CONGESTIVE HEART FAILURE

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ABSTRACT

When the heart is no longer able to adequately provide the body’s organs and tissues with vital oxygen and nutrients, heart failure has occurred. A well-functioning heart circulates blood throughout the body, and when the heart fails to operate at optimal levels, fluid consisting of mostly water leaks from capillary blood vessels. This fluid interferes with the body’s normal processes, most notably the lungs, causing shortness of breath and general weakness and fatigue. Although science has provided several treatment options that can extend the lives of many patients with congestive heart failure, patients have to follow treatment approaches carefully to avoid repeat hospital readmissions.
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This educational activity is credited for 3.5 hours. Nurses may only claim credit commensurate with the credit awarded for completion of this course activity.

Pharmacology content is 0.5 hours (30 minutes).

Statement of Learning Need
Heart failure early intervention and early recognition prevent worsening of symptoms and improve patient survival. Nursing involvement in heart failure clinics and follow up improve patient compliance with treatment, disease progression and quality of life.

Course Purpose
To provide nurses and health associates knowledge about congestive heart failure throughout the course of patient care in different health settings.
**Target Audience**
Advanced Practice Registered Nurses and Registered Nurses
(Interdisciplinary Health Team Members, including Vocational Nurses and Medical Assistants may obtain a *Certificate of Completion*)

**Course Author & Planning Team Conflict of Interest Disclosures**
Jassin M. Jouria, MD, William S. Cook, PhD, Douglas Lawrence, MA,
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There is no commercial support for this course.

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Please take time to complete a self-assessment of knowledge, on page 4, sample questions *before* reading the article.

Opportunity to complete a self-assessment of knowledge learned will be provided at the end of the course.
1. The following terms are used in the classification of CHF except
   a. Acute and chronic heart failure
   b. High output and low output heart failure
   c. Upper and lower heart failure
   d. Left sided and right sided heart failure

2. Left sided heart failure is also known as
   a. Systemic failure
   b. Pulmonary edema heart failure
   c. Respiratory failure
   d. circulation failure

3. Which of the following is also termed as systemic failure?
   a. Acute heart failure
   b. Chronic heart failure
   c. Right sided heart failure
   d. Biventricular failure

4. Following are the most common causes of CHF except
   a. Hypertension
   b. Heart attack
   c. Ischemic heart disease
   d. Appendicitis

5. Deficiency of vitamin B1 leads to which disease?
   a. Paget’s disease
   b. Beriberi
   c. Biventricular failure
   d. Scurvy
Introduction

Congestive heart failure is currently one of the most common life-threatening conditions affecting individuals. The main problem with congestive heart failure (CHF) is that the pumping mechanism of the cardiac muscle is no longer effective, causing an adverse effect on the blood tissue perfusion and thereby resulting into many systemic problems. The development of CHF may be considered as either acute or chronic. An acute condition is usually the result of traumatic injuries to the myocardium while chronic conditions may be related to the presence of other cardiac conditions.

Congestive heart failure affects approximately 5 million individuals in the United States alone, but this number can go higher, as there are approximately 550,000 new cases reported each year.¹ Most of these individuals diagnosed with CHF are between 55 to 70 years old for both genders. However, it has been found that women have lower risk for the development of this condition before the menopausal stage, but the risk equals those of the male population after cessation of menses.² In both cases, most of those who were diagnosed to have CHF are reported to die within five to eight years after the initial diagnosis was made.³

The main problem with congestive heart failure is not the entire cessation of the pumping mechanism of the heart, but rather its inability to meet the demand for oxygen and nutrients for the cells of the body.¹ When there is insufficient or too little oxygen and amount of nutrients reaching to the cells of the body, the normal physiologic processes of the body cells are adversely affected, contributing to multiple organ system failure and the existence of symptoms, such as, shortness of breath, fatigue, and other systemic symptoms like edema and ascites.⁴
When an individual is diagnosed with congestive heart failure, his or her quality of life (QOL) severely decreases over time. This is despite the fact that most of those diagnosed with the condition report no symptoms during the earliest stages.\textsuperscript{1,3} Due to the chronic nature of the condition, the treatment is usually a lengthy process, with pharmacologic and non-pharmacologic measures employed to alleviate the condition. The treatment is totally dependent on the stage at which the individual patient is diagnosed. The lifestyle and dietary changes that need to be followed by the individual is probably one of the most challenging aspects of treatment for nurses and other members of the health care team to implement. The need to drastically shift dietary patterns, give up habits such as alcohol intake and cigarette smoking, are some of the most challenging aspects in the patient’s treatment plan. There is also the presence of psychosocial problems that should be addressed and dealt with not only at the level of the individual but also including his or her family.

