CardioMEMS Heart Failure System: Keeping Patients out of the Hospital

Michelle Leyland
Otterbein University, michelle.leyland@otterbein.edu

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When heart, or myocardium, cannot pump enough blood throughout the body, for whatever reason, it is called congestive heart failure (CHF) (Shah et al., 2011). CHF is a complex condition that involves the metabolic, neuroendocrine, and immune systems (Wigle et al., 2015, Shim, & Shimizu, 1999). An acute or chronic event such as myocardial infarction, cardiomyopathy, can be categorized as either acute decompensation or chronic decompensation. Both can be caused by ischemic or dilated, hypertrophic, or restrictive cardiomyopathies (Randall, Castellone, 2009). All of which may lead to CHF. There are two main categories of HF, systolic dysfunction or diastolic dysfunction (Yancy et al., 2013). In the heart, the systole is the phase in which the blood pressure in the left ventricle increases in order to maximize blood flow per beat. The diastolic phase is the phase in which the blood pressure is lower, allowing for the heart muscle to relax and fill. It is important to note that whilst the heart muscle contracts and ejects blood from the left ventricle during systole, this also means that the blood pressure in the aorta increases, and so also in the right atrium. After systole, there is a period of diastole where the heart muscle relaxes and fills with blood. This period is known as the ventricular filling phase and is further divided into early diastole and late diastole. The point at which the heart muscle is most relaxed is termed end-diastole. When cardiac tissue is injured, monocytes become activated and migrate to the site of injury, where they differentiate into macrophages and begin to phagocytose damaged myocytes. This macrophage response in the heart is followed by a significant increase in the production of pro-inflammatory cytokines, such as tumor necrosis factor alpha (TNF-α), interleukin-1 beta (IL-1β), and interleukin-6 (IL-6) (et al., 2011). These cytokines work to recruit neutrophils to the site of injury, where they release enzymes and reactive oxygen species that cause further damage to the heart muscle. As inflammation continues, oxidative stress, which results when aerobic cells produce an excessive amount of reactive oxygen species (ROS), causes further damage to cellular and genetic material (Bergmann et al., 2009). When the heart tissue becomes chronically inflamed, it may become fibrotic, stiff, it cannot pump as forcefully. Nitric oxide is also produced which results in constriction of the myocardium (Wigle et al., 2015).

5. When blood is not adequately pumped out of the heart, blood volume backs up into other areas of the body, such as the lungs or peripheral circulation. In patients with CHF, left-sided heart failure, blood backs up into the lungs and lungs. This results in pulmonary edema. Acute decompensation causes shortness of breath and even respiratory failure. In patients with chronic pulmonary edema, the pressures in the pulmonary vessels are directly measured through remote monitoring (Bergman et al., 2004). Of note, 98.8% of patients with CHF have symptoms related to decompenstated HF. Thus far, the studies related to CHF are small, battery powered, an antenna, and a wireless electronic unit. The device is activated by a physician under fluoresce into the PA via the femoral venous route (St. Jude Medical, 2014). The CHFmonitor clinical trial studied patients with class III or IV heart failure (Nicholson, 2014). Of note, 98% of patients with CHF have symptoms related to decompenstated HF. (Bergman et al., 2014). Each patient is monitored by a clinician to perform pulmonary artery pressure. The device can access (St. Jude Medical, Inc., 2014). Of note, 98.8% of patients with CHF have symptoms related to decompenstated HF. In this study, the patient data was analyzed and the patient data is reported here. This is important because it may improve the quality of life for patients with CHF. However, CHF is a complex condition that involves the metabolism, neuroendocrine, and immune systems (Wigle et al., 2015, Shim, & Shimizu, 1999). An acute or chronic event such as myocardial infarction, cardiomyopathy, can be categorized as either acute decompensation or chronic decompensation. Both can be caused by ischemic or dilated, hypertrophic, or restrictive cardiomyopathies (Randall, Castellone, 2009). All of which may lead to CHF. There are two main categories of HF, systolic dysfunction or diastolic dysfunction (Yancy et al., 2013). In the heart, the systole is the phase in which the blood pressure in the left ventricle increases in order to maximize blood flow per beat. The diastolic phase is the phase in which the blood pressure is lower, allowing for the heart muscle to relax and fill. It is important to note that whilst the heart muscle contracts and ejects blood from the left ventricle during systole, this also means that the blood pressure in the aorta increases, and so also in the right atrium. After systole, there is a period of diastole where the heart muscle relaxes and fills with blood. This period is known as the ventricular filling phase and is further divided into early diastole and late diastole. The point at which the heart muscle is most relaxed is termed end-diastole. When cardiac tissue is injured, monocytes become activated and migrate to the site of injury, where they differentiate into macrophages and begin to phagocytose damaged myocytes. This macrophage response in the heart is followed by a significant increase in the production of pro-inflammatory cytokines, such as tumor necrosis factor alpha (TNF-α), interleukin-1 beta (IL-1β), and interleukin-6 (IL-6) (et al., 2011). These cytokines work to recruit neutrophils to the site of injury, where they release enzymes and reactive oxygen species that cause further damage to the heart muscle. As inflammation continues, oxidative stress, which results when aerobic cells produce an excessive amount of reactive oxygen species (ROS), causes further damage to cellular and genetic material (Bergmann et al., 2009). When the heart tissue becomes chronically inflamed, it may become fibrotic, stiff, it cannot pump as forcefully. Nitric oxide is also produced which results in constriction of the myocardium (Wigle et al., 2015).

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