

## **MODULE 17**

### **CONGESTIVE HEART FAILURE**

#### **Unit 1**

#### **Overview**

#### **OBJECTIVES**

Upon completion of this unit, you should be able to:

- Define congestive heart failure (CHF).
- Describe the etiology of CHF.
- Differentiate between the pathophysiology and clinical manifestations of right-sided and left-sided CHF.
- Explain the effect CHF has on the cardiovascular, respiratory, acid-base, and fluid and electrolyte systems.
- Describe the diagnostic measures of CHF.

#### **COMMENTS**

Congestive heart failure (CHF) is becoming ever more common as more patients survive cardiac damage or live long enough to develop cardiac weakness. This disorder has pervasive physiological effects that challenge your nursing skills. In its early stages, its subtle and almost imperceptible signs challenge your skills or observation and assessment. In its most severe stage, pulmonary edema, its life-threatening complications challenge your skill in providing emergency care.

#### **DEFINITION**

Congestive heart failure is a syndrome characterized by impaired pump performance (decreased cardiac output) or by frank heart failure and abnormal circulatory congestion. It is a chronic disorder in which the heart can no longer pump sufficient blood to meet metabolic demands of the body. This causes blood to back up into vital organs, resulting in congestion and edema. This syndrome can result from left ventricular failure (left heart failure), which primarily causes pulmonary congestion, or from right ventricular failure (right heart failure), which primarily causes congestion of the peripheral tissues and viscera. Because a properly functioning heart depends on both ventricles, failure of one ventricle almost always leads to failure of the other.

#### **ETIOLOGY**

Congestive heart failure is not a disease process in itself; it is a syndrome that results from another disease process. If the cardiac output (the amount of blood pumped out of the ventricle per minute) is low or normal, congestive heart failure is usually attributed to pathology in the cardiovascular system. Examples of such processes may be pericardial

myocardial, or endocardial disease; coronary artery disease; systemic hypertension; or pulmonary pathology. Congestive heart failure with a high cardiac output may be due to such conditions as Paget's disease, beriberi, hyperthyroidism, and severe anemia. See Table 1 for a list of pathophysiological processes, which can lead to CHF.

**Table 1**  
**Causes of CHF**

Cardiovascular Causes	Noncardiovascular Causes
Coronary artery disease	Severe mental or physical stress
Hypertensive heart disease	Acute blood loss
Rheumatic heart disease	Pulmonary embolism
Congenital heart disease	Severe infection
Arterioscleratic heart disease	Chronic obstructive pulmonary disease
Myocardial infarction	Drugs
Ischemic heart disease	Obesity
Valvular disease	Hyperthyroidism
Dysrhythmia	Circulatory overload
Non-compliance with treatment	
Cor pulmonale	

### **Pathophysiology**

Chronic CHF may include dysfunction in one or both ventricles. Normally, the pumping actions of the left and right sides of the heart compliment each other, producing a continuous flow of blood. (See Figure 1 for a view of normal anatomy of the heart.) However, due to pathological conditions, one side may fail while the other side continues to function normally over a period of time. Due to the prolonged strain, the functioning side of the heart will eventually fail, resulting in total heart failure. For clarity, left and right heart will be discussed as separate entities. A discussion of how heart failure affects the cardiovascular, respiratory, acid-base, and fluid and electrolyte systems follows the discussion of left and right heart failure.

### **Left-side Failure**

The most common form of initial heart failure is left-sided failure. This will usually lead to and is the main cause of right-sided failure. The majority of individuals with cardiac disease will eventually develop CHF.

Left-sided failure results from left ventricular dysfunction, which causes blood to back up through the left atrium and into the pulmonary veins. The increased pressure causes fluid extravasation from the pulmonary capillary bed, which is manifested as pulmonary congestion and edema. The most common causes are diseases of the coronary arteries, hypertension, rheumatic heart disease, and valvular dysfunction. (See Figure 2.)

**Figure 1**  
Overview of Cardiovascular System

Section showing blood flow through atria and ventricles

- |                           |                             |                           |
|---------------------------|-----------------------------|---------------------------|
| 1. Aortic arch            | 7. Right atrium             | 13. Left ventricle        |
| 2. Superior vena cava     | 8. Tricuspid valve          | 14. Mitral valve          |
| 3. Right pulmonary artery | 9. Right ventricle          | 15. Aortic valve          |
| 4. Pulmonary truck        | 10. Inferior vena cava      | 16. Left atrium           |
| 5. Right pulmonary veins  | 11. Aorta                   | 17. Left pulmonary veins  |
| 6. Pulmonary valve        | 12. Interventricular septum | 18. Left pulmonary artery |

## Right-sided Failure

Right-sided failure from a weakened right ventricle causes venous congestion in the systemic circulation and results in peripheral edema. The primary cause of right-sided failure is left-sided failure. In this situation, left-sided failure results in pulmonary congestion and increased pressure in the blood vessels of the lung (pulmonary hypertension). Eventually, pulmonary hypertension results in right-sided failure.

Cor pulmonale (right ventricular dilation and hypertrophy due to pulmonary pathophysiology) can also cause right-sided failure. Causes for cor pulmonale include chronic obstructive pulmonary disease and pulmonary emboli.

Distended neck veins can be seen when a client with right-sided failure is in a semirecumbent (at least 30° angle or greater) position. This is due to increased pressure in the right atria. (See Figure 3.)

