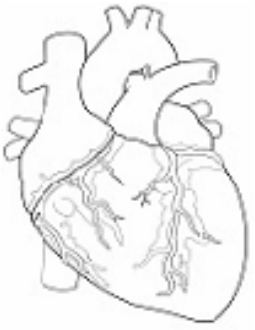

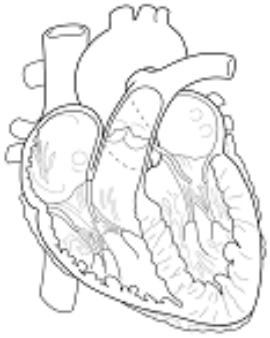

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Case Study

Mark's Failing Heart: A Look at Three Blood Volume Regulating Hormone Systems

by
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The heart is an integral organ of the cardiovascular system. It needs to remain healthy and strong to compensate for change in blood volume and pressure during daily activities. The heart must maintain enough pressure throughout the cardiovascular system to meet the demands of the body (McKinley, O'Loughlin, & Biddle, 2018). The average blood pressure within the cardiovascular system is referred to as the mean arterial pressure (MAP). Blood volume along with cardiac output and peripheral resistance determines MAP. Maintenance of adequate Mean Arterial Pressure is essential to prevent organ shutdown. The heart pumps 50 to 70 ml of blood under high pressure to the aorta per contraction and 4 to 6 liters over a minute. This is known as cardiac output. Total peripheral resistance, the result of constriction or dilation of arteries, principally arterioles, either increases or restricts blood flow. The relationship between MAP and resistance is direct; if resistance increases, MAP will increase. Maintaining adequate MAP within a normal range through adjustments in cardiac output or peripheral resistance is vital for perfusion of tissues and essential for survival. Blood volume is the other component that maintains MAP. This case study reinforces information on the heart and three blood volume/pressure regulating hormone systems, taught in Human Anatomy and Physiology: Renin-Angiotensin-Aldosterone-System (RAAS), Antidiuretic Hormone (ADH), Atrial Natriuretic Peptide (ANP) and Brain (ventricular) Natriuretic Peptide (BNP). RAAS and ADH are activated during low blood volume or pressure and stimulate multiple pathways to increase volume/pressure. ANP is released from the atria and BNP is released from the ventricles when too much blood volume causes over stretching of the heart's chambers. ANP/BNP stimulates multiple pathways to decrease volume and pressure. There are many causes and risk factors that can affect the health of the heart muscle, reducing its capacity as a pump and potentially leading to heart failure (see page listing causes and risk factors). As a result a person can develop heart failure on either side. As a result of left ventricle heart failure, the left ventricle of the heart muscle has a reduced ability to pump; this causes MAP to decease and trigger

activation of the RAAS and release of ADH. The consequence of either a blood volume or blood pressure surge as compensation could have a detrimental effect on an already weak heart in a person with left ventricle failure.

Mark (hypothetical person) is a 65 year old retired male playing baseball on a warm day with his family while attending a summer picnic. After hitting the baseball he runs to first base and feels shortness of breath, wheezing, fatigue, and has a hard time concentrating. His son takes him to the emergency room. There, the attending cardiologist obtains his history, physical exam and runs appropriate tests (see hospital chart). The results indicate that Mark has acute left ventricle heart failure and a background of chronic heart failure.

Chronic heart failure is very common. Symptoms appear slowly over time and gradually get worse. Acute heart failure develops suddenly and symptoms are initially severe. Acute heart failure may follow a heart attack. It may also be caused by a sudden lack of ability by the body to compensate for chronic heart failure https://www.heartfailurematters.org/en_GB/Understanding-heart-failure/What-are-the different-types-of-heart-failure. Heart failure can result in an alteration in ventricular structure (ventricular remodeling). The ventricle tends to become enlarged, its general shape becomes more globular and less elliptical, muscular wall of the ventricle often becomes thinner and volume of the chamber larger. These changes may be the result of myocyte hypertrophy, myocyte apoptosis, and interstitial fibrosis. This remodeling occurs due to myocardial injury and mechanical stress on the heart muscle (Konstam, Kramer, Patel, Maron, Udelson, 2011).

Activities

Students will work in groups of 3 or 4 to tackle this case study exercise. Groups will summarize discussions, describe the relevance of the case; break it down, highlighting important points; and answer the questions that are part of the case study. Students in groups will be encouraged to contribute thoughts, ask creative questions, and prod the group to action and additional ideas to lift individual students and the group to further pursuit of the subject and a better understanding of the topic. Students groups will have 1 to 1 ½ hours to answer the questions. The instructor will facilitate the process.

