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Anatomical Survey: Observation of Physical and Functional Changes due to Congestive Heart Failure

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## **Abstract**

To gain a better understanding and appreciation of the complexity of cardiopulmonary anatomy and physiology as well the greatly varied nature heart disease, this project emphasized observations of both typical and atypical cardiopulmonary conditions. Specifically, comparisons were made between a heart affected by congestive heart failure (CHF) and a non-affected heart. The hearts of a male (CHF patient) and female cadaver were extracted and dissected. Qualitative observations and basic measurements were taken and a comprehensive literature review was conducted to compare and contrast the expected functionality of the subject's cardiopulmonary systems. Observations and measurements taken included heart size, weight, general features of the pericardium, myocardium, and endocardium, as well as qualitative observations of the lungs. These measurements were then compared to research and descriptions available in various literary sources (as working with donated cadaver subjects prevented in-depth analysis of living conditions). Both qualitative and quantitative comparisons of the normal and CHF hearts indicate that congestive heart failure greatly changes many aspects of cardiac functionality. It should be concluded that more research is required to understand the extent of CHF changes and the effects on internal systems as well as better methods of prevention and disease improvement.

For much of written history, mankind has sought to understand the intricate mechanisms and complex biological systems that compose the human body. In fact, many of the technological and pharmacological advancements that have been seen in the past two centuries have been spurred by two primary sources: the desire to understand pathology and its effects on people and the desire to rid people of those pathologies. Despite efforts that would have astounded the ancient people who created the framework for modern biology and medicine, man has made seemingly little progress. This rather bleak position is not without support: the CDC, just last year, indicated that cardiovascular disease remained the number one cause of death with an annual mortality of 614,348 (CDC, 2015). This was the case in spite of the report that 1 of every 6 dollars spent in the US healthcare system is used on treatment of cardiovascular disease (NCSL, 2016). As research continues to probe the unexplored facets of cardiopulmonary health, the nation must strongly promote education and understanding of the cardiovascular and pulmonary systems to combat rising mortality rates. The purpose of this discussion, therefore, is to provide an overview of both the important anatomical features of those systems and their physiological mechanisms, as well as an explanation of congestive heart failure (a common pathology that affects both the heart and lungs).

The heart is the primary structure of the cardiovascular system and arguably the most important organ in the body. In anatomy it is all at once, simple yet complex. The heart is a two-sided pump that sits in the center of the chest, between the left and right lung (NHLBI, 2011). Specifically, the heart rests within the cardiac impression formed in the left lung. It is encased by a series of membranous sacs that form the pericardium. The outermost layer, or fibrous pericardium, roots the heart to the diaphragm and back of the sternum (Grine, 2014). Deep to the fibrous pericardium is the serous pericardium: a sac that is divided into the parietal serous

pericardium and visceral serous pericardium (also referred to as the epicardium, due to its adherence to the outermost layer of the heart) (Grine, 2014). The heart itself is composed of four, blood-containing chambers: two atria and two ventricles. Atria are filling chambers that collect incoming blood (either from the lungs via the pulmonary veins or the body via the inferior and superior vena cava). These chambers are closed off from the more inferior ventricles by a pair of valves (tricuspid on the right side, bicuspid on the left). The ventricles, or pumping chambers, are made up of specialized muscle tissue called myocardium. They fill with blood from the atria and then forcefully contract, ejecting blood on both sides through yet another set of valves (the pulmonary semilunar valve on the right side and aortic semilunar valve on the left) into the circulation. The right ventricle ejects blood into the pulmonary trunk, where it can be carried to the lungs for oxygenation. Conversely, the left ventricle pumps oxygenated blood.

Aside from the structures involved with pumping, the heart also contains the base for all major blood vessels in the body. These can be divided into systemic and coronary arteries and veins. As their names imply, the systemic vessels carry blood to and from the tissues of the entire body while coronary vessels provide the heart with its requisite supply. The main systemic artery is the aorta. The aorta begins at the left ventricle and continues across the superior aspect of the heart, its many branches supplying oxygenated blood. The aorta itself is divided into ascending and descending sections as well as the aortic arch. The arch gives rise to the left and right subclavian arteries as well as the left and right carotid arteries (Grine, 2014). The descending aorta is continuous with the abdominal aorta and eventually gives rise to several essential vessels in the lower body such as the celiac trunk, superior and inferior mesenteric arteries, and iliac branches. Venous return of blood to the heart is handled by branches of the vena cava. Like the aorta, the vena cava has superior and inferior branches. Notable examples utilized in blood return

include jugular and brachiocephalic veins in the upper body and inferior and superior mesenteric veins in the lower body. Finally, a set of pulmonary arteries carries blood ejected by the right ventricle to the lungs for oxygenation and pulmonary veins return that blood to the left atrium to be pumped into bodily circulation.