## Simultaneous Right and Left Failure

In many elderly patients, who tend to have both arteriosclerosis and age-related degenerative changes, both ventricles may begin to falter simultaneously in their capacity to pump blood. Consequently, signs of left and right heart failure develop simultaneously, so their pattern may not be clear-cut and onset may be insidious.

## Effects on the Cardiovascular System

The cardiovascular system can be divided into two segments. The peripheral vascular system and the right ventricle constitute one segment. The pulmonary vascular system and left ventricle constitute the other. The ventricles must be perfectly synchronized and eject the same amount of blood or disequilibrium will result. If the ventricles are not able to pump equal amounts of blood, fluid accumulates in either the pulmonary or peripheral vascular system. This eventually leads to cardiac overload. The ventricles are no longer able to eject the amount of blood delivered to them. This pressure overload requires the heart to greatly increase the force of contraction to maintain cardiac output. Progressive failure of the pump may result from either a weakened cardiac muscle or severe peripheral vasoconstriction. The cardiac muscle will begin to fail and a period of compensation occurs. The muscle will make three compensatory physiological adjustments to cope with the increased workload. These compensatory mechanisms are dilation, hypertrophy, and tachycardia. They are primarily regulated by the autonomic nervous system.

The first mechanism, **dilation**, is in accordance with the length-tension principles of Starling's Law. Cardiac dilation refers to a lengthening of the cardiac muscle fibers. This occurs in conjunction with an increase in the ventricular end-diastolic pressure. Lengthening of the fibers leads to an increase in the contractile force of the muscle,

which facilitates an increase in the total stroke volume. Consequently, cardiac output is maintained.

The second mechanism is **hypertrophy**. Hypertrophy is an increase in the muscle mass and the cardiac wall thickness due to strain. It occurs slowly because it takes time for muscle tissue to develop. As myocardial mass increases, the need for additional blood and oxygen grows. This additional demand cannot always be met in the client with heart disease.

The third mechanism, **tachycardia**, occurs because of an increase in the release of catecholamines from the sympathetic nervous system. The heart is unable to maintain adequate cardiac output, thus the pressure receptors in the carotid and aortic bodies detect a lowered volume. The pressure receptors then send less impulses to the inhibitory portion of the vasomotor center. The excitatory portion of the vasomotor center then takes over with a resultant increase in sympathetic outflow. The effect of this sequence causes an increase in heart rate and contraction. Peripheral vasoconstriction also occurs.

All three of these mechanisms – dilation, hypertrophy, and tachycardia – are self-limiting.

**Pulmonary Capillary Bed**

**Figure 2**  
**Pulmonary Edema in Left-Heart Failure**

**Figure 3**  
**Systemic Edema in Right-Heart Failure**

All place a workload on the heart, which increases the oxygen consumption of the cardiac muscle. The ventricles struggle to eject large amounts of blood into constricted vessels. There is an increased contraction rate and force because of sympathetic stimulation. Cardiac decompensation occurs when output can no longer be maintained with these compensatory mechanisms.

### **Effects on the Respiratory System**

Left ventricular failure generally leads to congestion in the pulmonary system. As discussed earlier, the two ventricles must be synchronized and pump equal amounts of blood. When the left ventricle fails, the right ventricle pumps more blood into the lungs than can be removed by the left ventricle. This causes engorged pulmonary veins with resulting pulmonary edema.

Pulmonary edema can also occur in severe peripheral vasoconstriction. This vasoconstriction is present due to stimulation from the sympathetic nervous system. The increased venous tone prevents effective cardiac output. As a result, the blood backs up into the pulmonary edema. This will quickly lead to suffocation and death if immediate life-saving measures are not instituted.

The sequential accumulation of fluid in the pulmonary system causes the signs and symptoms of pulmonary edema. As the pulmonary capillaries become increasingly engorged, the fluid is forced into the interstitial spaces. This fluid will accumulate around the alveoli and small airways, which potentiates congestion of the peripheral air spaces. If the fluid in the interstices exceeds the lymphatic reabsorptive capabilities, alveolar edema will occur. (See Figure 4 for normal anatomy of the respiratory system.)

The alveolar edema will produce signs and symptoms of severe dyspnea, orthopnea, and paroxysmal nocturnal dyspnea. Inspiratory crackles (rales) which do not clear with coughing are audible. Copious amounts of frothy, bloody fluid are produced.

### **Effects on the Acid-Base Concept**

The resultant pulmonary edema from left ventricular failure often manifests hypoxemia. Due to the accumulation of fluid in the alveolar spaces, adequate oxygen ( $O_2$ ) and carbon dioxide ( $CO_2$ ) perfusion is prohibited. Respiratory acidosis occurs. The  $PCO_2$  levels may be normal or elevated and the  $PO_2$  levels are below normal due to the prolonged alveolar perfusion time.

