic reticulum than skeletal muscles and require Ca+2 from extracellular fluid for contraction.
		4. They form two separate functional networks in the heart: the atrial and the ventricular networks.
			1. Fibers within the networks are connected by intercalated discs, which consist of desmosomes and gap junctions.
			2. The intercalated discs allow the fibers in the network to work together so that each network serves as a functional unit.
			3. Regeneration of heart cell has much potential in treating heart problems (Clinical connection)
	2. **Autorhythmic Cells: The Conduction System**
		1. Cardiac muscle cells are autorhythmic cells because they are self-excitable. They repeatedly generate spontaneous action potentials that then trigger heart contractions.
			1. These cells act as a pacemaker to set the rhythm for the entire heart.
			2. They form the conduction system, the route for propagating action potential through the heart muscle.
		2. Components of this system are the sinoartrial (SA) node (pacemaker), atrioventricular (AV) node, atrioventricular bundle (bundle of His), right and left bundle branches, and the conduction myofibers (Purkinje fibers) (Figure 20.10)
		3. Signals from the autonomic nervous system and hormones, such as epinephrine, do modify the heartbeat (in terms of rate and strength of contraction), but they do not establish the fundamental rhythm.
		4. Artificial pacemakers stimulate heart muscle and provide a normal rhythm (Clinical Connection).
	3. **Action potential and contraction of contractile fibers**
		1. An impulse in a ventricular contractile fiber is characterized by rapid depolarization, plateau, and repolarization (Figure 20.11).
		2. The refractory period of a cardiac muscle fiber (the time interval when a second contraction cannot be triggered) is longer than the contraction itself (Figure 20.11).
	4. **ATP production in cardiac muscle**
		1. Cardiac muscle relies on aerobic cellular respiration for ATP production.
		2. Cardiac muscle also produces some ATP from creatine phosphate
		3. The presence of creatine kinase (CK) in the blood indicates injury of cardiac muscle usually caused by a myocardial infarction.
	5. **Electrocardiogram**
		1. Impulse conduction through the heart generates electrical currents that can be detected at the surface of the body. A recording of the electrical changes that accompany each cardiac cycle (heartbeat) is called an electrocardiogram (ECG or EKG).
			1. The ECG helps to determine if the conduction pathway is abnormal, if the heart is enlarged, and if certain regions are damaged.
			2. Figure 20.12 shows a typical ECG.
			3. In a typical Lead II record, three clearly visible waves accompany each heartbeat.
		2. Correlation of ECG waves with atrial and ventricular systole
			1. A normal ECG consists of a P wave (atrial depolarization - spread of impulse from SA node over atria), QRS complex (ventricular depolarization - spread of impulse through ventricles), and T wave (ventricular repolarization).
			2. The P-Q (PR) interval represents the conduction time from the beginning of atrial excitation to the beginning of ventricular excitation.
			3. The S-T segment represents the time when ventricular contractile fibers are fully depolarized, during the plateau phase of the impulse.
			4. Figure 20.13 illustrates the timing and route of action potential depolarization and repolariztion through the conduction system and myocardium
5. **THE CARDIAC CYCLE**
	1. A cardiac cycle consists of the systole (contraction) and diastole (relaxation) of both atria, rapidly followed by the systole and diastole of both ventricles (Figure 20.13).
	2. Pressure and volume changes during the cardiac cycle
		1. During a cardiac cycle atria and ventricles alternately contract and relax forcing blood from areas of high pressure to areas of lower pressure.
		2. Figure 20.14 shows the relation between the ECG and changes in atrial pressure, ventricular pressure, aortic pressure, heart sounds . ventricular volume and cycle phases during the cardiac cycle.
	3. The act of listening to sounds within the body is called auscultation, and it is usually done with a stethoscope. The sound of a heartbeat comes primarily from the turbulence in blood flow caused by the closure of the valves, not from the contraction of the heart muscle (Figure 20.15).
		1. The first heart sound (lubb) is created by blood turbulence associated with the closing of the atrioventricular valves soon after ventricular systole begins.
		2. The second heart sound (dupp) represents the closing of the semilunar valves close to the end of the ventricular systole.
		3. A heart murmur is an abnormal sound that consists of a flow noise that is heard before, between, or after the lubb-dupp or that may mask the normal sounds entirely. Some murmurs are caused by turbulent blood flow around valves due to abnormal anatomy or increased volume of flow. Not all murmurs are abnormal or symptomatic, but most indicate a valve disorder (Clinical Connection).