Questions. Based on what you have read and learned in class please answer the following questions.

1. What is Mean Arterial Pressure and why is it important?

2. Identify three factors that maintain MAP.

3. What role does the heart play in regulating MAP?

4. What role does peripheral resistance play in maintaining MAP?

5. What affect would acute heart failure have on MAP?

6. What affect does Angiotensin II of RAAS have on blood volume and pressure and how does it achieve this?

7. What is Antidiuretic hormone (ADH)? What affect does it have on blood volume and pressure, and what stimulates its secretion and what are its actions?

8. What affect would the change in MAP due to heart failure have on RAAS and ADH? How and why?

9. What affect would activation of Angiotensin II and ADH have on blood flow to the heart and pressure in the heart chambers?

10. There are many factors that can contribute to left ventricle heart failure. Based on what you have read above and on the Heart Failure Causes and Risk Factors page and after reviewing Mark's history, life style and physical examination, what risk factors do you think are contributing to Mark's heart failure and should be addressed as treatment?

11. The echocardiogram reveals a low ejection fraction and dilated left ventricle. What do the results of this test tell you about the efficiency of the heart?

12. X-ray shows an enlarged heart and edema around the lungs. What is the cause of the edema of the lungs and throughout the body?

13. Why would a change in MAP and blood distribution due to heart failure cause body fatigue?

14. BNP is elevated in Mark's blood. What affect does BNP have on blood volume and pressure and how does it achieve this?

The cardiologist suggests that Mark start taking a drug called Entresto (sacubitril/valsartan). This drug received U.S. Food and Drug Administration approval in July 2015 for use in patients with chronic heart failure (see page listing medications used to treat heart failure).

Entresto consists of two drugs:

1. Sacubitril inhibits the enzyme neprilysin, which is responsible for the degradation of BNP and ANP.
2. Valsartan is an Angiotensin II Receptor Antagonist (Fala, Loretta, 2015).

15. Sacubitril's job is to keep Brain Natriuretic Peptide (BNP) levels in the blood elevated. What beneficial affect does Sacubetril have on a person with left ventricle heart failure?

16. Valsartan's job is to inhibit the release of Angiotensin II.
    Why would you want to inhibit the production of Angiotensin II by Valsartan in a person with left ventricular heart failure?

17. What role do the kidneys play in the RAAS and BNP system?

18. Describe the homeostatic mechanism in this patient with acute heart failure.

Hospital Chart

Patient: Mark Doe **DOB:** 6/24/54 **Gender:** Male

Height: **5' 9"**

Weight: 240 lbs. Normal 128 – 168 lbs.

Blood Pressure: 145/95 Normal systolic value: < 120 mmHg
Normal diastolic value < 80 mmHg

History: Mark is a 65 year-old retired man who is over-weight, smokes, and at times drinks to excess. He seldom goes to a physician. He states he is not very active and over the last two years has felt tiredness on exertion with hard time breathing.

