

Otterbein University

Digital Commons @ Otterbein

Nursing Student Class Projects (Formerly MSN)

Student Research & Creative Work

2017

Pathophysiology of Heart Failure

Tori Morrow

tori.morrow@otterbein.edu

Follow this and additional works at: https://digitalcommons.otterbein.edu/stu_msn



Part of the [Nursing Commons](#)

Recommended Citation

Morrow, Tori, "Pathophysiology of Heart Failure" (2017). *Nursing Student Class Projects (Formerly MSN)*. 246.

https://digitalcommons.otterbein.edu/stu_msn/246

This Project is brought to you for free and open access by the Student Research & Creative Work at Digital Commons @ Otterbein. It has been accepted for inclusion in Nursing Student Class Projects (Formerly MSN) by an authorized administrator of Digital Commons @ Otterbein. For more information, please contact digitalcommons07@otterbein.edu.

Pathophysiology of Heart Failure

Tori Morrow RN, BSN

Otterbein University, Westerville, Ohio

Introduction

As a nurse in the Emergency Department at Mount Carmel West, many patients come in exhibiting signs and symptoms of heart failure. Not every patient displays the same symptoms and not all patients are on the same treatment regimen. It is beneficial for nurses taking care of these patients to be knowledgeable on the disease process and what symptoms patients can experience.

Heart failure can be defined as the inability of the heart to pump effectively enough to meet metabolic demands of the body's tissues (Lodge & Yousef, 2016, p. S12). This could be caused by damage to cardiac tissue or as a result of disease process (Casey, 2013, p. 20). Many patients have multiple medical conditions that can contribute to heart failure. As a result, compensatory mechanisms may be initiated to counteract the side effects of the disease causing systemic changes.

The prevalence of this disease is very high in the United States, and it is going to continue to rise. According to Reddy & Borlaug (2016), it is expected that approximately 8 million adults in the United States will have heart failure in 2030. Prognosis is often poor and quality of life for patients is diminished, causing stress and hardship for patients and their family members. Roughly half of the people who develop heart failure die within five years of diagnosis (Briasoulis, Androulakis, Christophides, & Tousoulis, 2016).

With such a widespread disease, it is vital that healthcare providers become familiar with disease presentation, contributing factors, and treatment options. According to Reddy & Borlaug (2016), the cost of healthcare for heart failure was estimated to be \$21 billion in 2012. Consequently, it is important for an advanced practice nurse frequently encountering these patients to understand the pathophysiology behind the disease process to determine the proper choice of treatment to help further reduce cost.

Signs and Symptoms

According to Nicholson (2014), signs of heart failure include tachypnea, tachycardia, abnormal pulse, displaced apex beat, extra heart sound, raised jugular venous pressure, heart murmur, wheezing, lung crepitations, weight gain and edema, pleural effusions, hepatomegaly, and tissue wasting. Many of these signs are caused by abnormalities of the myocardial tissue as a result of the compensatory mechanisms that produce fluid retention and vasoconstriction in order to improve cardiac output and perfusion.

According to Nicholson (2014), symptoms of heart failure include breathlessness, orthopnea, paroxysmal nocturnal dyspnea, nocturnal cough, sleep disorders, fatigue, reduced exercise tolerance, peripheral edema, loss of appetite, reduced feeling, confusion, palpitations, angina, syncope, depression and anxiety.

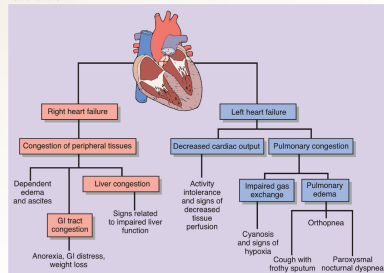
Underlying Pathophysiology

SNS

As stated previously, the disease process of heart failure can be caused by multiple factors. There are three contributors to heart contractibility, these include preload, contractility, and afterload, which all impact stroke volume (Lekavich et al., 2015). Preload is the volume of blood filling the ventricle before it contracts, where afterload is referred to as the pressure the ventricle has to work against when ejecting blood out of the ventricle generated by the pulmonary and systemic circulatory systems (Casey, 2013, p. 22). Preload is reflected by blood volume, therefore a change in levels alters preload and stroke volumes. Afterload increases with a rise in peripheral vascular resistance, causing the ventricles to work harder than normal. Contractility is referred to as the heart muscle's ability to eject the blood out of the ventricles (Casey, 2013, p. 22). Contractility is most often altered as a result of damage after a heart attack. Contractility can also be altered by changes in blood calcium level, a process that occurs with myocardial cell injury, which reduces cardiac output (Marín-García, 2016).