6. **CARDIAC OUTPUT**
	1. Since the body’s need for oxygen varies with the level of activity, the heart’s ability to discharge oxygen-carrying blood must also be variable. Body cells need specific amounts of blood each minute to maintain health and life.
	2. Cardiac output (CO) is the volume of blood ejected from the left ventricle (or the right ventricle) into the aorta (or pulmonary trunk) each minute.
		1. cardiac output equals the stroke volume, the volume of blood ejected by the ventricle with each contraction, multiplied by the heart rate, the number of beats per minute.
		2. Cardiac reserve is the ratio between the maximum cardiac output a person can achieve and the cardiac output at rest.
	3. **Regulation of Stroke Volume**
		1. Three factors regulate stroke volume: preload, the degree of stretch in the heart before it contracts; contractility, the forcefulness of contraction of individual ventricular muscle fibers; and afterload, the pressure that must be exceeded if ejection of blood from the ventricles is to occur.
			1. Preload: Effect of Stretching
				1. According to the Frank-Starling law of the heart, a greater preload (stretch) on cardiac muscle fibers just before they contract increases their force of contraction during systole.
				2. The Frank-Starling law of the heart equalizes the output of the right and left ventricles and keeps the same volume of blood flowing to both the systemic and pulmonary circulations.
			2. Myocardial contractility, the strength of contraction at any given preload, is affected by positive and negative inotropic agents.
				1. Positive inotropic agents increase contractility and negative inotropic agents decrease contractility.
				2. Thus, for a constant preload, the stroke volume increases when positive inotropic agents are present and decreases when negative inotropic agents are present.
			3. The pressure that must be overcome before a semilunar valve can open is the afterload.
				1. In congestive heart failure, blood begins to remain in the ventricles increasing the preload and ultimately causing an overstretching of the heart and less forceful contraction (Clinical Connection).
	4. **Regulation of Heart Rate**
		1. Cardiac output depends on heart rate as well as stroke volume. Changing heart rate is the body’s principal mechanism of short-term control over cardiac output and blood pressure. Several factors contribute to regulation of heart rate.
		2. Autonomic regulation of the heart
			1. Nervous control of the cardiovascular system stems from the cardiovascular center in the medulla oblongata (Figure 20.16).
			2. Proprioceptors, baroreceptors, and chemoreceptors monitor factors that influence the heart rate.
			3. Sympathetic impulses increase heart rate and force of contraction; parasympathetic impulses decrease heart rate.
		3. Chemical regulation of heart rate
			1. Heart rate affected by hormones (epinephrine, norepinephrine, thyroid hormones).
			2. Ions (Na+, K+, Ca+2) also affect heart rate.
			3. Other factors such as age, gender, physical fitness, and temperature also affect heart rate.
			4. Figure 20.17 summarizes the factors influencing both stroke volume and heart rate in the overall increase of cardiac output.
7. **EXERCISE AND THE HEART**
	1. Sustained exercise increases oxygen demand in muscles.
	2. Among the benefits of aerobic exercise (any activity that work large body muscles for at least 20 minutes, preferably 3-5 times per week) are increased cardiac output, increased HDL and decreased triglycerides, improved lung function, decreased blood pressure, and weight control.
8. **HELP FOR FAILING HEARTS**
	1. a heart fails, a person’s mobility decreases. Heart transplants may help such individuals (Figure 20.18).. Other possibilities include cardiac assist devices and surgical procedures. Table 20.1 describes several devices and procedures.
9. **DEVELOPMENT OF THE HEART**
	1. The heart develops from mesoderm before the end of the third week of gestation (Figure 20.19).
	2. The endothelial tubes develop into the four-chambered heart and great vessels of the heart (Figure 20.20).
10. **DISORDERS: HOMEOSTATIC IMBALANCES**
	1. Coronary artery disease (CAD), or coronary heart disease (CHD), is a condition in which the heart muscle receives an inadequate amount of blood due to obstruction of its blood supply. It is the leading cause of death in the United States each year. The principal causes of obstruction include atherosclerosis, coronary artery spasm, or a clot in a coronary artery.
		1. Risk factors for development of CAD include high blood cholesterol levels, high blood pressure, cigarette smoking, obesity, diabetes, “type A” personality, and sedentary lifestyle.
		2. Atherosclerosis is a process in which smooth muscle cells proliferate and fatty substances, especially cholesterol and triglycerides (neutral fats), accumulate in the walls of the medium-sized and large arteries in response to certain stimuli, such as endothelial damage (Figure 20.18).
		3. Diagnosis of CAD includes such procedures as cardiac catherization and cardiac angiography.
		4. Treatment options for CAD include drugs and coronary artery bypass grafting (Figure 20.19).