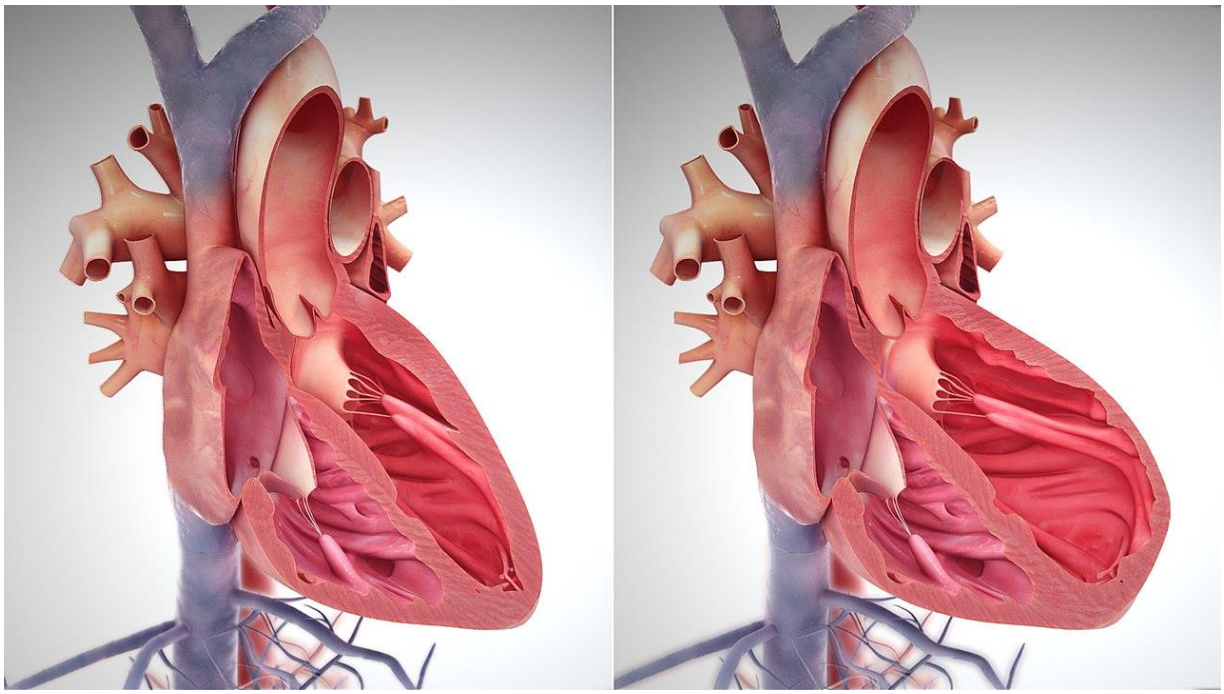
Physical Exam in Emergency Department: Mark has high blood pressure. Tachycardia (rapid heart beat). He has marked edema with abdominal and ankle swelling, distended jugular vein and has gained 5 pounds. He is fatigued and has shortness of breath. Auscultation of the lungs by stethoscope revealed crackles and rales at the base of the right and left lung (rattling, or crackling are noises made by the lungs during respiratory congestion).

BNP:	Mark Doe	Male Normal
	1200 pg/mL	<400 pg/mL

Brain natriuretic peptide (BNP) is a hormone produced in the ventricles of the heart and released when the ventricles are stretched and working hard to pump blood. Although the hormone is produced by the ventricles it is called BNP because it was first discovered in the brain. BNP is followed rather than ANP, which is produced by the atria, because it is used to detect and evaluate the degree of left ventricular failure.

Echocardiograph Measure of Ejection Fraction. It is the measurement of the percent of blood leaving the heart each time it contracts. Normal is 50 to 70%.
Mark Doe's Ejection Fraction is 44%
The echocardiograph reveals a dilated left ventricle.

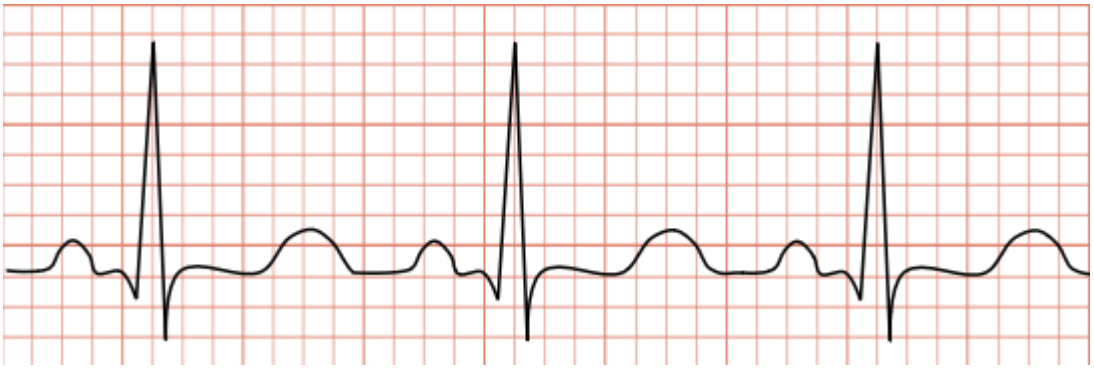
Please use the link below to view a short video on ejection fraction in patients with left ventricular failure https://www.youtube.com/watch?v=qS4jHMppRms
(American Heart Association, 2017)



A depiction of heart enlarged during heart failure by Manu5
https://commons.wikimedia.org/wiki/File:Right_side_heart_failure.jpg

A comparison of healthy heart with contracted muscle (left) and a weakened heart with over-stretched muscle and dilated ventricle (right).