The aforementioned coronary vessels carry blood to and from cardiac tissue and stretch across both anterior and posterior surfaces of the organ. Like the systemic vessels, coronary blood vessels are divided into arteries and veins. Moreover, the vessels of the heart are divided into left and right coronary branches (Grine, 2014, pg 306). The left coronary supply branches off into the anterior interventricular and circumflex arteries, and the right branches into the posterior interventricular and marginal arteries (Grine, 2014, pg 306). Venous return of the coronary supply is handled by the great, middle and small cardiac veins and the coronary sinus (where blood is emptied back into the right atrium to be oxygenated).

Just the anatomy of the heart does little to explain how it functions within the cardiovascular system. While anatomy provides details about the structures involved, physiology explains the biochemical and physical mechanisms that give rise to the function of those structures. Cardiac physiology has two primary components that interact to create a working heart: an electrical and mechanical component. That is not to say that neurological input should be ignored, but the heart can function without neurological signals and so they will be excluded.

The heart relies on an interconnected network of electrically conductive cells to carry the signals required for cardiac function. These signals are able to be generated due to the existence of cell membrane potentials. Cardiac cells, like other cells in the body, have a constantly maintained electrical potential across their membranes (Klabunde, 2012, pg. 10). Electrical potential can be viewed as the energy that moves charge in space. In the case of cardiac cells,

potential refers to the energy used to move essential ions in and out of the cell (especially  $\text{Na}^+$ ,  $\text{K}^+$ , and  $\text{Ca}^{2+}$ ). Before a signal is generated, a cardiomyocyte is considered to be at rest and its electrical potential is about  $-90 \text{ mV}$  (Klabunde, 2012, pg. 10). The negative potential is created by a constant leak of  $\text{K}^+$  from the cell into the extracellular fluid through resting channels that are always open. This leak, in turn, is facilitated by a constantly maintained ionic gradient where  $\text{K}^+$  is highly concentrated within the cell and less concentrated in the extracellular fluid (the fluid surrounding the cells). Also contributing to the electrical potential is the high concentration of  $\text{Na}^+$  and  $\text{Ca}^{2+}$  found in the extracellular fluid that does not exist within the cell. Cells actively maintain the ionic concentrations by using a pump referred to as  $\text{Na}^+/\text{K}^+$ -ATPase that pumps sodium out of the cell and potassium into the cell (Klabunde, 2012, pg. 13). The constant gradient sets the cardiac cells up for action potentials: electrical depolarization and repolarization (Klabunde, 2012, pg. 17).

Action potentials are the primary signal for cardiomyocytes to begin working. Action potentials in the heart are divided into two types: pacemaker and nonpacemaker (Klabunde, 2012, pg. 17). Pacemaker action potentials are capable of being spontaneously generated by pacemaker cells (discussed in a later section) while nonpacemaker action potentials require a current to be transferred between adjacent cells (via cellular intercalations). Cardiac action potentials are quite different from those seen in skeletal muscles in that they are much longer at 200-400 milliseconds rather than 1-2 milliseconds (Klabunde, 2012, pg. 17). When an action potential is triggered, voltage-gated channels open that allow the cell to be flooded with both sodium and calcium. This response is replicated throughout the myocardium.

Calcium is central to the contraction of ventricles through the same mechanisms seen in skeletal muscle.  $\text{Ca}^{2+}$  enters the cell from the extracellular fluid via L-type, voltage-dependent

channels. This initial influx is required to trigger receptors found in muscle tissue called ryanodine-sensitive calcium channels. This, in turn, results in a massive release of calcium from the sarcoplasmic reticula of the activated cardiomyocytes. The released calcium then binds to troponin-tropomyosin complexes within the muscles, causing them to reconfigure into a shape that allows actin and myosin components of muscle to interact with each other (Klabunde, 2012, pg. 44). This is the biochemical process that results in ventricular contraction. When the action potential is done, the cells return to -90 mV through the active removal of sodium and calcium from the intracellular fluid as well as voltage-gated potassium channels that open. This eventually returns the cell to resting state.