**Figure 4**  
**Lungs**

A General arrangement of the bronchial tree	3 Lower lobe of right lung	12 Terminal bronchus
	4 Upper lobe of left lung	13 Lobular bronchiole
B Gross structure of the conducting and respiratory parts of the trachea and lungs at segmental level	5 Lower lobe of left lung	14 Terminal bronchiole
	6 Pharynx	15 Respiratory bronchiole
	7 Trachea	16 Alveolar duct
	8 Principal bronchi	17 Alveolar sac
	9 Lobar bronchi	18 Alveolus
1 Upper lobe of right lung	10 Segmental bronchi	
2 Middle lobe of right lung	11 Bronchioles	

Metabolic acidosis can also occur. This results from an inadequate oxygen supply to the tissues. To maintain normal cellular functioning the body converts to anaerobic metabolism. This causes an elevation of pyruvic acid which is reduced to lactic acid. Lactic acidosis causes depression of the central nervous system with resultant depression, stupor or coma. The respiratory rate is also increased by the acidotic effects on the central and peripheral chemoreceptors. When lactic acidosis occurs in CHF, satisfactory treatment and reversal of its damaging effects on the body are difficult.

### **Fluid and Electrolyte Concept**

Failure of either ventricle may cause multiple imbalances in the fluid and electrolyte status of the body. Right-sided heart failure generally results from persistent left-sided heart failure. An exception to this is right ventricular failure with cor pulmonale, which is a chronic enlargement of the right ventricle resulting from pulmonary hypertension. In both instances the right ventricle is unable to maintain adequate cardiac output. Peripheral vascular congestion is the end result. Angiotension II is markedly elevated causing renal and peripheral vasoconstriction. The glomerular filtration rate (GFR) may initially be maintained by this compensatory mechanism, but eventually both renal blood flow and GFR are inadequate. This limits the amount of sodium reaching the proximal tubules; consequently, more sodium is reabsorbed.

Angiotension stimulates the secretion of aldosterone from the adrenal cortex. Aldosterone acts on the distal tubules to enhance sodium and chloride reabsorption, and potassium and hydrogen excretion. Antidiuretic hormone (ADH) is also elevated. It is postulated that this enhances the already vasoconstrictive properties apparent in CHF patients. Antidiuretic hormone allows water and urea to be reabsorbed into the medullary interstitium which allows the urine to become concentrated. This hormone potentiates the already present problem of abnormal sodium and water retention. (See Figure 5 for schematic representation.)

**Figure 5**

Edema may be present in the CHF patient. This results from dilutional hyponatremia and water retention via the aforementioned mechanisms. This may occur in the systemic and/or pulmonary circulatory system.

Oliguria is a late sign of CHF resulting from depressed renal blood flow and low cardiac output. Nocturia may also occur.

## Signs and Symptoms

Persons experiencing left-sided heart failure initially show signs and symptoms of fatigue, dyspnea, orthopnea, dry hacking cough, and poor gas exchange (decreased PO<sub>2</sub>, increased PCO<sub>2</sub>). Auscultation of the lungs reveals crackles and wheezes. Auscultation of the heart reveals an S<sub>3</sub> heart sound. Later signs and symptoms of left-sided heart failure include left ventricular hypertrophy, tachycardia, and dependent edema (see Table 2).

Persons experiencing right-sided heart failure initially show signs and symptoms of dependent edema, jugular vein distention, and weight gain. Later signs and symptoms of right-sided failure include such manifestations as anasarca and hepatomegaly (see Table 2).

**Table 2**  
**Signs and Symptoms of Heart Failure**

<u>Left Heart Failure</u>	<u>Right Heart Failure</u>
Left ventricular hypertrophy (PMI displayed inferiorly and posteriorly)	Weight gain
Poor oxygen exchange (arterial blood gases: decreased PO <sub>2</sub> , increased PCO <sub>2</sub> )	Edema of dependent body parts (sacrum, tibia, pedal edema)
Pulmonary edema	Anasarca (massive generalized body edema)
Unproductive cough	Jugular vein distention
Dyspnea (shallow respirations up to 32 – 40/minute)	Liver engorgement (hepatomegaly)
Orthopnea, paroxysmal nocturnal dyspnea	
Cough (dry hacking caused by alveolar irritation from fluid accumulation)	
S <sub>3</sub> heart sound	

## Diagnosis

The primary goal in diagnosis is to determine the underlying cause of heart failure. Diagnostic measures to assess the degree of heart failure include chest x-ray, ECG, stress testing, and echocardiography.

**Chest x-ray.** The chest x-ray is the most important diagnostic measure for assessing and monitoring heart failure. Initial abnormalities in CHF, such as prominent, congested upper lobe pulmonary veins, can be seen on x-rays. Later changes, such as interstitial pulmonary edema and pulmonary effusion, can also be visualized. The degree of cardiac

enlargement is also readily observed. The chest x-ray may also reveal an enlarged cardiac shadow, reflecting dilatation or hypertrophy.

**ECG.** An ECG is of no value in detecting heart failure. However, it may detect cardiac problems which potentiate heart failure, such as increased right atrial filling pressure of dysrhythmias.

**Echocardiography.** Echocardiography can be used to measure the size of the cardiac chambers and to assess ventricular function. It also can reveal valve incompetence of stenosis, or areas of decreased ventricular wall motion.

The New York Heart Association has developed function guidelines for classifying individuals with CHF. The classification is based on the individual's tolerance to physical activity (see Table 3).

**Table 3**  
**New York Heart Association**  
**Functional Classifications of Persons**  
**With Congestive Heart Failure**

Class I

No limitation on physical activity. Ordinary physical activity does not result in symptoms. Good prognosis.

Class II

Slight limitation on physical activity. No symptoms at rest, but symptoms may be produced with ordinary physical activity. Good prognosis.

