Topic Page: congestive heart failure

Definition: **congestive heart failure** from *Merriam-Webster's Collegiate(R) Dictionary*

(1930) **:** heart failure in which the heart is unable to maintain an adequate circulation of blood in the bodily tissues or to pump out the venous blood returned to it by the veins

Image from: **Several factors, including obesity and...** in *Encyclopedia of Obesity*

Summary Article: **Congestive Heart Failure**
From *Encyclopedia of Health and Aging*

Congestive heart failure (CHF) is a clinical syndrome characterized by an inability of the heart to pump enough blood and oxygen to meet the energy needs of the body. Heart failure is a common and costly disease that has a great impact on quality of life, mortality, and functional status. Approximately 5 million people in the United States have CHF, which affects mainly older adults (6% to 10% of those age 65 years and older). Approximately 5% of all medical admissions to the hospital are attributable to CHF, and it is the most common reason for admission for older adults. Fully 30% to 40% die within the first year after diagnosis, and 60% to 70% die within 5 years. CHF accounts for approximately 2% of all U.S. health care expenditures—$28 billion in direct and indirect health care costs.

Etiology

Risk factors for developing CHF in the United States include coronary artery disease, high blood pressure (hypertension), defective heart valves, diabetes mellitus, cardiac dysrhythmias, and cardiomyopathy. Cardiomyopathy can be idiopathic or caused by drugs, infection, thyroid disease, or alcohol.

The most common cause of CHF is left ventricular (LV) systolic dysfunction, resulting from damage to the heart muscle primarily from coronary artery disease. Damage to the LV decreases its strength, measured as the ejection fraction (EF) or proportion of blood that is pumped through the heart with each contraction (systole). A reduced ejection fraction results in decreased forward blood flow with each systole and, therefore, diminished perfusion of other organs. Remodeling of the undamaged heart muscle also occurs, leading to changes in the size and function of the left ventricle that can further decrease LV systolic function. Remodeling of the heart and organ hypoperfusion lead to activation of systemic neurohumoral pathways, such as the plasma rennin–angiotensin–aldosterone system, which can cause further decompensation of cardiac function, creating a vicious cycle of multisystem failure.

As LV systolic function (and therefore EF) declines, an increase in heart rate typically occurs to increase cardiac output. However, the increased rate strains an already weakened heart by increasing myocardial oxygen demand, resulting in further decompensation of function. The rapid heart rate also decreases LV filling time and can, paradoxically, decrease cardiac output.

However, not all heart failure results from LV systolic dysfunction. In community studies, approximately half of patients with symptomatic heart failure had preserved systolic function of the left ventricle. In the Cardiovascular Health Study, heart failure with preserved LV systolic function was common (55%), especially in women and older adults. Although the pathophysiology is not well understood, poor compliance is a likely mechanism. Compliance is the ability of the heart muscle to relax between

contractions (diastole). Poor compliance reduces the size of the LV that can be filled with blood prior to the next contraction. Impaired diastolic filling results in decreased forward blood flow (although the EF remains normal) and decreased perfusion of organs similar to LV systolic dysfunction.

Symptoms

The cardinal symptoms of CHF include shortness of breath (dyspnea), cough, swelling of the feet and ankles (peripheral edema), and chronic fatigue. The impaired ability of the heart to pump blood results in blood “backing up” into the pulmonary veins, with leaking of fluid into the lung tissue (pulmonary edema). Pulmonary edema contributes to dyspnea and cough. Fluid leaks out from other veins, causing peripheral edema. Reduced perfusion of muscles, as the body attempts to direct impaired blood flow to more vital organs such as the brain and kidneys, contributes to fatigue. Other symptoms include nighttime urination (nocturia) and disordered breathing during sleep.

Multiple studies have also shown that CHF is a risk factor for disability, mortality, and decreased quality of life. A growing body of literature also suggests that changes in cognitive function may be associated with CHF.