As previously stated, the heart is able to generate the required electrical signals independent of neurological input. This is because of the heart's intrinsic pacemaker cells, collectively referred to as the sinoatrial (SA) node. Located in the posterior wall of the right atrium, the SA node is capable of spontaneously generating action potentials to facilitate the pumping of the heart (Klabunde, 2012, pg. 18). After a signal is generated at the SA node, it travels through intermodal fibers to the atrioventricular (AV) node (a clump of tissue near the tricuspid valve) (McArdle, Katch, & Katch, 2015, pg. 326). Here, the signal stalls to give the atria an opportunity to contract and squeeze blood into the ventricles before traveling down a 1 cm long bundle of fibers referred to as the bundles of His (AV bundle) (McArdle, Katch, & Katch, 2015, pg. 326). Finally, the electrical signal travels up the sides of both ventricles via the Purkinje system, sending the signal into cells that carry the signal through intercalated disks to all of their neighboring cardiomyocytes until contraction is achieved (McArdle, Katch, & Katch, 2015, pg. 326-327)



The electrical system of the heart, while absolutely critical, is only half of cardiac physiology. The mechanical processes of cardiac function also give rise to essential features of its physiological state. These processes occur during either of two phases: systole or diastole. Ventricular systole refers to the contraction phase of cardiac action while ventricular diastole is the relaxation phase of the muscle fibers (McArdle, Katch, & Katch, 2015, pg. 309). Both phases together represent the primary activity of the heart, referred to as the cardiac cycle. During diastole, the ventricles are not contracting and the atrioventricular valves are closed initially. The atria fill with blood, from various parts of the vasculature and the valves open due to a pressure differential between the atria and ventricles. This allows blood to passively pour into the ventricles, aided by atrial contractions that squeeze any blood that remains into the chamber. The amount of blood in the ventricle at the end of diastole is referred to as end-diastolic volume (McArdle, Katch, & Katch, 2015, pg. 346). During the second phase of systole, ventricles undergoes isovolumetric contraction, increasing interventricular pressure until the semilunar valves open and blood is ejected, either into the pulmonary trunk on the right side or the aorta on the left. The amount of blood left in the ventricle after systole is referred to as end-systolic volume (McArdle, Katch, & Katch, 2015, pg. 342-343). The difference between end-diastolic and end-systolic volume is stroke volume, and represents the amount of blood ejected per beat (McArdle, Katch, & Katch, 2015, pg. 342). Stroke volume and heart rate can be used to determine cardiac output, the amount of blood moved per minute (McArdle, Katch, & Katch, 2015, pg. 342). The equation for doing so is as follows:

$$\text{Cardiac Output} = \text{Stroke Volume} \times \text{Heart Rate}$$

From this equation, it can be easily observed how cardiac efficiency can be improved or reduced: increases or decreases of either stroke volume or heart rate could dramatically affect

overall output. This point brings up another important physiological aspect of heart mechanics: ventricular contractility. Contractility essentially refers to the strength with which the ventricles contract during systole. This aspect of the cardiomyocytes is affected by a number of factors including neurohormonal influence and cardiovascular training (McArdle, Katch, & Katch, 2015, pg. 345). One of the most important factors that increases contractility, though, is stretch activation due to increased filling of the ventricles. This is illustrated by Starling's Law of the Heart which states that contractility is directly proportional to the length of the myocardium (McArdle, Katch, & Katch, 2015, pg 345). If the amount of blood in the ventricles increases, the resulting stretch of the ventricular walls activates receptors that trigger more forceful contractions (increasing stroke volume and, therefore, cardiac output).