Class III

More severe limitations. Client is usually comfortable at rest. Symptoms are manifested with many unusual physical activities. Fair prognosis.

Class IV

Inability to carry on any physical activity without producing symptoms. Symptoms may be present at rest. Poor prognosis.

**Unit 1**  
**Self-Test**

1. Congestive heart failure:
  - a. is a (n) \_\_\_\_\_ disorder which affects the heart \_\_\_\_\_
  - b. results in the inability of the heart to pump blood \_\_\_\_\_.
  - c. causes blood to \_\_\_\_\_ up into vital \_\_\_\_\_, resulting in \_\_\_\_\_ and \_\_\_\_\_.
  - d. can result from \_\_\_\_\_ or \_\_\_\_\_ ventricular failure.
  
2. Causes of CHF are primarily \_\_\_\_\_ and \_\_\_\_\_.
  
3. The most common cause of initial heart failure is \_\_\_\_\_.
  
4. This will usually lead to and is a main cause of \_\_\_\_\_.
  
5. Left-sided failure results from left \_\_\_\_\_ dysfunction. This causes blood to back through the \_\_\_\_\_ atrium and into the \_\_\_\_\_ veins.
  
6. Left-sided heart failure is manifested as
  - a. \_\_\_\_\_ and
  - b. \_\_\_\_\_
  
7. Right-sided failure results in \_\_\_\_\_.
  
8. Three compensatory mechanisms which occur to improve cardiac output are
  - a. \_\_\_\_\_
  - b. \_\_\_\_\_
  - c. \_\_\_\_\_
  
9. Accumulation of fluid in the pulmonary system can lead to alveolar edema. List three signs/symptoms of alveolar edema.
  - a. \_\_\_\_\_
  - b. \_\_\_\_\_
  - c. \_\_\_\_\_
  
10. Because of the accumulation of fluid in the alveolar spaces, gas exchange of \_\_\_\_\_ and \_\_\_\_\_ are impaired.
  
11. Metabolic acidosis occurs because of \_\_\_\_\_.

12. CHF patients have decreased renal perfusion. List three compensatory mechanisms which occur as a result of inadequate renal perfusion.

- a. \_\_\_\_\_
- b. \_\_\_\_\_
- c. \_\_\_\_\_

13. List three signs and symptoms of left heart failure.

- a. \_\_\_\_\_
- b. \_\_\_\_\_
- c. \_\_\_\_\_

14. List three signs and symptoms of right heart failure.

- a. \_\_\_\_\_
- b. \_\_\_\_\_
- c. \_\_\_\_\_

15. The primary goal of diagnosis is to determine

\_\_\_\_\_  
\_\_\_\_\_  
\_\_\_\_\_

16. Diagnostic measures to assess the degree of heart failure include:

- a. \_\_\_\_\_
- b. \_\_\_\_\_
- c. \_\_\_\_\_

17. Of these diagnostic measures, the most important is

\_\_\_\_\_.

## Unit 2 Treatment

### OBJECTIVES

Upon completion of this unit, you should be able to:

- Describe the medical management of CHF.
- Explain the purpose of cardiac glycosides, diuretics, and vasodilators in the pharmacological management of CHF.
- Plan a sodium-restricted menu for dietary management of CHF.

### COMMENTS

The management of congestive heart failure is a team effort. Medical, pharmacological, dietary, and nursing management of the CHF person are discussed in this unit.

#### Medical Management

There are two major approaches to the medical management of congestive heart failure. The first is to reduce or eliminate causative factors. Examples of this would be medical treatment or cardiac dysrhythmias, hypertension, and infection. Also included in the approach is connection of any underlying entity which may be causing CHF, such as surgical treatment of structural abnormalities.

The second approach is the treatment of the heart failure itself. This approach is directed towards improving cardiac performance by use of medication. This is discussed below.

#### Pharmacological Management

There are four categories of drugs which are used to improve cardiac performances in the CHF person: inotropics, diuretics, vasodilators, and ACE inhibitors.

**Inotropics.** Digitalis preparations are commonly used cardiac glycosides for treatment of CHF. The main actions of these drugs are to strengthen the contraction of the heart (inotropic effect), decrease the conduction speed within the myocardium (dromotropic effect), and slow the heart rate (chronotropic effect). All digitalis preparations differ in characteristics of dose, onset of action, peak effect, average half-life, and route of elimination. All digitalis preparations require a loading dose to achieve an adequate blood level.

There are several nursing considerations in managing the person taking cardiac glycosides. The nurse must recognize that the margin between the therapeutic dose and the toxic dose is narrow. The nurse must observe the person for signs of toxicity. One of the earliest signs of digitalis toxicity is anorexia. Other digitalis toxic signs are fatigue, loss of visual acuity, color vision – usually green or yellow with colored halos – and

nausea and headache. Since hypokalemia greatly increases the incidence of digitalis toxicity, be alert for early signs of potassium deficiency (depressed reflexes, anorexia, muscle weakness). The pulse should be monitored since cardiac glycosides slow the heart. The nurse is responsible for assessing the person's apical pulse before administering the cardiac glycoside. As a general rule, if the pulse is below 60 beats per minute, hold the medication and contact the physician for further orders.

