



## **Congestive Heart Failure and the Elderly** **Sharon Lefebvre, RN, BSN**

### **Definition**

Congestive heart failure (CHF) is a syndrome, which refers to an inability of the heart to effectively pump blood, and therefore oxygen and nutrients, to the organs of the body. The heart does not suddenly fail or stop. Rather, CHF develops progressively over time as the left ventricle remodels in response to an initial insult to the heart, resulting in loss of contractile functioning. The size, shape, and functioning of the affected chamber is distorted. CHF develops following injury or dysfunction of the myocardium.

"Congested" refers to the buildup of blood in the vasculature resulting from the inability of the heart to pump efficiently.

### **Initial compensatory mechanisms to counter the decreasing output include:**

Sympathetic nervous system stimulation, resulting in norepinephrine increasing the heart rate and causing vasoconstriction.

Renin-angiotensin mechanism is activated, releasing antidiuretic hormone (ADH), resulting in vasoconstriction and sodium and water retention.

Ventricle dilatation to accommodate excess volume of blood, resulting in an initial increased force of contraction.

Ventricular hypertrophy results from the cardiac muscle enlargement due to overload and strain.

These compensatory mechanisms, or "responses to injury," eventually harm the heart's function by increasing myocardial oxygen demand, resulting in the clinical manifestations of left-sided heart failure.

### **Types:**

- A. Left-sided- the left ventricle of the heart does not pump effectively, due to hypertrophy or dilatation, leading to pulmonary edema.

- B. Right-sided- right side of the heart does not pump effectively, leading to peripheral edema and organ edema. Caused by left-sided heart failure or by pulmonary disease.
- C. Systolic-results from the inability of the ventricles to eject an adequate volume of blood. The ejection fraction, or amount of blood ejected from the ventricle during a contraction of the heart, is less than 40%; normal is 50-60%. Pulmonary congestion results from increased pressure due to the decreased ejection fraction.
- D. Diastolic- results from the inability of the left ventricle to fill fully during diastole. The heart has lost its ability to relax; therefore the pressure needed to fill the chamber is increased. Pulmonary congestion results from decreased cardiac output. This type is less common than systolic heart failure and is caused by long-standing hypertension.

### **Causes, or Initial insults to the heart:**

- A. Intrinsic heart pathology such as:

Coronary artery disease.  
Valvular disease.  
Chronic arrhythmias.  
Cardiomyopathies.  
Atherosclerosis.

- B. Extrinsic factors such as

Increased afterload such as in uncontrolled hypertension.  
Increased body demands- high output failure (i.e., pregnancy).  
Alcohol abuse.  
Infectious disease.

### **Normal changes related to cardiovascular system due to aging**

**Structurally**, the left ventricle thickens due to hypertrophy of individual myocytes with a gradual loss of myocyte numbers (apoptosis). Net result is a less-effective contractile force.

- Accumulation of interstitial connective tissue in the left ventricle.
- Accumulation of amyloid deposits in the left ventricle.
- Aortic and mitral valves thicken due to increased fibrosis and calcification.
- The base of the aortic cusps stiffen, resulting in incomplete opening and a partial

- Obstruction to blood flow during systole. This is called stenosis.
- Aorta and peripheral arterial system become stiff and twisted due to an increase in collagen fibers and a loss of elasticity in the middle layer of the artery. This is known as arteriosclerosis.
- Atherosclerosis due to years of buildup of fatty plaque narrows vessel lumens, resulting in increased blood pressure.

**Functionally**, aging results in a reduced ability to increase cardiac output in response to increased demand. The heart rate does not increase significantly in response to increased demand for oxygenated blood due to infection, exercise, or fever, due to loss of cells in the sinoatrial node, or pacemaker, of the heart.

**These normal changes in the heart are amplified by development of CHF.**

### **Pathological Changes in CHF:**

PaP

In left-sided heart failure, left atrial pressure increases due to ineffective pumping of the left ventricle, and blood backs up into the pulmonary circulation. The left ventricle has become hypertrophied and/or dilated due to one of the causes listed above. The most severe expression of CHF, pulmonary edema, develops when there is an increase in lung fluid due to leakage from pulmonary capillaries into the interstitium and alveoli of the lungs, caused by an increase in hydrostatic pressure within the capillaries of the lungs because the left ventricle is unable to effectively pump blood out.

Right-sided heart failure is caused by left-sided heart failure or by lung disease such as chronic obstructive pulmonary disease (COPD). Systemic edema results from backup of blood in the right ventricle.

### **Epidemiology**

The American Heart Association estimates that 4.7 million people suffer from heart failure, and that about 400,000 new cases are diagnosed each year in the United States. Heart failure is the most common hospital diagnosis for individuals over 65 years of age. Fifty percent of hospitalizations for CHF occur in persons over age 75. As the demographics of the United States change, the amount of people over age 65 will increase significantly. This fact will translate to even more cases of CHF. Prevalence of CHF is 1-2% of the general population. However, incidence increases with increasing age. Ten percent of the population over age 75 have CHF. Among those aged 40-75, CHF is more frequent in males than in females. After age 75, however, no sex-related predisposition exists.