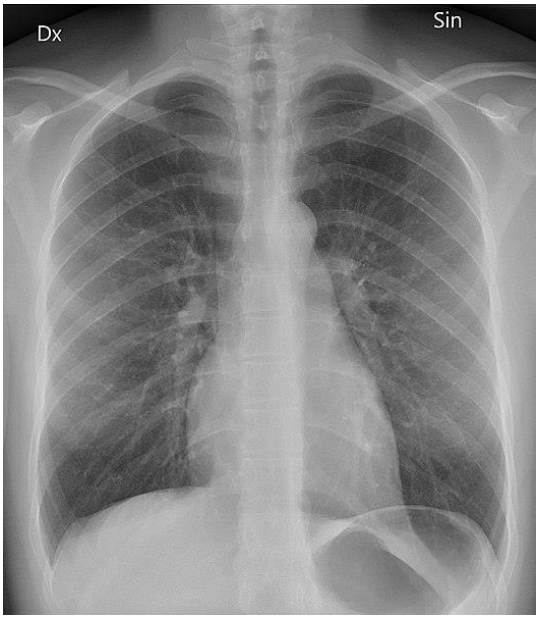
Electrocardiogram (ECG)

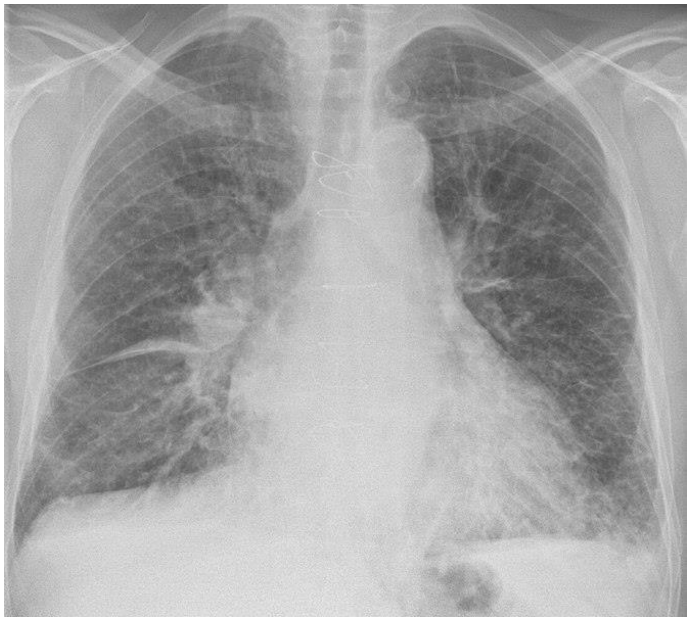


Tachycardoia ECG by Madhero88
https://commons.wikimedia.org/wiki/File:Tachycardia_ECG_paper.svg

ECG reveals tachycardia (increased heart rate)

X-ray of Lungs



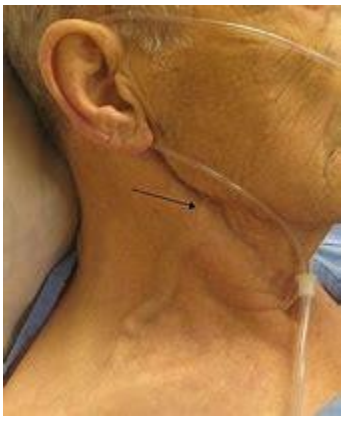


Normal posteroanterior (PA) chest radiograph (X-ray) by Mikael Häggström
https://commons.wikimedia.org/wiki/File:Normal_posteroanterior_(PA)_chest_radiograph_(X-ray).jpg

Chest radiograph of a lung with Kerley B lines by Mikael Häggström
https://commons.wikimedia.org/wiki/File:Chest_radiograph_of_a_lung_with_Kerley_B_lines.jpg

Left Chest X-Ray is Normal

Right Chest X-Ray shows Congestive Heart



Elevated JVP by James Heilman, MD.
https://commons.wikimedia.org/wiki/File:Elevated_JVP.JPG

A man with congestive heart failure and marked jugular venous distension. External jugular vein marked by an arrow.

Please look at the osmosis video concerning congestive heart failure
https://www.osmosis.org/learn/Congestive_heart_failure
(Osmosis video, 2016)

Heart Failure Causes and Risk Factors

Any of the following conditions can damage or weaken the heart and can cause heart failure.

