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The Pathophysiology of Heart Failure

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Introduction

- Heart Failure (HF) is an abnormal clinical condition involving impaired cardiac pumping which results in characteristic pathophysiologic changes related to vasoconstriction and fluid retention (Lewis, Dirksen, Heitkemper, & Butcher, 2011).
- In most nations, HF has become a major health problem with increasing incidence. Currently, about 5.1 million people in the United States are living with heart failure. This equates to an estimated $32 billion dollars each year in national healthcare costs (Alan et al., 2013).
- I have chosen HF as my topic of choice, mainly due to its prevalence in the patient population that I serve. I currently practice on an intermediate unit where most patients suffer from multiple comorbidities including some form of heart failure. What I find most fascinating about HF is the multiple ways the human body attempts to compensate for impaired cardiac pumping and the clinical symptoms associated with its disease process. HF can influence a multitude of body systems including the sympathetic nervous system, kidney, immune and pulmonary systems.

Signs & Symptoms

- Clinical presentation of HF will depend on the patient's severity of HF and particular pattern of disease.
- Common manifestations of HF include fatigue, dyspnea, exercise intolerance, and fluid retention, which may lead to pulmonary and peripheral edema symptoms such as adventitious lung sounds and weight gain (Nicholson, 2014).
- Signs and symptoms of advanced HF consist of progressive exertional dyspnea, paroxysmal nocturnal dyspnea, orthopnea, fatigability, loss of appetite, abdominal bloating, and nausea or vomiting.

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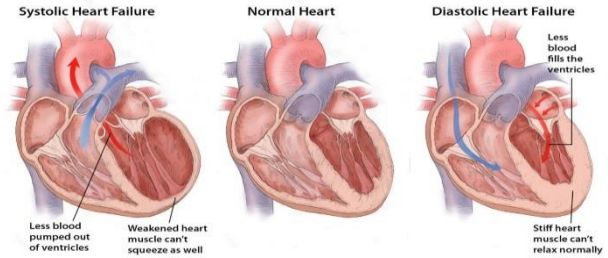
Underlying Pathophysiology

- HF is caused by any interference with the normal mechanisms of cardiac output (CO). CO is dependent on preload, afterload, and myocardial contractility. Accordingly, some origins of HF include CAD, cardiomyopathy, valvular disorders, MI, dysrhythmias, HTN, and pulmonary disorders (Piepoli & Crisafulli, 2014).
- Collectively these causes increase the workload of the ventricles, resulting in a decompensated condition that leads to decreased myocardial function and an impaired ability of the heart to maintain adequate CO.
- HF is classified as systolic or diastolic.
- **Systolic HF** is the most common cause of heart failure and results from the inability of the ventricles to contract and pump blood. Systolic failure is a result of impaired contractile function which can be caused by increased afterload due to HTN, MI, cardiomyopathies, and valvular disorders. The left ventricle (LV) loses the ability to generate enough pressure to eject blood forward through the aorta and over time the LV becomes dilated, thin-walled, and hypertrophied (Lewis et al., 2011). The hallmark of systolic dysfunction is a decrease in the left ventricular ejection fraction.
- **Diastolic Failure** is an impaired ability of the ventricles to relax and fill with blood during diastole. Decreased filling of the ventricles leads to decreased stroke volume and CO. Diastolic failure is characterized by high filling pressures due to stiff or noncompliant ventricles. This results in venous backflow in both the pulmonary and systemic vascular systems (Komamura, 2013). The diagnosis of diastolic failure is made on the basis of the presence of pulmonary congestion, pulmonary hypertension, ventricular hypertrophy, and a normal EF (Lewis et al., 2011).



Significance of Pathophysiology

- As the heart begins to fail the body's metabolic demands, several compensatory mechanisms are activated to maintain adequate tissue perfusion and cardiac output. The main compensatory mechanisms include the sympathetic nervous system (SNS) activation, neurohormonal responses, and cardiac remodeling.