Congestive heart failure is explained as a disease process that involves not only a physiologic problem but encompasses the affected individual’s life as a whole. Since the effect of the condition on those affected by it is systemic in nature, it can be said that an understanding and the treatment of CHF requires a holistic approach in order to appropriately deal with it throughout the patient’s course of care.\textsuperscript{1,3,5}

This course addresses the clinical problem that is congestive heart failure, its commonly known classifications or sub-types, causes, signs and symptoms. Moreover, the different diagnostic tools and procedures employed to determine the existence of CHF are discussed in detail; and, in addition, the various treatment options, chronic management and palliative care regimens. Most importantly, this course is specially
designed to include practical guidelines for nurses that care for patients with CHF and may benefit from a review of the protocol for follow up assessment and teaching strategies for patients in terms of attaining an improved outcome.

### Classifications Of Congestive Heart Failure

The signs and symptoms manifested and felt by the individual who is suffering from congestive heart failure varies depending on how the condition has affected the heart’s ability to function properly and supply the demands placed upon it. A CHF classification system is outlined in this section, which helps health providers to obtain a better understanding of the patient’s level of heart failure depending upon the side of the heart affected, type of failure, and the pathophysiology. Heart failure can be classified as high-output heart failure, low-output heart failure, acute versus chronic heart failure, left-sided failure, right-sided failure and biventricular failure, which are further explained in the sections below.

**High output congestive heart failure**

High output congestive heart failure is usually diagnosed when an individual manifests with a condition in which the cardiac output is relatively higher than the expected norm, or larger than the usual demand of the body. This occurs due to the unusually high demand of blood by the body; and, means that the cardiac output reported in patients are usually higher than 8 liters of blood per minute, as compared to the normally accepted cardiac output range of 4 to 8 liters per minute.

When a condition of high output happens it causes overload of the blood in the circulation (circulatory overload). This overload leads to the presence of backflow of blood into the pulmonary cavity (pulmonary edema). The presence of pulmonary edema is usually a result of the elevation of the diastolic pressure in the left ventricle of the heart, which
is responsible for pumping out oxygen and nutrient-rich blood into the systemic circulation.

Most patients with high output congestive heart failure reveal normal systolic pressures upon assessment, but are seen with other symptoms related to heart failure. The literature suggest that because of the imbalance between the systolic and diastolic functioning of the heart, there is presence of an underlying heart problem associated with high output heart failure. Moreover, since the persistence of a larger workload given to the cardiac muscles and higher pressures within the left ventricle results in further deterioration of the heart muscle, which can lead to other physical cardiac defects such as dilatation of the ventricles, cardiac hypertrophy, valvular anomalies, persistence of symptoms such as tachycardia and palpitations and an eventual failure of systolic functioning.

Several factors have been linked to the development of high output congestive heart failure. One of the primary reasons pointed out is the presence of impairment in the systemic vascular resistance. This is a result of defects in the shunting between the arterial and venous systemic circulations or dilation of vessels in the peripheral regions of the body. When either of the two conditions occur the systemic arterial blood pressure can suffer from a substantial drop, which also happens to be one of the primary signs seen in those with low output congestive heart failure. When this happens, activation of the sympathetic nervous system occurs, causing a compensatory reaction to increase the cardiac output and activation of the neurohormonal mediators such as vasopressin and the rennin-angiotensin-aldosterone system (RAS). This series of reactions can result in retention of sodium and water in the systemic circulation and can be the cause of overt congestive heart failure symptoms.
Apart from the mechanisms mentioned above, high output congestive heart failure may also be a result of excessive administration of fluids or blood transfusion, or water retention secondary to steroid therapy. Other health conditions may also be the cause of high output failure such as anemia (in most cases, the severe form of the disease), the presence of arteriovenous (AV) shunts or fistulas, severe renal disease, liver disease, Paget’s disease of the bone, hormonal imbalances such as hyperthyroidism, sepsis and other health conditions. Furthermore, conditions such as obesity and pregnancy are also linked to the existence of high output congestive heart failure. The common denominators among these conditions are their ability to cause an increase in the blood pressure of the individual, systemic and/or peripheral vasodilatation and rise of the total circulating blood volume.8-10