Severity of clinical symptoms in CHF is rated according to the New York Heart Association classification system:

Class I (mild): no symptoms

Class II (mild): mild symptoms with activity but no symptoms at rest

Class III (moderate): noticeable limitation in ability to exercise due to symptoms; comfortable only at rest

Class IV (severe): unable to do any activity without decompensation; symptoms at rest

Diagnosis

The diagnosis of CHF is based on clinical symptoms. However, making the diagnosis can be challenging, especially in older adults. Many of the cardinal symptoms can occur in other conditions such as anemia, chronic lung disease, pneumonia, renal disease, and venous insufficiency. The presentation can be sudden, especially if related to myocardial ischemia, or can develop slowly over time. Frequently, CHF is characterized by chronic symptoms with intermittent exacerbations.

Examination of a patient with CHF may reveal labored respirations, hypoxia, distended neck veins due to increased jugular venous pressure, a rapid heart rate, and leg swelling. A chest X-ray is commonly used to identify an enlarged heart and pulmonary edema as well as to exclude other pulmonary disease. Echocardiography can be used to evaluate LV function and valve disease. An electrocardiogram (ECG) can identify cardiac ischemia, abnormal cardiac rhythms, or abnormal conduction that can contribute to CHF. Measurement of blood B-type natriuretic peptide, secreted by myocytes in response to excessive stretching, is elevated in decompensated heart failure and can aid in the diagnosis.

Treatment

Although there is no cure for CHF, several treatment modalities are available to improve symptoms, delay progression, and decrease hospital admissions.

Drugs are the primary treatment for CHF and include diuretics, angiotensin-converting enzyme inhibitors (ACE-I), angiotensin II receptor blockers (ARBs), β-blockers, digoxin, and blood thinners.

Diuretics, or water pills, promote salt wasting and water loss. A decrease in total body fluid results in decreased ventricular filling pressures and, therefore, decreased work of the heart. Diuretics also help rid the lungs of fluid, thereby relieving dyspnea. *ACE-I* and *ARBs* cause dilatation of the venous system, thereby decreasing blood pressure. With a lower blood pressure, the heart needs to work less hard to maintain forward blood flow and perfusion. Treatment with ACE-I and ARBs has been shown to not only decrease symptoms but also slow progression of heart failure through cardiac remodeling. β-*Blockers* decrease the heart rate, allowing longer filling time during diastole. This greater filling of the ventricles increases cardiac output and, therefore, perfusion of the body. *Digoxin* is an inotrope that can increase the force of contractions of the heart and, therefore, increase the ejection fraction. Digoxin can also be used to control the heart rate in atrial fibrillation; that is, an irregular heart rhythm that impairs ventricular filling. Reduced EF results in stasis of blood within the heart cavities. Stasis can increase stroke risk through formation of blood clots. *Blood thinners*, such as warfarin, are commonly used in more severe stages of heart failure where the risk of stasis and clots is higher.

Treatment of the underlying causes of CHF is also essential. This includes management of hypertension and coronary artery disease to decrease the risk of ischemia and strain on the heart. Replacement of the poorly functioning heart valves may be indicated. In severe heart failure, an evaluation for cardiac transplant may be appropriate. However, transplantation is limited by the number of available organs.

Lifestyle changes may also contribute to a decreased risk of developing CHF, an improvement in symptoms, a decrease in the number of hospitalizations for CHF exacerbations, and improvement in quality of life. Low-salt diets, regular exercise, weight loss, smoking cessation, and avoidance of caffeine all have been shown to have some benefit in the management of CHF.

Cardiac resynchronization therapy has proven to be beneficial in more severe CHF, especially when medical treatment fails to control symptoms. Cardiac remodeling in CHF can cause desynchrony between contractions of the ventricles of the heart due to delayed transmission of electrical impulses. This results in impaired perfusion. A device similar to a pacemaker can be implanted to send small electrical impulses to the ventricles to synchronize contraction, leading to increased pump strength, increased perfusion, and improved symptoms.

Because of the complexity of diagnosis and management of CHF, patient education and multidisciplinary support outside of the hospital setting are also necessary to help improve understanding and adherence to treatment regimens and, subsequently, to decrease symptoms, hospital admissions, and health care costs and to improve quality of life.

See also

Cardiovascular System; Diabetes; Hypertension; Valvular Heart Disease

Further Readings and References

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