RAAS

When the heart is unable to effectively function as a pump the renin-angiotensin-aldosterone system (RAAS) is activated. Decreased blood flow due to poor cardiac output stimulates the kidneys to release renin, which turns angiotensinogen into angiotensin II (Lodge & Yousef, 2016, p. S13). Angiotensin II has detrimental effects to cardiac tissue. When there are consistent elevated levels of Aldosterone II, vasoconstriction occurs causing an increase in blood pressure and myocyte hypertrophy (Lodge & Yousef, 2016, p. S14). The synthesis of aldosterone is stimulated by angiotensin II (Souza-Pinto, Ferreira-Pinto, Santos, & Leite-Moreira, 2014). Aldosterone is a hormone that causes sodium and water retention, which increases blood volume, and cardiac output (Lodge & Yousef, 2016, p. S14). Increased blood volume due to aldosterone and vasoconstriction from angiotensin II, causes further myocyte damage and fibrosis occurs. Another hormone that retains water and causes vasoconstriction is arginine vasopressin (antidiuretic hormone [ADH]). ADH is released as a result of atrial stretching and a decrease in cardiac output (Lodge & Yousef, 2016, p. S14). Overstretching as a result of increased volume, therefore increased preload, decreases cardiac output. Afterload is increased due to vasoconstriction and water retention, causing the heart to increase workload resulting in an increase in metabolic demands. When the myocytes are unable to keep up with demands, hypertrophy and remodeling occurs. This perpetuates the cycle of heart failure.



When there is a decrease in blood pressure and cardiac output, the sympathetic nervous system (SNS) is activated. Stimulation of the sympathetic nervous system generates the release of norepinephrine (noradrenaline), which triggers beta-receptors to increase heart rate, strength and speed of contractions (Casey, 2013, p. 22). As a result of increased heart rate, the ventricles begin to thicken (hypertrophy) and collagen deposition occurs, causing dysfunction, this is called remodeling (Andersen, Andersen, de Man, & Nielsen-Kudsk, 2015). Ventricular hypertrophy has a significantly negative effect on the heart's contractility. Mechanical stimulation to the myocardium is the main factor of myocardial hypertrophy, whereas myocardial fibrosis may be caused by neurohumoral compensatory mechanisms such as cytokines and hormones (Komamura, 2013). Over time, this compensatory mechanism can become toxic to the heart cells, therefore rendering the cells unable to respond to the body's attempts to compensate.

Remodeling

According to Marín-García (2016), "three major cell death modalities, apoptosis, necrosis and autophagy, occur in cardiomyocytes, and both gradual and acute cell death are features of the cardiac pathophysiology, including ischemia, myocardial infarction, and progressive heart failure" (p. 1). Inflammation is important to repair damaged tissue, but over activation can cause extensive destruction. As a result of prolonged inflammation, especially in later stages of heart failure, hypertrophy, fibrosis, and cell death occur in cardiac tissue, which is known as remodeling (Briasoulis, Androulakis, Christophides, & Tousoulis, 2016). Damaged myocardium produces reactive oxygen species (ROS) which causes stress, inflammation, and cell death, resulting in the release of pro-inflammatory cytokines interleukin-1 (IL-1) and TNF-α (Souza-Pinto, Ferreira-Pinto, Santos, & Leite-Moreira, 2014). These molecules exacerbate heart failure by increasing leukocyte attraction, proliferating the inflammatory response, and increasing endothelial destruction. Endothelial destruction causes a reduced production of nitric oxide (a known vasodilator) therefore resulting in vasoconstriction (Briasoulis, Androulakis, Christophides, & Tousoulis, 2016). Vasoconstriction continues to stimulate the neurohumoral and inflammatory response, therefore proliferating the cycle of damage.

