Summer 2015

The Pathology of Heart Failure

Kevin Ahman
Otterbein University, kevin.ahman@otterbein.edu

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

Part of the Cardiovascular Diseases Commons, Medical Pathology Commons, and the Nursing Commons

Recommended Citation
https://digitalcommons.otterbein.edu/stu_msn/88

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 Master of Science in Nursing (MSN) Student Scholarship by an authorized administrator of Digital Commons @ Otterbein. For more information, please contact shickey@otterbein.edu.
The symptoms of heart failure can be divided into two categories: normal and typical. Typical symptoms include shortness of breath, orthopnea, paroxysmal nocturnal dyspnea, and syncope. Signs and symptoms include elevated jugular venous reflux, a third heart sound, hepatomegaly, ascites, and cachexia.

### Underlying Pathology

Heart failure is a disease that results from the heart’s inability to pump sufficient blood to meet the body’s needs (pulmonary edema, ascites, and peripheral edema). Less specific signs include ejaculatory, impulse, irregular pulse, rales, cyanosis, hepatomegaly, ascites, and cachexia. Dyspnea and edema are the most common symptoms of heart failure. Risk factors include a family history of heart disease, hypertension, diabetes, and obesity.

### Sympathetic Nervous System – The Neurohumoral Response

The sympathetic nervous system (SNS) begins the response to subendocardial oxygen deficit (O2). It begins the process by signaling for the adrenal glands to release epinephrine (E), and for neurohumors/neurons to release norepinephrine (NE). EPI and NE target

- pulmonary arteries, mesenteric vessels, and coronary arteries, and activation of the renin-angiotensin-aldosterone system (RAAS) to increase cardiac output.

### Dysfunction

1) The hyperpolarization-activated cyclic nucleotide-gated (HCN) channel generates the hyperpolarization-activated current inward current, which causes depolarization of the cells of the heart to increase the heart rate.

2) Calcium, a modulator of the sarcoplasmic reticulum associated ATP-dependent calcium pump, increases calcium release from the sarcoplasmic reticulum, increasing cardiac contractility.

3) Troponins I and myosin binding protein C (MyBP-C) decrease myocardial contractility by decreasing the number of calcium channels and increasing calcium uptake in the sarcoplasmic reticulum. The terminal phase of calcium cycling is initiated.

4) Phospholamban, a subunit of Na+/Ca2+ ATPase, inhibits inhibitory influence and results in the stimulation of the sodium pump. The end result is increased heart rate and contractility.

### Renin-Angiotensin-Aldosterone System

The RAAS plays an important role in increasing vascular volume and blood pressure. RAAS is activated by beta I, low blood pressure, or stretch. Angiotensinogen is converted to angiotensin I, which is converted to angiotensin II (Ang II) and vasopressin. Ang II causes the release of norepinephrine (NE), that undergoes proteolytic cleavage to form angiotensin I. Angiotensin II (Ang II) causes the release of renin from the kidneys, which results in increased aldosterone. Ang II also activates the renin-angiotensin-aldosterone system (RAAS). Ang II is responsible for increasing blood pressure. If the blood pressure does not increase, patients with heart failure may need to be treated in the ICU with inotropic drugs and vasopressors. If the blood pressure does not increase, the heart does not pump blood effectively, and signs and symptoms of heart failure begin.

### Remodeling

Once the above phenomenon has taken place, the remodeling process results, which is what puts a patient into heart failure. Although an exact picture of all the pathways and cells involved in remodeling is still unclear, the following scenario has been proposed at a molecular level. Myocyte stretch, local norepinephrine activity and angiotensin and endothelin release are increased. These changes stimulate expression of aldosterone and myocyte hypertrophy. The end result is further deterioration in cardiac performance and increased neurohumoral activation. In addition, increased activation of aldosterone and cytokines may also stimulate collagen synthesis, thus leading to fibrosis and remodeling of the extracellular matrix. The leading cause of death in heart failure is pulmonary edema. It is caused by the disease, but its slow progression and makes management more difficult. Patients in acute heart failure will need emergent interventions, including immediate administration of norepinephrine and diuretics as appropriate with BP. Inotropic agents are generally not recommended unless the patient is in cardiacogenic shock, because nitropress drugs increase the workload of the heart. If cardiac output is severely compromised, inotropic med may be considered. Dobutamine is the best choice for increasing cardiac output, and epinephrine and peripheral vasodilation.

### Diastolic Dysfunction

Diastolic dysfunction involves the thickened heart muscle that reduces maximum filling of the heart. Sympathetic nervous input is too great to allow increased filling of blood during the filling cycle, and makes symptoms more difficult.