**Diuretics.** The aim of diuretic therapy is to reduce the pulmonary venous pressure and to promote the excretion of edema fluid. There are a variety of diuretics available, all of which have special implications. For clarity purposes, the diuretics are grouped into categories.

**Thiazide diuretics.** Chlorothiazide (Diuril), Hydrochlorothiazide (Hydro Diuril). These are widely used diuretics because they can be administered easily and are relatively inexpensive. The thiazide diuretics promote excretion of sodium, chloride, and water by blocking their reabsorption in the renal tubules. Adverse side effects of thiazide diuretics include hypokalemia and hyperglycemia.

**Loop diuretics.** Bumetanide (Bumex), Furosemide (Lasix), ethacrynic acid (Edecrin). These are very potent diuretics and are often used when pulmonary congestion must be reduced rapidly. Loop diuretics promote excretion of sodium and water by blocking their reabsorption in the Loop of Henle). Over-vigorous therapy of loop diuretics could lead to volume depletion which could lead to a decrease in cardiac output. Other adverse effects of loop diuretics include hypokalemia, hyponatremia, and hyperglycemia.

**Potassium-sparing diuretics.** Spironolactone (Aldactone), Triamterene (Dyrenium). The mechanism by which Triamterene works to reduce edema is unknown. Spironolactone produces its diuretic effect by inhibiting the action of aldosterone in the distal tubule which leads to sodium excretion and potassium retention. An adverse effect of both Aldactone and Triamterene is hyperkalemia.

When administering diuretics, the nurse must be alert to several considerations. Be aware that diuretics can produce dehydration and electrolyte depletion which can cause circulatory collapse. For this reason, monitor intake, output, and weight as well as serum electrolyte levels. Caution patients to change positions slowly as orthostatic hypotension may be present. It is generally appropriate to administer diuretics in the morning to avoid nocturia.

**Vasodilators.** Hydralazine, Nitrates, Nitroprusside, Prazosin. Vasodilator drugs aid in treatment of CHF by relaxing the pulmonary arterial and venous vessels, dilating vessels causing pooling of blood and reducing the resistance in the systemic arterial vessels. With pooling of blood in the peripheral veins, there is less blood available to the right heart for delivery to the pulmonary circulation. Relaxation of the pulmonary vessels decreases the pressure in the pulmonary capillaries and allows fluid to be absorbed from the interstitium of the lungs and from the alveoli. With a decrease in the resistance of the systemic arterial vessels, there is less pressure against which the left heart must pump and

leads to a decreased workload of the left ventricle. Because of the vasodilation, the person taking vasodilators, especially nitrates, may experience headaches. The nurse should assess for this and administer prescribed analgesic. Vasodilation can cause hypotension, so remind the person to change positions slowly.

**Angiotension-Converting Enzyme Inhibitors (ACE Inhibitors).** Captopril, benazepril, and enalapril aid in the treatment of CHF caused by left ventricular systolic dysfunction. ACE inhibitors interrupt the reninangiotensin – aldosterone cycle. These drugs promote vasodilation and diuresis by decreasing afterload and preload. By doing so, they decrease the workload of the heart. Vasodilation decreases resistance to left ventricular ejection of blood diminishing the workload of the heart and improving ventricular emptying. With diuresis, the secretion of aldosterone is decreased, affecting the kidney's ability to retain sodium. By stimulating the kidneys to excrete sodium and fluid, left ventricular filling pressure is decreased and pulmonary congestion is reduced. ACE inhibitors allow the kidneys to retain potassium while excreting sodium and fluid. Therefore, patients receiving potassium-sparing diuretics must be carefully monitored for hyperkalemia, an increased level of potassium in the blood.

### **Dietary Management**

For management of edema associated with CHF, a sodium-restricted diet is often ordered. The American Heart Association has divided sodium-restricted diets into four levels depending on the amount of sodium. The following is a guideline for use of the restricted diets.

Mild sodium restriction (2 to 3 grams sodium). Salt may be added lightly when cooking food but salt is not to be added at the table. Foods in which salt has been used as a preservative or for flavor are removed from the menu. These food items would include such items as pickles, ham, olives, chips, luncheon meats, and bouillon cubes.

Moderate sodium restriction (1,000 mg). To maintain this level of sodium restriction, sodium is neither added during cooking nor at the table. No salty foods may be used. There is some substituting of natural foods, such as substituting salt-free canned vegetables for regular canned vegetables. Milk and meat products can be used in moderation.

Strict sodium restriction (500 mg). This level of restriction limits the intake of milk, meat, and eggs, as well as adhering to the sodium restriction in the moderate restriction menu. Daily consumption of milk is limited to two cups, meat to 5-6 ounces, and one egg may be used. All higher sodium foods are restricted.

Severe sodium restriction (250 mg). This diet is very limiting in choices of foods. Low sodium milk is substituted for regular milk, meat is limited to 2 – 4 ounces daily, and three eggs may be used weekly. Because of the highly limited choices of this menu, it is rarely used.

## Nursing Management

Regardless of the work setting, the nurse is often the first person to assess the patient. The nurse must be alert to what the patient is saying (subjective data) as well as to findings obtained during physical examination (objective data). Table 4 includes examples of subjective data which may be obtained from the CHF patient. Table 5 includes examples of objective data which may be obtained from the CHF patient.