Significance of Pathophysiology

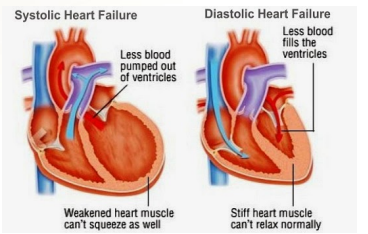
Understanding the pathophysiology allows caregivers to determine what type of heart failure the patient has and what signs and symptoms to expect. This information also provides the caregiver with knowledge of other diseases the patient is at risk for and what illnesses may have contributed to the patient's heart failure. It is vital for the provider to understand all factors and manifestations the patient has in order to properly identify the patient's ability for treatment and recovery. There are several different types of heart failure. These include left ventricular failure (systolic and diastolic heart failure) and right ventricular failure.

Risk Factors

- Common causes of heart failure are ischemic heart disease, myocardial infarction, hypertension, and arrhythmias (Nicholson, 2014). Other causes include cardiomyopathies, myocarditis, valve disorders, amyloidosis, sarcoidosis, and excessive alcohol use (Nicholson, 2014).
- Risk factors for diastolic heart failure are well established and include hypertension, older age (especially in women), kidney disease, anemia, diabetes, coronary artery disease, sedentary lifestyle, and obesity (Reddy & Borlaug, 2016). Komamura (2013) points out the significant finding of a high comorbidity rate of obesity, diabetes, and chronic kidney disease with diastolic heart failure, therefore emphasizing the importance of patient education and lifestyle modification.
- The greatest risk factors for systolic heart failure are myocardial infarction and hypertension (Reddy & Borlaug, 2016). Hypertension increases peripheral vascular resistance, therefore causing the left ventricle to work harder, dilate, and have decreased contractility.
- Right sided heart failure is commonly associated with pulmonary disease when not related to left ventricular dysfunction. Chronic obstructive pulmonary disease (COPD) causes resistance against the right ventricle when pumping blood into the pulmonary circulation, bringing about damage.

Left Heart Failure versus Right Heart Failure

Left heart failure is commonly called congestive heart failure (CHF). It can be further categorized by systolic heart failure or diastolic heart failure based off of ejection fraction. Left heart failure is essentially the incapability of the heart to perfuse the body's vital organs. Right heart failure is the inability of the heart to adequately perfuse the pulmonary circulation. Right heart failure is caused by increased resistance of the pulmonary circulation, causing the right ventricle to fail. Left heart failure is related with pulmonary edema whereas right heart failure is connected to peripheral and visceral edema (Casey, 2013, p. 21). Most often, right heart failure occurs as a result of left sided heart failure.



Systolic Heart Failure

Systolic heart failure is the inability of the left ventricle to contract effectively causing reduced blood circulation to the body (Casey, 2013, p. 21). Often, it is referred to as heart failure with reduced ejection fraction (HFrEF). Ejection fraction is simply the percentage of blood coming out of the left ventricle with each contraction. Diagnosis of HFrEF is an ejection fraction <40% with a normal being >50% (Reddy & Borlaug, 2016). The distinguishing factors in systolic heart failure is ventricular dilatation and markedly depressed contractility (Reddy & Borlaug, 2016). The chief outcomes of HFrEF are pulmonary congestion due to increase in intral pressure and poor circulation due to decreased cardiac output.

Diastolic Heart Failure

Diastolic heart failure is referred to as heart failure with preserved ejection fraction (HFpEF). Diastolic heart failure is when a normal ejection fraction occurs with impaired left ventricular relaxation. Typically, diagnosis of HFpEF is an ejection fraction of >50%, which is normal (Lekavich et al., 2015). Myocardial fibrosis or hypertrophy occur often as a result of hypertension, ending in increased left ventricular stiffness with decreased filling ability (Komamura, 2013). The left ventricle is not filling enough in diastole, so to compensate, preload is increased, which in turn increases left atrial pressure and as a result directly causes pulmonary congestion (Komamura, 2013).