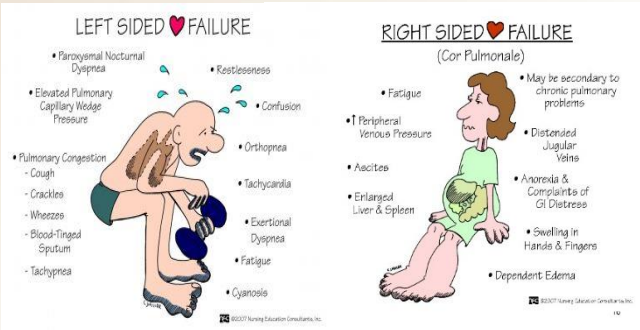
Sympathetic Nervous System (SNS) Activation

- Baroreceptors respond to a diminished CO and low BP by stimulating the SNS. Activation of SNS promotes the maintenance of cardiovascular homeostasis by inducing positive chronotropic and inotropic effects in the heart (increasing HR and myocardial contractility), as well as increased peripheral arterial vasoconstriction (Souda-Pinto, Ferreira-Pinto, Santos, Leite-Moreira, 2014).
- Initially these compensatory changes improves CO, however over time these factors act in detrimental fashion by increasing the heart's need for oxygen and the workload of the already failing heart (Lewis et al., 2011). Arterial vasoconstriction increases CO by causing an increase in preload; however this leads to a higher afterload pressure and increase of venous return thus creating additional ventricular strain on the already volume overloaded heart.
- Overactivation of the SNS is an important pathophysiological factor in HF. Although it may first be an adaptive response, chronic adrenergic overactivation is considered detrimental in the long run because it promotes cardiac remodeling and subsequent dysfunction (Andersen, Andersen, de Man, & Nielsen-Kudsk, 2015).

Significance of Pathophysiology (cont.)

Neurohormonal Responses: The Renin-Angiotensin-Aldosterone System (RAAS)

- When the kidney senses a decrease in perfusion, the renin–angiotensin-aldosterone system (RAAS) is called into action. Angiotensin II is the primary effector hormone secreted in response to physiological stimuli and is formed from its precursor, angiotensinogen, by two enzymatic cleavages (Shearer & Struthers, 2013). Angiotensinogen, released by the liver, is converted to angiotensin I by the action of renin. Angiotensin I is then cleaved by angiotensin converting enzyme (ACE) from the lungs to angiotensin II. Circulating angiotensin II promotes aldosterone secretion from the adrenal cortex which increases water and sodium reabsorption. This leads to a boost in intravascular fluid volume, and enhanced cardiac preload. It also causes systemic vasoconstriction, increasing vascular resistance and cardiac afterload (Shearer & Struthers, 2013).
- Activation of the SNS and the neurohormonal response lead to elevated levels of norepinephrine, angiotensin II, aldosterone, endothelin, and proinflammatory cytokines. Together these factors result in an increase in cardiac workload, myocardial dysfunction, and eventual ventricular remodeling (Lewis et al., 2011).



Cardiac Remodeling

- Another compensatory mechanism of the heart (in efforts to maintain adequate CO) is ventricular remodeling. Left ventricle (LV) remodeling involves changes in cavity size, wall thickness, mass, and shape; all of which reflect adaptations to myocardial injury and alterations in LV load (Ky et al., 2012).
- **Dilation** is an enlargement of the chambers of the heart and occurs when pressure in the heart chambers is elevated overtime. The muscle fibers of the heart stretch to accommodate blood volumes in the heart at the end of diastole. At first this added stretch leads to increase in contraction force (Frank-Starling Law), but eventually this mechanism becomes inadequate because the elastic elements of the muscle fibers are overstretched and can no longer contract effectively, thereby decreasing CO (Lewis et al., 2011).
- During chronic HF, the cardiac muscle adapts to volume overload and strain by increasing cardiac muscle mass and cardiac wall thickness. This is called **hypertrophy**. Hypertrophy further increases the contractile power of the muscle fibers causing a short-term increase in tissue perfusion and CO. However, the hypertrophic heart muscle has poor contractility, and requires more oxygen to perform work (Piepoli, & Crisafulli, 2014).

Types of Heart Failure: Left-sided Failure vs. Right-sided Failure

- HF usually manifests as biventricular failure, however one side may fail while the other side continues to function normally for a period of time.
- **Left-sided heart failure** is most common and results from left ventricular dysfunction which prevents normal blood flow through the aorta and to the body. This causes blood to back up into the left atrium and into the pulmonary veins. The increased pulmonary pressure causes fluid extravasation from capillary beds into the interstitium and then the alveoli, which presents clinically as pulmonary congestion and edema (Lewis et al., 2011).
- The primary cause of **right-sided heart failure** is left-sided heart failure. As previously mentioned, left-sided heart failure results in pulmonary congestion and pulmonary hypertension. Eventually this increase in pulmonary pressure causes right-sided hypertrophy and failure. Right-sided failure causes a backup of blood into the right atrium and venous circulation. The major syndrome of RV failure is volume overload and fluid retention in the systemic circulation, including peripheral edema, jugular venous distention, and ascites (Guglin & Verma, 2012).