Coronary artery disease a common cause of heart failure, is due to an occlusion of arteries by fatty build-up, and this reduces perfuse of heart muscle. **High blood pressure,** or hypertension, increases afterload causing the left ventricle to work harder to pushing blood to the aorta. This extra exertion by the left ventricle can make the heart muscle weak and ineffective. **Faulty heart valves** can also force the heart to work harder and make it weaker. **Heart muscle damage (cardiomyopathy)** can have many causes including genetics, diseases, infections, alcohol, and drugs. **Myocarditis,** an inflammation of the heart muscle, can lead to left-sided heart failure. **Congenital defects** of the heart or its valves put a strain on the healthy parts to compensate leading to heart failure. **Heart arrhythmias** such an increased heart beat may create extra work for the heart. A slow heartbeat also may lead to heart failure. **Diseases** such as diabetes, HIV, hyperthyroidism, hypothyroidism, or a buildup of iron (hemochromatosis) or protein (amyloidosis) can also contribute to heart failure. **Certain medications** such as diabetes drugs rosiglitazone (Avandia) and pioglitazone (Actos) have been found to increase the risk of heart failure in some people. Another class of medications that may increase the risk of heart problems is nonsteroidal anti-inflammatory drugs (NSAIDs). **Sleep apnea** can deliver a lower than normal amount of oxygen to the heart, leading to weakening of the heart muscles. **Obesity**, **alcohol, and tobacco use** can also increase the risk of heart failure (Mayo Clinic, 2017).

Medications Used to Treat Heart Failure

The following list of medications used to treat heart failure is provided by the American Heart Association. Patients may need multiple medications. Each medication treats a different symptom or contributing factor.

Angiotensin-Converting Enzyme (ACE) Inhibitors is a drug used for the treatment of **hypertension** and congestive heart failure. It blocks the conversion of Angiotensin I to Angiotensin II. It lowers peripheral resistance, increase venous capacity and decreases cardiac output. **Angiotensin II receptor blockers (ARBs)** are medications that block the action of angiotensin II by preventing angiotensin II from binding to receptors on the smooth muscles in the walls of blood vessels. As a result, blood vessels dilate and blood pressure is reduced. **Angiotensin-Receptor Neprilysin Inhibitors (ARNIs)** is a new drug combination of a neprilysin inhibitor and an ARB, Sacubitril/valsartan (drugs in Entresto). Sacubitrilat inhibits the enzyme neprilysin that degrades natriuretic peptides. The natriuretic peptides cause blood vessel dilation and stimulates sodium excretion which reduces extracellular fluid. **Beta blockers, also known as beta-adrenergic blocking agents** are medications that reduce blood pressure by reducing heart rate. **I_f Channel Blocker (or inhibitor)** are a drug class that reduces the heart

rate, similar to beta blockers. This is used for heart failure not fully managed by beta blockers. **Aldosterone receptor antagonists** block the effects of aldosterone which can cause heart failure to get worse. They help lower blood pressure, reduce congestion and thus protect the heart. **Hydralazine and isosorbide dinitrate** a drug treatment approved by the Food and Drug Administration (FDA) to be used to treat African Americans with congestive heart failure. It is a combination of hydralazine (an antihypertensive) and isosorbide dinitrate (a vasodilator). **Diuretics** is a medication that causes the body to rid itself of excess fluids and sodium through the kidneys. Helps to relieve the heart's workload and decreases the buildup of fluid in the lungs and other parts of the body. Other medications that might be prescribed: **Anticoagulants (blood thinners)** may be prescribed if a heart failure patient also has an increased risk of blood clot formation such as atrial fibrillation. **Cholesterol-lowering drugs (statins)** may be prescribed if a patient has high cholesterol or have had a heart attack. This class of drugs is not used for heart failure, but other conditions. **Digoxin** Some heart failure patients might be prescribed this drug if necessary. (American Heart Association, 2017).

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Case Teaching Notes

Introduction / Background

In this directed case study, students are provided information about a 65 year old man with acute left ventricle heart failure in the background of chronic heart failure. Students are provided his hospital chart with results of his history, physical exam, blood test, chest X-ray, ECG and ejection fraction. They are also provided causes and risk factors and different medications used to treat heart failure. Students are first given information about the importance of maintaining Mean Arterial Pressure (MAP) and those factors that contribute to MAP including cardiac output, total peripheral resistance and blood volume. They are given information about three volume/pressure regulating hormone systems including Renin Angiotensin, Aldosterone System (RAAS), Antidiuretic Hormone and Atrial Natriuretic Peptide (ANP) and Brain (ventricular) Natriuretic Peptide (BNP). RAAS and ADH raise blood volume and pressure when they are low. ANP and BNP lower blood volume and pressure when they are high. The case involves an individual with a weaken ventricle that causes a reduced cardiac output. Students are provided a scenario concerning the individual's acute heart failure event and hospital chart with results suggesting heart failure. The case study also involves the use of a novel drug in the treating a patient with acute heart failure Entresto (sacubitril/valsartan). Sacubitril inhibits the enzyme neprilysin, which is responsible for the degradation of BNP. Valsartan inhibits the release of Angiotensin II of the RAAS. These drugs work together to prevent the surge of blood volume and pressure in a patient with a weakened heart.