One final aspect of cardiac physiology that is central to cardiovascular and pulmonary function is blood pressure. The heart is, put simply, a hydraulic pump. It is constantly generating force to push fluid through increasingly small components of bodily vasculature. Blood pressure is divided into a systolic and diastolic pressure. Systolic pressure is an estimation of the workload of the heart and is generated when the heart ejects blood during systole (McArdle, Katch, & Katch, 2015, pg. 309). Diastolic pressure, on the other hand, refers to the resistance that exists within the periphery that impedes the flow of blood (McArdle, Katch, & Katch, 2015, pg. 309). Blood pressure can be affected by a number of things including: fluid levels of the blood, sodium levels (which affects fluid levels), blood viscosity, skeletal muscle tension (in the case of diastolic pressure), force of ventricular contraction, and vessel size (with smaller vessels leading to an increase in pressure).

Understanding cardiovascular anatomy and physiology makes it easier to understand and discuss the contributions of the pulmonary system to cardiopulmonary interaction. The

pulmonary system is comprised of a series of airways, two lungs, and internal sacs that are utilized in gas exchange. These components are divided into the respiratory portion and conducting portion (Grine, 2014, pg. 286). The conducting structures of the pulmonary system are involved in moving air in and out of the lungs. These structures include the trachea, bronchi (which subdivide into primary, secondary and tertiary branches), and bronchioles (McArdle, Katch, & Katch, 2015, pg. 257). The respiratory structures are those involved in gas exchange on the molecular level inside the lungs and include the respiratory bronchioles, alveolar ducts, and alveolar sacs (McArdle, Katch, & Katch, 2015, pg. 257). The lungs themselves fill most of the thoracic cavity of the body and are covered in sacs similar to the heart (such as the visceral pleura) (Grine, 2014, pg. 286). They are lobed organs, separated by specific divisions called fissures. The left lung is made up of a superior and inferior lobe while the right has an additional middle lobe. The difference is due to the left lung containing the cardiac impression where the heart sits. Between the two of them, the lungs contain more than 600 million alveoli (McArdle, Katch, & Katch, 2015, pg. 255). Alveoli are the thin, membranous sacs that readily allow oxygen and carbon dioxide to diffuse across their cell walls during respiration.

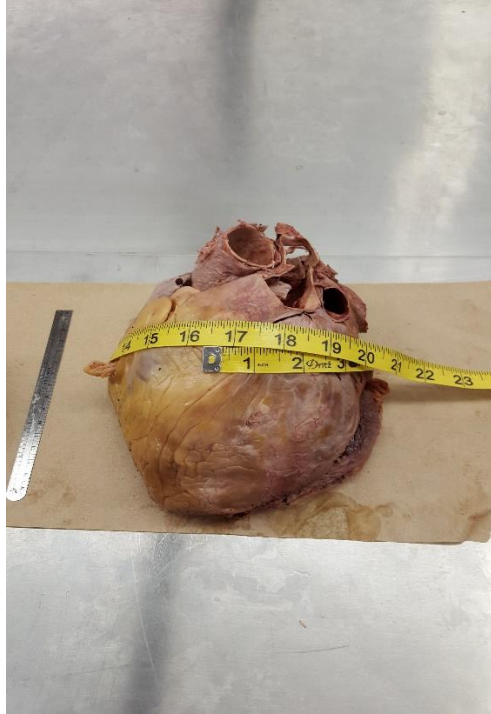
Pulmonary physiology is dependent, in many ways, on cardiovascular physiology. For example, adequate blood supply is required to move the required amount of carbon dioxide out and the required amount of oxygen into the cells. Its physiology is also heavily dependent on the properties of the gases in the air. In particular, pulmonary physiology utilizes the individual pressures exerted by carbon dioxide and oxygen when they are part of a mixture. This pressure, referred to as partial pressure, creates a diffusional gradient that allows oxygen to readily diffuse into the blood stream and CO<sub>2</sub> to diffuse into the lungs for expiration. Similar to the ionic gradient discussed earlier, the pressure gradient is created by gas concentrations. Specifically,

oxygen concentration is perpetually lower in the blood stream and higher in the atmosphere. Conversely, carbon dioxide is always higher in concentration in the blood stream than in the atmosphere. These conflicting concentrations increase the need to exhale CO<sub>2</sub> and inhale O<sub>2</sub>, as the pressure differential essentially force the gases in and out.