Nursing Implications

- For nurses taking care of patients with heart failure, it is important to consider all factors when addressing their care. The patient and family should most importantly be educated on the disease itself in order to improve self-management. Lifestyle changes commonly recommended include smoking cessation, decreased alcohol consumption, and increase in exercise (Nicholson, 2014). As noted by Casey (2013), emphasis was placed on the importance of diet modification including low sodium and fluid intake, in addition to close monitoring of daily weights (p. 23). A note should be given to care providers to address the topic of depression since the prevalence among those diagnosed with heart failure is high.
- Appropriate control of comorbidities such as diabetes, hypercholesterolemia and kidney disease improves cardiac outcomes (Nicholson, 2014). Proper interventions should be administered based off of the patient's presenting signs and symptoms.
- The goal of treatment is to improve life expectancy and quality of life. Common pharmacological strategies include the use of angiotensin-converting enzyme (ACE) inhibitors, diuretics, and beta-adrenergic blockers (beta blockers).
- ACE inhibitors are prescribed to patients first in HFpEF due to their ability to hinder the productions of angiotensin II causing arteriole vasodilation (Lekavich et al., 2015). Therefore, they reduce preload and afterload.
- Diuretics are recommended for all types of heart failure for their capability of reducing fluid retention and overload, therefore relieving the patient's symptoms of heart failure (Reddy & Borlaug, 2016). As a result of excess fluid being excreted, preload is reduced.
- Beta blockers are typically given in addition to ACE inhibitors due to their ability to decrease the heart rate and therefore prolong diastole and improve ventricular filling (Reddy & Borlaug, 2016).

Conclusion

Heart failure is a common disease that exists always in the presence of other ailments. The detriment of the illness can be monumental for patients and families. With the improvement of health care quality, the occurrence is expected to increase. Knowing the pathophysiology allows the caregiver to appropriately determine what treatment is the best choice for that patient. The choice of treatment should always be specific to the patient based off of their current disease process and include the patient's wishes. Successful management can result in decrease hospital admissions and improved quality of life.

References

- Andersen, S., Andersen, A., de Man, F. S., & Nielsen-Kudsk, J. E. (2015). Sympathetic nervous system activation and β -adrenoceptor blockade in right heart failure. *European Journal Of Heart Failure*, 17(4), 358-366. doi:10.1002/ehf.253
- Briasoulis, A., Androulakis, E., Christophides, T., & Tousoulis, D. (2016). The role of inflammation and cell death in the pathogenesis, progression and treatment of heart failure. *Heart Failure Reviews*, 21(2), 169-176. doi:10.1007/s10741-016-9533-z
- Casey, G. (2013). Heart failure. *Kai Tiaki Nursing New Zealand*, 19(2), 20-24.
- Komamura, K. (2013). Similarities and differences between the pathogenesis and pathophysiology of diastolic and systolic heart failure. *Cardiology Research & Practice*, 1-6. doi:10.1155/2013/824135
- Lekavich, C., Barksdale, D., Neelon, V., Wu, J., Lekavich, C. L., & Barksdale, D. J. (2015). Heart failure preserved ejection fraction (HFpEF): an integrated and strategic review. *Heart Failure Reviews*, 20(6), 643-653. doi:10.1007/s10741-015-9506-7
- Lodge, F. M., & Yousef, Z. (2016). The pathophysiology of heart failure. *Primary Care Cardiovascular Journal (PCCJ)*, 512-516.
- Marín-García, J., & Marín-García, J. (2016). Cell death in the pathogenesis and progression of heart failure. *Heart Failure Reviews*, 21(2), 117-121. doi:10.1007/s10741-016-9538-7
- Nicholson, C. (2014). Chronic heart failure: pathophysiology, diagnosis and treatment. *Nursing Older People*, 26(7), 29-38. doi:10.7748/nop.26.7.29.e584
- Reddy, Y. N., & Borlaug, B. A. (2016). Heart failure with preserved ejection fraction. *Current Problems in Cardiology*, 41(4), 145-188. doi:10.1016/j.cpcardiol.2015.12.002
- Sousa-Pinto, B., Ferreira-Pinto, M. J., Santos, M., & Leite-Moreira, A. F. (2014). Central nervous system circuits modified in heart failure: pathophysiology and therapeutic implications. *Heart Failure Reviews*, 19(6), 759-779. doi:10.1007/s10741-014-9427-x



OTTERBEIN
UNIVERSITY