Based on the information students have gathered from the case study, hospital chart and lecture and laboratory about cardiac output, total peripheral resistance and volume and pressure regulating hormones students will be asked to answer questions.

The case study was developed for college-level pre-nursing, pre-med, biology, and physiology students. Instructors can adjust questions and classroom management to fit the case study to their course. In order to answer the case study questions, students will need to have an understanding of the
anatomy and physiology of the heart, MAP, factors that affect MAP, cardiac output and total peripheral resistance. They will need to understand the three hormone mechanisms which regulate blood volume, the Renin-Angiotensin-Aldosterone-System (RAAS), antidiuretic hormone (vasopressin) and Atrial Natriuretic Peptide and Brain (ventricular) Natriuretic Peptide. They will also need to have a basic understanding of heart failure. Background information on the above topics was obtained from Anatomy and Physiology: An Integrative Approach (McKinley, O'Loughlin, & Biddle, 2018) and the pathophysiology of heart failure (Kemp, Conte, 2012).

To answer the case study questions, students will need to apply what they know about the anatomy and physiology of the heart and blood volume/pressure regulating mechanisms to the scenario of heart failure.

Objectives:
Upon completion of this case, students should understand:

- MAP and the factors that influence MAP including blood volume, cardiac output and total peripheral resistance.
- The three hormone systems that regulate blood volume and pressure (RAAS, ADH and ANP/BNP).
- Left ventricle heart failure, its potential causes, and treatment.

CLASSROOM MANAGEMENT
This case study was developed for group work with three to four students in a group. Anatomy and Physiology at Monroe Community College consist of three hours of lecture and three hours of laboratory a week. The case study is presented to students in lecture or laboratory after the topic is covered in lecture.

Student groups are provided a hard copy and electronic copy of the case study with questions, instructions and references. The instructor, as the facilitator, presents the case study to the students.

Option I
Case study is completed in one session. Groups are asked to summarize discussions, describe the relevance of the case; break it down, highlighting important points and answer the questions that are part of the case study. Students in groups will be encouraged to contribute thoughts, ask creative questions, and prod the group to action and additional ideas so as to lift individual students and the group to further pursuit of the subject and a better understanding of the topic. At the end of the session the instructor can facilitate a class discussion of the case study and answers to questions. Time commitment is 1 to 1 ½ hours. The process would be more effective if at least one student in the group has a computer to look up internet references. You might want to ask all students to bring their computers.

Instructors can approach the case study in different ways. There is a separate hospital chart with history and test results, a page of causes and risk factors, and a page of medications used to treat heart failure. An instructor can provide the students the separate information pages with the case study or withhold the information pages initially asking the groups to surmise what tests they think should be done on the patient, causes and risk factors and medications to treat the patient.

Option II
An instructor can choose to break the case study into multiple sessions giving groups one week between sessions to complete different questions. Students within groups are encouraged to contact each other during the week by e-mail, phone, or meetings. A discussion board within an available learning management system (LMS) can be used. Students name their group, which is used to identify the LMS discussion group to which only their group can post. Instructors have access to all discussion groups and can make comments. However, any form of contact can be used. During the following week's lecture or laboratory the instructor facilitates a class discussion of the case study and answers to questions. The time commitment is 30 to 45 minutes each week for a total of 1 to 1 ½ hours.

Option III

Instructor breaks case study into multiple sessions giving groups one week between sessions to complete different questions. Instructor can choose to assign homework to each student in a group, which can be graded. Each student within a group is responsible for the development of an additional unique question related to the case study. Assignments are due the following week in lecture or laboratory. The assignment are one page long and graded on relevance of question, organization, knowledge of topic, evidence and arguments. Students within groups may contact each other during the week. When groups get together the following week they will decide who within the group will present their question to the class. The class will discuss the presentation. The time commitment is 30 to 45 minutes per week for a total of 1 to 1/12/ hours. (Cronmiller, 2015).