The discussion of cardiopulmonary anatomy and physiology and their interacting features is directly related to those that surround the pathologies that afflict those systems, both in tandem and separately. To explore those pathologies, focus will not be shifted to congestive heart failure. Heart failure is one of the most common forms of cardiovascular disease and its pervasive nature affects aspects of heart, lung, and overall health. The CDC estimates that 5.1 million people in the US have heart failure (CDC, 2015). Moreover, 1 in 9 deaths in the US is said to have been caused by heart failure (CDC, 2015). Heart failure has many potential causes including cardiomyopathy (damage or malfunction of the cardiac muscles), coronary artery disease, and hypertension (NIH, 2014). Congestive heart failure is a weakening of the ventricles that causes fluid to build up in various parts of the body such as the legs and around the lungs (NIH, 2014). The heart becomes incapable of pumping a sufficient amount of blood, leading to widespread organ damage.

Many changes occur in cardiovascular anatomy and physiology in response to heart failure. As the ventricles weaken, cardiac output sees a sharp decrease due to decreased stroke volume (precipitated by decreased myocardial contractility). To compensate, heart rate increases in an attempt to deliver the same amount of blood (Parmley, 1985). This increased cardiac activity causes the heart to consume far more oxygen than a normal heart. In the case of accompanying coronary artery disease, O<sub>2</sub> deprivation may occur in the myocardium and cause muscle death. In the systemic vasculature, less blood being pumped into the system results in

hypotension (low blood pressure). The body responds by synthesizing angiotensin, a vasoconstrictor that increases peripheral resistance (with a subsequent increase in blood pressure). This characteristic of congestive heart failure: decreased systolic pressure but increased diastolic pressure (Parmley, 1985). As the heart works harder, the myocardium of the ventricles may hypertrophy, further reducing their contractility and efficiency. Complicating this matter is the buildup of fluid around the heart (a central feature of congestive heart failure), which increases the pressure the heart has to generate to overcome vascular impedance. This also creates another problem: the right side of the heart begins pumping blood into the pulmonary arteries with much more force than is naturally intended. Increased pressure in the pulmonary vasculature likely damages many of the capillaries that surround the alveoli, as they were not meant to bear the same level of pressure generated by the left side. Death of those capillaries can increase physiologic dead space in the lungs (regions that cannot partake in gas exchange due to death or otherwise lack of alveoli), leading to dyspnea and other breathing difficulties. The vicious cycle, then, is as follows: a decrease in cardiac output leads to increased vascular resistance because of decreased blood pressure; the heart responds by pumping faster, increasing workload and hypertrophying; the hypertrophy of the right ventricle causes damage to the lungs, decreasing oxygen supply, making the heart work harder still to compensate again. Below are images depicting the radical changes in cardiac anatomy resulting from congestive heart failure with accompanying coronary artery disease:





The objective of these discussions was twofold: to provide a basic overview of the anatomical and physiological features of the cardiopulmonary system and to show how anatomy and physiology interact to form normally functioning systems. The second objective was primarily accomplished through discussion of the changes that occur during a specified disease and how those changes result in pathology. Yet this also plays into the greater goal of promoting an understanding and appreciation of the complex systems that keep humans alive. This is a critical part of combating the rising costs of both life and capital of cardiopulmonary disease. In terms of absolute dollars, cardiovascular disease costs the US about 215.6 billion dollars (AHA, 2015). The loss of life, however, is immeasurable. Inspiring future generations of scholars and researchers is always one of the goal of research, and it is that goal that will eventually eradicate the threat of death from cardiopulmonary disease.

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## Lab Portion: Examination of a Heart Affected by Congestive Heart Failure

### **Objective:**

To cultivate a better understanding of the cardiovascular and pulmonary systems and to promote critical thinking about the complexity of cardiopulmonary anatomy and physiology.