**Table 4**  
**Subjective Data**

“I get short of breath when I walk.”  
 “I can’t sleep at night without propping up on two pillows.”  
 “My shoes fit tight.”  
 “My belt and pants fit tight at the waist.”  
 “I don’t have an appetite.”  
 “My urine is dark.” (concentrated)  
 “I get up in the night to go to the bathroom.”  
 “I have a hacky cough.”

**Table 5**  
**Objective Data**

Increased respirations  
 Dyspnea  
 Cough: dry and hacky  
 May be blood-tinged sputum  
 Neck vein distention  
 Dusky, pale skin  
 Edema in legs, feet, and sacrum  
 Ascites may be noted  
 Decreased intensity of peripheral pulses  
 Diminished breath sounds: wheezes or crackles

After the pertinent subjective and objective data have been obtained, the RN must formulate a plan of care. Appropriate nursing diagnosis with corresponding interventions for the person experiencing CHF include those in Table 6.

**Table 6**  
**Care Plan for a**  
**Congestive Heart Failure Patient**

<b>Nursing diagnosis</b>	<b>Interventions</b>	<b>Rationale</b>
Decreased cardiac output related to alternation in mechanical and electrical factors of the heart.	<p>Bed rest, quiet, relaxed atmosphere</p> <p>Place in semi-Fowler's or Fowler's position</p> <p>Administer appropriate medications as ordered:</p> <ul style="list-style-type: none"> <li>• cardiac glycosides</li> <li>• diuretics</li> <li>• nitrates/vasodilators</li> </ul>	<p>Decrease O<sub>2</sub> demand for myocardium.</p> <p>Allows for greater lung expansion and decreased abdominal organ pressure on the diaphragm.</p> <p>Increase myocardial contractility thus improving cardiac output.</p> <p>Reduce venous return to heart.</p> <p>Promote vasodilation and peripheral vascular pooling thereby decreasing systemic and pulmonary pressure.</p>
Decreased tissue perfusion related to decreased cardiac output.	<p>Assess for confusion, dizziness, syncope.</p> <p>Assess skin color and temperature.</p> <p>Assess peripheral pulses.</p> <p>Assess bowel sounds and note abdominal distention. Provide small, frequent meals.</p>	<p>Cerebral perfusion is directly related to cardiac output.</p> <p>As venous congestion increases, the skin becomes blue or mottled.</p> <p>As cardiac output decreases, these pulses may be weak, thready or unobtainable.</p> <p>CHF can lead to visceral congestion.</p> <p>Improves nutritional intake and avoids causing abdominal discomfort.</p>

<b>Nursing diagnosis</b>	<b>Interventions</b>	<b>Rationale</b>
	<p>Apply antiembolic stockings. Elevate legs, avoid pressure under knees. Encourage range of motion exercises; ambulate if possible.</p>	<p>Also decreases workload of heart during digestion.</p> <p>Decrease venous stasis and reduce incidents of thrombophlebitis.</p>
<p>Excess fluid volume related to ineffective contractility of the heart.</p>	<p>Assess breath sounds (crackles/wheezes).</p> <p>Inspect for distended neck veins or peripheral veins. Inspect for edema in ankles and sacrum.</p> <p>Monitor abdominal girth</p> <p>Weigh daily.</p> <p>Monitor intake and output. Establish a fluid intake schedule for each nursing shift.</p> <p>Elevate feet when sitting in chair.</p> <p>Administer appropriate medications as ordered; diuretics.</p>	<p>Indicate pulmonary congestion.</p> <p>Excessive fluid volume engorges veins and causes edema.</p> <p>If increases, could indicated presence of ascites.</p> <p>Indicator of fluid retention. A gain of 5 pounds indicates approximately 2 liters of fluid has been retained.</p> <p>Provides a more controlled fluid therapy regimen.</p> <p>Increases venous return and reduces swelling in ankles/legs.</p> <p>Increases excretion of fluid.</p>

<b>Nursing diagnosis</b>	<b>Interventions</b>	<b>Rationale</b>
Impaired gas exchange related to pulmonary congestion.	<p>Monitor respirations (rate, rhythm, use of accessory muscles); monitor lung sounds (crackles, wheezes).</p> <p>Note presence of pink frothy sputum, distended neck veins, apprehension.</p> <p>Turn, cough and deep breathe. Suction if needed.</p> <p>Place in semi-Fowler's position; support arms on pillows.</p> <p>Monitor arterial blood gases.</p> <p>Monitor chest x-ray.</p> <p>Administer oxygen as needed.</p> <p>Administer appropriate medications as ordered:</p> <ul style="list-style-type: none"> <li>• diuretics</li> <li>• vasodilators</li> <li>• cardiac glycosides</li> </ul>	<p>Provides for baseline information to document progress of CHF</p> <p>All are manifestations of pulmonary edema.</p> <p>Clears airway; improves oxygenation.</p> <p>Allows for greatest lung expansion to promote better oxygenation of lungs.</p> <p>Provides data for documentation of progress of CHF.</p> <p>Indicates increase or decrease in congestion of lungs.</p> <p>Increases alveolar oxygen concentration.</p> <p>Promote excretion of fluid thereby decreasing pulmonary congestion.</p> <p>Reduce venous return thereby reducing pulmonary congestion.</p> <p>Reduce pulmonary congestion by improving myocardial contractility and improving cardiac output.</p>

Nursing diagnosis	Interventions	Rationale
Impaired skin integrity related to decreased tissue perfusion and edema.	Assess skin for color, temperature and integrity.	Alteration in particular areas may indicate damage to skin by pressure or decreased circulation.
	Massage reddened areas.	Increases blood flow to area thereby increasing oxygenation to tissue.
	Reposition frequently.	Reduces amount of time there is pressure on one area.
	Keep skin dry.	Moisture damages skin and could lead to skin breakdown.
	Provide alternate mattress, egg crate.	Reduces pressure on skin.