Implications for nursing care

- The main goals in treatment of HF is to treat the underlying cause, maximize CO, improve ventricular function, provide treatment to alleviate symptoms, improve quality of life, and improve mortality and morbidity.
- However, these goals can only be achieved if both the patient and caregiver and both understanding of the patient's plan of care. Patient's and caregivers require education about heart failure to develop the staff-patient relationship and improve treatment concordance (Nicholson, 2014). Education empowers patients, increases successful self-management, and decreases rates of hospitalization. Lifestyle modifications such as diet, exercise, and smoking cessation should be discussed.
- Patients should also be taught that good control of comorbid conditions such as diabetes, high cholesterol, and kidney disease improves cardiac outcomes (Nicholson, 2014). Education of early warning signs such as weight gain and respiratory distress should also be addressed.
- Medications are the mainstay of heart failure treatment. Medications can relieve symptoms, reduce hospitalizations, shorten length of stay and improve quality of life and prognosis (Nicholson, 2014). Again, treatments such as medications only work if the clinician and patient both understand the treatment plan and then carry it out together.

Implications for nursing care (cont.)

- Medication treatment focuses to improve cardiac pump function, augment fluid removal, and enhance tissue perfusion. These goals are achieved through the use of ACE inhibitors, diuretics, and beta blocker medications.
- ACE inhibitors impede the neurohormal response of the RAAS system thus preventing systemic vasoconstriction and water retention. Angiotensin receptor blockers (ARBs) also inhibit the RAAS system and can be utilized in patients who cannot tolerate ACE inhibitors.
- Diuretics are used to decrease intravascular volume. The reduction in venous return reduces preload and allows the overfilled LV to contract more efficiently and improve CO. The increase in left ventricular function, decreases pulmonary vascular pressures, and improves gas exchange. Hydralazine and nitroglycerin are additional medications that cause systemic vasodilation and a reduction in afterload and preload, thus eliminating some ventricular strain.
- Beta-adrenergic blockers directly block the negative effects of the SNS on the failing heart such as increased HR and systemic vasoconstriction. A reduction in HR and systemic pressure allows increased cardiac perfusion and lower pressures to overcome during contraction.
- This medication list is not all-inclusive as medication therapies should be tailored to each patient. Additionally, new medication and surgical therapies are being discovered every day.

Conclusions

- Current treatment for HF involves lifestyle changes, patient education, disease-modifying and symptom-controlling medications. Unfortunately, none of these treatment methods provide a cure for HF, but only make symptoms more manageable and slow disease progression. Because of this, it is of paramount importance for both patient and provider to understand and adhere to education and treatment plans. Only by working together can therapeutic goals be achieved and quality of life sustained.

References

Alan S. G., Mozaffarian, D., Roger, V. L., Benjamin, E. J., Berry, J. D., Borden, W. B., . . . Turner, M. B. Heart disease and stroke statistics – 2013 update. *Circulation, 127* (2), 6-245.

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/ejhf.253

Guglin, M., & Verma, S. (2012). Right side of heart failure. *Heart Failure Reviews, 17*(3), 511-527 17p. doi:10.1007/s10741-011-9272-0

Komamura, K. (2013). Similarities and Differences between the Pathogenesis and Pathophysiology of Diastolic and Systolic Heart Failure. *Cardiology Research and Practice, 28*(4), 118-128. doi:10.1155/2013/824135

Ky, B., Plappert, T., Kirkpatrick, J., Silvestry, F. E., Ferrari, V. A., Keane, M. G., & ... St John Sutton, M. (2012). Left ventricular remodeling in human heart failure: quantitative echocardiographic assessment of 1,794 patients. *Echocardiography, 29*(7), 758-765. doi:10.1111/j.1540-8175.2012.01701.x

Lewis, S. L., Dirksen, S. R., Heitkemper, M. M., Butcher, L. (2011). *Medical-surgical nursing: assessment and management of clinical problems.* St. Louis, Missouri: Elsevier.

Nicholson, C. (2014). Chronic heart failure: pathophysiology, diagnosis and treatment. *Nursing Older People, 26,*(7), 29-38.

Piepoli, M. F., & Crisafulli, A. (2014). Pathophysiology of human heart failure: importance of skeletal muscle myopathy and reflexes. *Experimental Physiology, 99*(4), 609-615. doi:10.1113/expphysiol.2013.074310

Shearer, F., Lang, C. C., & Struthers, A. D. (2013). Renin-angiotensin-aldosterone system inhibitors in heart failure. *Clinical Pharmacology And Therapeutics,94*(4), 459-467. doi:10.1038/clpt.2013.135

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