Examples of a student questions and brief summary answers:

- Can Entresto cause low blood pressure (hypotension)? Studnets would need to explain why it can cause low blood pressure, why this can be a problem and treatments.
- My father takes an Angiotensin-Converting Enzyme Inhibitor (ACE inhibitor) for high blood pressure.Why? Students would need to explain why an ACE inhibitor may be given to a person with high blood pressure and what affect it would have on blood pressure.
- Is the sympathetic nervous system involved as a consequence of acute heart failure? They would need to explain why a person with heart failure could have an increase in heart rate, why you would give a person with increased heart rate a Beta Blocker and explain the action of a Beta Blocker.

BLOCKS OF ANALYSIS

Mean Arterial Pressure (MAP)

MAP is an important cardiovascular condition that is regulated by multiple homeostatic mechanisms. It determines perfusion of tissues. It is the driving force of flow. The normal range of MAP is 70 to 100 mmHg. Both cardiac output (below) and total peripheral resistance (below) contribute to MAP.

Cardiac output pumps blood into the aorta raising MAP and total peripheral resistance increases or decreases flow away from the aorta which changes MAP. In addition blood volume must be adequate to maintain pressure. Volume and pressure changes need to be controlled by the three hormonal systems RAAS, ADH and ANP/BNP (below). If MAP is low there is inadequate perfusion of tissue and if it is high it can cause heart attack, heart failure, renal failure and stroke and impair cognition to name a few.

Cardiac Output

Cardiac output is the amount of blood pumped from each ventricle per minute and averages 5 liters/min at rest. It is one component that contributes to mean arterial pressure (MAP). It is a function of heart rate (beats/min) and stroke volume (amount of blood pumped by each ventricle with each beat (ml/beat) per contraction). Heart rate is (a function of the sinoatrial node and is adjusted by the autonomic nervous system and hormones (epinephrine), while stroke volume is influenced by three factors: **Preload: is an intrinsic control mechanism associated with the volume of blood filling the ventricle at the end of diastole, end-diastolic volume (EDV).** This volume stretches the myocardial sarcomere actin and myosin proteins increasing the force of

contraction. This mechanism was first described by Frank and Starling and is known as the Frank-Starling Law of the Heart and the Frank-Starling Curve is used to describe the relationship between EDV and stroke volume. **Contractility:** The strength of contraction independent of end-diastolic volume and is a function of extrinsic control, such as the sympathetic nervous system. **Afterload:** The pressure against which the heart must work to eject blood during systole. A normal healthy left ventricle is able to contract more forcefully to overcome afterload. High blood pressure increases afterload (McKinley, O'Loughlin, & Biddle, 2018).

Total Peripheral Resistance

Total peripheral resistance is the regulation of blood flow to tissues by the constriction or dilation of small arteries and arterioles. If the arteries are constricted there is an elevation of MAP and if dilated MAP goes down. The hormones mentioned in the case study all have an effect on total peripheral
resistance to help maintain blood pressure. RAAS and ADH cause vasoconstriction of arteries which increases resistance and MAP. ANP/BNP cause dilation of arteries decreasing resistance and lowering MAP.

Renin-Angiotensin-Aldosterone-System (RAAS)

This case study was developed to reinforce information on the heart and three blood volume regulating hormone systems: Renin-Angiotensin-Aldosterone System (RAAS), ADH and Atrial Natriuretic Peptide (ANP)/Brain Natriuretic Peptide (BNP) taught in Human Anatomy and Physiology. Blood volume regulation is essential to maintain adequate flow and perfusion of body tissues. If blood volume is low, the Renin-Angiotensin-Aldosterone-System (RAAS) is activated. Renin is released by the kidneys in response to low volume and pressure passing though the kidneys. Angiotensinogen is produced in the liver and released into circulation; it is converted by renin to Angiotensin I. Angiotensin I is converted to Angiotensin II by Angiotensin Converting Enzyme (ACE) released by pulmonary capillary endothelium and to a lesser extent, endothelium of capillaries throughout the body.
Angiotensin II has multiple effects on the body. It stimulates adrenal gland secretion of aldosterone, which directly increases reabsorption of Na^+ and passively increases retention of water (Na^+ captures water); secretion of Antidiuretic Hormone (ADH) by the posterior pituitary gland, resulting in reduction of water loss at the kidneys, thereby increasing blood volume; vasoconstriction of systemic arterioles, increasing Total Peripheral Resistance (TPR) and raising mean arterial pressure; thirst center in hypothalamus to increase volume intake. These effects all increase blood volume and pressure (McKinley, O'Loughlin, & Biddle, 2018).