### Complications

Pulmonary Edema and Pleural Effusion. As pulmonary congestion increases, the distended capillaries leak fluid into the alveoli. Pleural effusion results from increasing pressure in the pleural capillaries. A movement of fluid occurs from these capillaries into the pleural space. Nursing responsibilities in each stage of pulmonary edema are listed in Table 7.

**Table 7**  
**Managing Pulmonary Edema**

<b>Initial Stage</b>	<b>Acute Stage</b>	<b>Advanced Stage</b>
<p align="center"><u>Symptoms</u></p> <ul style="list-style-type: none"> <li>• Persistent cough</li> <li>• Slight dyspnea orthopnea</li> <li>• Exercise intolerance</li> <li>• Crepitant rales at lung bases</li> </ul> <p align="center"><u>Nursing responsibilities</u></p> <ul style="list-style-type: none"> <li>• Check color and amount of expectoration</li> <li>• Position patient for comfort</li> <li>• Medicate as ordered</li> <li>• Monitor apical and radial pulses</li> <li>• Assist patient with all needs to conserve strength</li> <li>• Provide emotional support (through all stages) for patient and family</li> </ul>	<p align="center"><u>Symptoms</u></p> <ul style="list-style-type: none"> <li>• Acute shortness of breath</li> <li>• Respirations – rapid, noisy (audible wheeze, rales)</li> <li>• Cough – more intense and productive of frothy, blood-tinged sputum</li> <li>• Cyanosis – cold, clammy skin</li> <li>• Tachycardia – arrhythmias</li> <li>• Hypotension</li> </ul> <p align="center"><u>Nursing responsibilities</u></p> <ul style="list-style-type: none"> <li>• Give oxygen (preferably by high-concentration mask or IPPB)</li> <li>• Insert IV, if not already done</li> <li>• Aspirate nasopharynx, as needed</li> <li>• Apply rotating tourniquets</li> <li>• Give digitalis, morphine, and potent diuretic (e.g. furosemide), as ordered</li> <li>• Insert Foley catheter</li> <li>• Calculate intake and output accurately</li> <li>• Attach cardiac monitor leads, and observe EKG</li> <li>• Prepare for phlebotomy, if necessary</li> <li>• Keep resuscitation equipment available</li> </ul>	<p align="center"><u>Symptoms</u></p> <ul style="list-style-type: none"> <li>• Decreased level of consciousness</li> <li>• Ventricular arrhythmias; shock</li> <li>• Diminished breath sounds</li> </ul> <p align="center"><u>Nursing responsibilities</u></p> <ul style="list-style-type: none"> <li>• Be prepared for cardioversion</li> <li>• Assist with intubation mechanical ventilation, and resuscitate if necessary.</li> </ul>

## **Prevention of Heart Failure**

The best therapy for congestive heart failure, as with many disorders, lies in prevention. Dietary and lifestyle changes which control the development of hypertensive vascular disease and the resultant increase in myocardial workload would be most beneficial. It has been shown, for example, that exercise conditioning may increase the functional aerobic capacity of the myocardium as much as 10 to 15 percent in a normal person.

Of course, the value of exercise conditioning in patients with chronic or progressive heart failure is much more controversial. When not overdone, mild exercise may help strengthen the heart muscle and improve its performance. Careful monitoring is required to determine a safe exercising level for the patient.

**Staging Pulmonary Edema.** A description of the three pulmonary edema stages including pathophysiology and signs and symptoms is illustrated in Table 8.

**Table 8**  
**Stages of Pulmonary Edema**

Stage	Pathophysiology	Signs & Symptoms
Initial	Usually left ventricle failure increases pulmonary vascular bed pressure, forcing fluid solutes from the intravascular compartment into the interstitium of the lungs. As the interstitium overloads with fluid, fluid enters the peripheral alveoli, impairing adequate gas exchange.	<ul style="list-style-type: none"> <li>• Persistent cough – patient feels like he or she “has a cold coming on”</li> <li>• Slight dyspnea/orthopnea</li> <li>• Exercise intolerance</li> <li>• Restlessness</li> <li>• Anxiety</li> <li>• Crepitant rales may be heard over the dependent portion of the lungs.</li> <li>• Diastolic gallop</li> </ul>
Acute	Fluid accumulation throughout pulmonary vasculature and further filling of the alveoli.	<ul style="list-style-type: none"> <li>• Acute shortness of breath</li> <li>• Respirations – rapid, noisy (audible wheeze, rales)</li> <li>• Cough more intense producing frothy, blood-tinged sputum</li> <li>• Cyanosis</li> <li>• Diaphoresis cold and clammy skin</li> <li>• Tachycardia dysrhythmia</li> <li>• Hypotension</li> </ul>
Advanced	Patient’s condition rapidly deteriorates as the bronchial tree fills with fluid.	<ul style="list-style-type: none"> <li>• Decreased level of consciousness</li> <li>• Ventricular dysrhythmia</li> <li>• Shock</li> <li>• Diminished breath sounds</li> </ul>

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**Unit 2**  
**Self Test**

1. The three main categories of drugs used in management of CHF include
  - a. \_\_\_\_\_
  - b. \_\_\_\_\_
  - c. \_\_\_\_\_
  
2. The three main actions of cardiac glycosides are to
  - a. \_\_\_\_\_
  - b. \_\_\_\_\_
  - c. \_\_\_\_\_
  
3. \_\_\_\_\_ is the electrolyte which is most important for the nurse to monitor in persons receiving cardiac glycosides.
  