### **Signs and Symptoms:**

Symptoms of CHF are characterized by symptoms of volume overload or inadequate tissue perfusion.

The cardinal sign of CHF is edema. Dependent edema is evident in the legs, and may be pitting. Dependent edema is due to a backup of fluid in the venous system and increased hydrostatic pressure because of the inefficiency of the right side of the heart. Here is a picture of pitting edema:

<http://pathophysiology.uams.edu/Fall02/GeneralPath/edem1/sld041.htm>

Weight gain and increased abdominal size (due to hepato-splenomegaly) may be indicators of edema. The jugular vein may be distended due to the congestion in the blood vessels.

<http://pathophysiology.uams.edu/Fall02/GeneralPath/edem1/sld010.htm>

<http://pathophysiology.uams.edu/Fall02/GeneralPath/edem1/sld011.htm>

Pulmonary edema is manifested by shortness of breath at rest and while lying.

Macroscopic and gross pictures of lungs with pulmonary edema are here:

<http://www-medlib.med.utah.edu/WebPath/LUNGHTML/LUNG003.html>

<http://pathophysiology.uams.edu/Fall02/GeneralPath/edem1/sld045.htm>

Capillary diffusion is impaired in the lungs because of the congestion, therefore enough oxygen is not getting into the pulmonary capillaries. Fatigue is a symptom of inadequate capillary gas exchange because the mitochondria of the cells need sufficient oxygen to manufacture adenosine triphosphate (ATP), or energy.

Abnormal lung sounds such as crackles and wheezing are evident while auscultating lung sounds due to fluid in the lungs.

Rust-colored (due to hemosiderin filled macrophages) sputum is produced by coughing.

Coughing is an effort by the lungs to remove the fluid.

Tachycardia, resulting in decreased filling time of the heart, and due to SNS activity and anxiety.

Presence of a third heart ("gallop") sound.

Cardiac enlargement.

Urinary retention.

<http://www-medlib.med.utah.edu/WebPath/LUNGHTML/LUNG101.html>

### **Diagnosis and Treatment:**

Diagnosis of CHF is made after reviewing the history and physical, and medical tests.

Presenting symptoms alone may be adequate to make the diagnosis. Diagnostic medical tests include:

Chest X-ray. Here is an X-ray of lungs with pulmonary edema:

<http://www.hcoa.org/hcoacme/chf-cme/chf00069.htm>

EKG to detect abnormal heart rhythms.

Echocardiogram to determine size/functioning of heart.

Lab tests- electrolytes, BUN, creatinine, BNP, CBC.

**Treatment consists of:**

Treatment of underlying cause.

Removal of contributing factors such as alcohol intake.

Oxygen by mask to increase diffusion in the lungs (acute).

Morphine to reduce anxiety, respiration rate, and heart rate (acute).

Nitroglycerin, to dilate veins and thus reduce amount of blood in pulmonary veins.

Diuretics, to decrease the stress on the heart by reducing fluid overload.

Angiotensin-converting enzyme (ACE) inhibitors, used as vasodilators of both arteries and veins.

*B* blockers, to decrease systemic blood pressure, pulmonary artery pressure, right atrial resistance, systemic vascular resistance, and heart rate.

Digoxin increases the force of each contraction.

Decreased sodium diet to prevent fluid retention.

Prog **Prognosis:**

Despite the advent of new medications with which to treat CHF, the prognosis for a patient diagnosed with CHF is poor. One-year mortality rates are 50-60% for patients diagnosed with severe heart failure, 15-30% for those with mild to moderate CHF, and approximately 10% for those with mild or asymptomatic CHF.

**Considerations affecting the ability of Elderly with CHF to comply with Prescribed Treatment**



Lack of social supports and isolation due to chronic fatigue and effects of diuretic therapy.

Depression related to illness and social isolation.

Inability to afford medications due to fixed income.

Lack of transportation to medical appointments.

Difficulty understanding and remembering medication regime and other instructions regarding treatment.

Malnutrition related to inability or lack of desire to cook, or inability to afford food; related to anorexia due to depression; related to difficulty in getting to the food store; related to purchase of convenience foods high in sodium; related to lack of knowledge regarding low sodium diet.

Co-morbid illnesses such as arthritis, resulting in decreased mobility, or urinary incontinence, made worse by diuretics.

Polypharmacy, with increased risk for drug interactions.

### **Additional Links**

[http://www.merck.com/mrkshared/mmanual\\_home/sec3/17.jsp](http://www.merck.com/mrkshared/mmanual_home/sec3/17.jsp)

<http://www.mtsinai.org/pulmonary/books/breathe/Sectm.htm>