Antidiuretic hormone (ADH)

ADH is also called Vasopressin. It is produced by neuroendocrine cells in the hypothalamus and released by their axon terminals into the posterior pituitary. It is released in response to increased osmolarity of blood. It stimulates the kidneys to retain water and causes peripheral vascular resistance through constriction of arterioles. Both actions raise blood volume and pressure. It is also released due to decreased arterial blood volume even in the face of decreased osmolality of the plasma.

Atrial Natriuretic Peptide (ANP)/Brain (Ventricle) Natriuretic Peptide

ANP & BNP Natriuretic Peptide hormones are produced when a larger than normal volume of blood enters the right atrium and ventricles, stretching their walls. Stretch receptors in the chambers sense this over filling and release the hormone ANP and BNP. They reduce blood volume by stimulating loss of water at the kidneys through the inhibition of Antidiuretic Hormone (ADH), promoting loss of Na^+ at the kidneys through inhibition of Aldosterone, and inhibition of the production of Angiotensin II, thereby preventing vasoconstriction and stimulating vasodilation.

Left Ventricle Heart Failure

Heart failure can be chronic or acute. Chronic heart failure is very common. Symptoms appear slowly over time and gradually get worse. Acute heart failure develops suddenly and symptoms are initially severe. Acute heart failure may follow a heart attack. It may also be caused by a sudden lack of ability by the body to compensate for chronic heart failure https://www.heartfailurematters.org/en_GB/Understanding-heart-failure/What-are-the different-types-of-heart-failure. Left ventricle heart failure is caused by any condition which reduces the efficiency of the heart muscle, including myocardial infarction (heart muscle is starved of oxygen and dies), or high blood pressure (after load), which increases the force of contraction needed to pump blood. The heart of a person with heart failure may have a reduced force of contraction due to overloading of the ventricle. A healthy heart compensates when there is increased filling of the ventricle by contracting more forcibly (Frank–Starling law) resulting in a higher cardiac output. In left ventricle heart failure the chamber is loaded with blood to the point where heart muscle contraction becomes less efficient due to reduced ability to cross-link actin and myosin protein filaments of the sarcomere. The ventricles cannot compensate when there is high load of blood volume. The force of contraction become less efficient with a lower stroke volume (lower blood output) (Kemp, Conte, 2012). The scenario in the case study could likely be experienced by a student in their future health care job because of the high incidence of heart failure 5.7 million in the USA, and over 23 million worldwide (Centers for Disease Control and Prevention, 2019). Risk factors for left ventricle heart failure include: high blood pressure, coronary artery disease, heart attack, diabetes, certain medications (such as NSAIDS), sleep apnea, heart defects, viruses that damage heart, alcohol use, tobacco use and obesity.

Entresto for a patient with acute heart failure

Entrestro is a drug that has received U.S. Food and Drug Administration approval for chronic Heart failure. The investigational drug consists of two medications, Sacubitril and Valsartan. Sacubitril inhibits the enzyme neprilysin, an endopeptidase responsible for the degradation of ANP and BNP. It therefore sustains the action of ANP and BNP. The reduction in blood volume lessens fluid load on the left ventricle preventing over-taxation of an already weak ventricle. Also, the reduced volume lowers Mean Arterial Pressure, reducing the amount of after-load, and decreasing the strain on the failing heart. ANP/BNP inhibits ADH and Angiotensin II. Valsartan is an Angiotensin II receptor antagonist, which inhibits the hormone's production. It keeps the blood volume at a reasonably low, acceptable level so as not to over tax the weakened heart.

One side effect of Entresto is low blood pressure (low MAP). Patients taking the drug should understand this and take appropriate action including contacting their physician (Fala, 2015).

Increased Heart Rate with Heart Failure

The patient in this case study has an increased heart rate which is common in patients with heart failure.

Beta blockers, also known as beta-adrenergic blocking agents, are medications that reduce heart rate

and force of heart contraction. They can be taken to reduce high blood pressure. Beta blockers work by blocking the receptors on the heart for epinephrine.

Heart Remodeling in a Patient with Heart Failure

Ventricular remodeling is an alteration in ventricular structure that can occur in patients with heart failure. The ventricle tends to become enlarged, its general shape becomes more globular and less elliptical, and the muscular wall of the ventricle often becomes thinner. This remodeling occurs due to myocardial injury and mechanical stress on the heart muscle. It can be the result of a combination of pathologic myocyte hypertrophy, myocyte apoptosis, and interstitial fibrosis (Konstam, Kramer, Patel, Maron, Udelson, 2011).