4. Thiazide diuretics exert their effect as diuretics by  
\_\_\_\_\_  
\_\_\_\_\_
  
5. Loop diuretics exert their effect as diuretic by  
\_\_\_\_\_  
\_\_\_\_\_
  
6. \_\_\_\_\_ and \_\_\_\_\_ are two signs the nurse must be alert for in persons receiving diuretic therapy.
  
7. Vasodilators are used in treating CHF because they exert their actions by  
\_\_\_\_\_  
\_\_\_\_\_
  
8. Sodium \_\_\_\_\_ diets are often ordered for management of edema.
  
9. The \_\_\_\_\_ sodium-restricted diet limits intake of milk, meat and eggs.
  
10. A mild sodium restriction constitutes intake of \_\_\_\_\_ grams of sodium per day.
  
11. List three examples of subjective data from a CHF person.
  - a. \_\_\_\_\_
  - b. \_\_\_\_\_
  - c. \_\_\_\_\_
  
12. List three examples of objective data that might be obtained when assessing a CHF

person.

- a. \_\_\_\_\_
- b. \_\_\_\_\_
- c. \_\_\_\_\_

13. List two nursing diagnosis for a CHF person with corresponding nursing interventions.

Nursing diagnosis

Nursing intervention

a.

b.

14. A major complication of CHF is

\_\_\_\_\_

\_\_\_\_\_

15. The stages of pulmonary edema are initial, acute, and advanced. Please list three symptoms for each stage.

Initial

- a. \_\_\_\_\_
- b. \_\_\_\_\_
- c. \_\_\_\_\_

Acute

- a. \_\_\_\_\_
- b. \_\_\_\_\_
- c. \_\_\_\_\_

Advanced

- a. \_\_\_\_\_
- b. \_\_\_\_\_
- c. \_\_\_\_\_

16. Changes in \_\_\_\_\_ and \_\_\_\_\_ are two ways of preventing heart failure.

**Module 17**  
**Answers to Self-Test**

**Unit 1**

1.
  - a. chronic, muscle
  - b. sufficiently
  - c. back, organs, congestion, edema
  - d. left, right
2. cardiovascular, noncardiovascular
3. left-sided failure
4. right-sided failure
5. ventricular, left, pulmonary
6.
  - a. pulmonary congestion
  - b. pulmonary edema
7. peripheral edema
8.
  - a. dilation
  - b. hypertrophy
  - c. tachycardia
9. possible answers: dyspnea, orthopnea, paroxysmal nocturnal dyspnea, inspiratory crackles, frothy blood-tinged sputum
10. oxygen, carbon dioxide
11. inadequate oxygen supply to tissue
12.
  - a. release of angiotension II
  - b. secretion of aldosterone
  - c. secretion of antidiuretic hormone
13.
  - a. left ventricular hypertrophy (PHI)
  - b. poor oxygen exchange (arterial blood gases)
  - c. pulmonary edema
  - d. unproductive cough
  - e. dyspnea (shallow respirations up to 32-40/minute)
  - f. orthopnea, paroxysmal nocturnal dyspnea
  - g. cough (dry, hacking)
  - h. S<sub>3</sub> heart sound

14.
  - a. peripheral edema
  - b. weight gain
  - c. edema of dependent body part
  - d. generalized body part edema (anasarca)
  - e. jugular vein distension
  - f. liver engorgement
15. the underlying cause of heart failure
16.
  - a. chest x-ray
  - b. ECG
  - c. Exhocardiography
17. chest x-ray

## **Unit 2**

1.
  - a. cardiac glycosides
  - b. diuretics
  - c. vasodilators
2.
  - a. strengthen contraction of heart
  - b. decrease conduction speed
  - c. slow heart rate
3. potassium
4. blocking reabsorption of sodium, chloride, and water in the renal tubules
5. blocking reabsorption of sodium and water in the Loop of Henle
6. dehydration, electrolyte imbalance
7. delating vessels and pooling blood
8. restricted
9. strict
10. 2-3 grams
11. See Table 4
12. See Table 5
13. See Table 6

14. pulmonary edema

15. Initial

- a. persistent cough
- b. slight dyspnea orthopnea
- c. exercise intolerance
- d. restlessness and anxiety
- e. crepitant rales at lung bases

Acute

- a. acute shortness of breath
- b. respirations – rapid, noisy (audible wheeze, rales)
- c. cough – more intense and productive of frothy, blood-tinged sputum
- d. cyanosis – cold, clammy skin
- e. tachycardia – arrhythmias
- f. hypotension

Advanced

- a. decreased level of consciousness
- b. ventricular arrhythmias
- c. diminished breath sounds

16. diet, lifestyle