### **Materials:**

- 1.) 1 Scalpel with detachable blade
- 2.) 1 Pair surgical forceps
- 3.) 1 Surgical Hammer
- 4.) 1 Chisel
- 5.) 1 Pair of pliers
- 6.) 1 Strainer
- 7.) Paper towels
- 8.) Gloves

### **Procedure:**

The subject for the dissection was a male who passed away at the age of 88 years old due to a combination of coronary artery disease and congestive heart failure. The profusion used to preserve the cadaver was an aqueous solution composed of ethanol and formaldehyde. A pacemaker was noted on a reflected skin layer, and leads from the pacemaker were discovered in the heart. In removing a heart and lung, a number of steps had to be followed to complete the dissection. First, the top layers of skin and connective tissue must be cut through, exposing muscle of the chest. Muscle such as the sternocleidomastoid, pectoralis major and minor, serratus anterior, and other muscles that either originate or insert on the sternum or clavicle must be cut and reflected laterally (away from the midline). Then the rib cage can be cut free using a high

powered saw. This part of the process was done for me. Once the pleural cavity was exposed, showing both lung and the heart, the removal of the organs could begin. This part of the process was done by myself with guidance from Dr. Olson. First, the clavicle were removed with a hammer, chisel, and pair of pliers. A hand was placed inside the pleural cavity, pushing the right lung laterally against the remnants of the body wall. A scalpel was used to sever the connective tissue hold the lung place as well as the pulmonary artery (there is one) and veins (there are two) that branched into the lung from the heart. The lung (in this case, the right lung) was then extracted. Attention was then turned to the heart. The heart is held in place by eight vessels as well as pericardial tissue. While most of the pericardium had already been removed, a scalpel was used to cut through the remaining fibrous pericardium that rooted the heart to the diaphragm. Dr. Olson then guided me through severing the aorta, superior vena cava, inferior vena cava, and pulmonary vessels that held the heart in the cardiac impression by the left lung. The heart was then extracted. Once removed, the heart was cut open with incisions made the apex the extended laterally on the both sides. This cut through the ventricles, interventricular septum, and atria, effectively opening the heart. Once opened, the heart was cleaned using water and a strainer (to catch coagulated blood that was still inside the heart). Internal structures of the heart were labeled include the valves (tricuspid and bicuspid), chordae tendineae, trabeculae carneae, and papillary muscles. Later examination of the heart included taking its weight and other measurements and locating the coronary arteries including: anterior and posterior interventricular arteries, the circumflex artery, and the marginal artery.

**Data:**

Though this was more of a practical survey in anatomy, some data was collected in regards to the dimensions of the heart. The results were as follows:

Left Myocardium Thickness (Anterior): 1 cm

Right Myocardium Thickness (Anterior): 1.5 cm

Left Ventricle Width: 7 cm

Left Ventricle Depth: 3.5 cm

Left Ventricle Length 7.4 cm

Right Ventricle Width: 5 cm

Right Ventricle Length: 6 cm

Right Ventricle Depth 2.2 cm

Heart Weight: 796.62 grams (1.75 pounds)

**Discussion:**

The first notable observation that could be made about the heart inside the congestive heart failure cadaver was its size: the heart was absolutely huge. When compared to a normal sized heart, the difference was even more striking. The heart I extract was over twice as large as the normal heart. Before the dissection took place, it took me a moment to realize I was even looking at a heart. It was so large that it nearly crushed the cadaver's left lung completely. At 1.75 pounds (28.1 oz), the heart found inside the cadaver was much larger than the average male heart (which is typically about 10 oz). While some expansion of the heart is expected in individuals who suffer from congestive heart failure, it was still very surprising to heart a heart that close to the same size as the person's liver. Aside from the heart, some abnormalities were also noted in the removed right lung. A large, blackened mass was found in inferior lobe, indicating some pulmonary issues accompanying the cardiovascular diseases. This, in all

likelihood, contributed to the thickness of the myocardium in the anterior wall of the right ventricle (which was actually thicker than the left ventricle's anterior myocardial wall). The right side of the heart does not need to pump very hard (relative to the left ventricle) to move blood into the lungs for oxygenation under normal circumstances, but a combination of the mass in the lung and the enlarging of the heart would have caused the heart to pump much harder to compensate for the lost efficiency. This would have greatly contributed to the hypertrophy of the right ventricular myocardium seen in the cadaver. It was also pointed out that the arterial tissue of his aorta had lost much the elasticity that is characteristic of arteries. It likely would not have been very effective at carrying blood. This was a strange contrast from the coronary arteries that were hard as stone. A probe was used to shed away fatty tissue and examine the coronary arteries on both surfaces of the heart. It is unclear in those arteries had the texture they did because of leads from the pacemaker or because of coronary artery disease.