Common signs and symptoms seen in patients with high output heart failure include shortness of breath, or dyspnea, upon exertion. There may also be reports of easy fatigability, activity intolerance, edema and tachypnea. As the condition progresses, patients may also be assessed for other signs and symptoms such as jugular vein distension, tachycardia, presence of pleural effusion and pulmonary rales.8,9 Since the nature of high output failure is mostly related to several underlying causes, its treatment is usually geared towards addressing these causes. If the patient is reported to have the problem due to sodium and water retention, diuretics may be given to reduce circulating blood volume and decrease cardiac workload. In other cases, drugs such as vasopressin are given to promote vasoconstriction in the peripheral vessels (epinephrine and phenylephrine may also be prescribed). Patients with respiratory symptoms are treated with mechanical ventilation or PEEP (peek end-expiratory pressure).9
From a prognostic point of view, high output congestive heart failure generally results in good long-term recovery and most of the cases are curable provided timely treated. Since the heart is normal in the high output syndrome, the treatment is more effective than other types of heart failure.\(^9\)

**Low output congestive heart failure**

Low output congestive heart failure is usually diagnosed when the patient manifests with a relatively low cardiac output. This means that the cardiac output that is usually obtained from the patient falls below 3 liters of blood pumped in 60 seconds. In this condition, there is an inability of the heart to meet the demand of the body for blood under normal conditions.\(^11\) There are two subtypes of low output heart failure: *systolic dysfunction* and *diastolic dysfunction*.

Systolic dysfunction is a problem that is characterized by impairment of the left heart ventricular contraction. In patients who are diagnosed to have congestive and chronic cases of heart failure, one of the primary causes of low output failure is the disturbance of the signals that serve to regulate cardiac rhythm and contractions. This disturbance causes a marked decline in the inotropic capacity of the heart muscles, otherwise known as its contractility.

The loss or reduction of an effective contracting mechanism of the heart results in a compensatory reaction of raising the preload and a subsequent reduction in the stroke volume. The compensatory increase in the preload (usually measured as the pulmonary capillary wedge pressure or the end-diastolic pressure of the ventricles) is due to the activation of the Frank-Starling mechanism in an effort to maintain an adequate stroke
volume despite the contractile problem. The rise of the preload in such cases is needed to prevent the further loss of stroke volume occurring due to decreased contractile power of the heart. In most instances, this results in hypertrophy of the ventricles, dilation or even a mixture of the two problems.\textsuperscript{12,13,14,15}

The usual causes of systolic dysfunction are structural defects in the heart muscle itself as in cardiomyopathies and valvular heart disorders. It can also be a complication of other conditions such as coronary artery disease and severe hypertension.\textsuperscript{13,15} Whereas, diastolic dysfunction is a condition in which a reduction in the performance of one or both ventricles of the heart occurs during the diastolic phase of the cardiac cycle.

The diastole is the phase in which there is relaxation of the heart muscle and the blood coming from the systemic circulation is routed to the right atrium via the superior and inferior vena cavae. Conversely, the blood that is oxygenated in the lungs is also routed into the left atrium via the pulmonary veins in this phase. Most patients that have diastolic dysfunction will present with little or no symptoms. When the symptoms are elicited during the individual assessment there is usually a determination of the pathologic cause of the symptoms. However, it should be also noted that some degree of diastolic dysfunction could occur in an otherwise healthy elderly person.\textsuperscript{11,12,13} The problem occurs when the process of filling up the atria cannot be completed during the diastolic phase because the walls of the heart are either too rigid or thick to allow for filling. This in turn results into a form of hypertrophy that is considered to be \textit{concentric hypertrophy} of heart.
Individuals that are diagnosed to have diastolic dysfunction usually present with an elevation of the diastolic pressure despite having a normal end diastolic volume. Usually the clinical findings include ventricular hypertrophy, an increase in the deposition of collagen in the interstitial spaces of the cells of the myocardium and a marked reduction in the ability of the heart muscles to distend and stretch in response to the demands placed upon it. Because of the lack of the heart’s capacity to adjust, the cardiac output is decreased, and the oxygen tissue perfusion becomes affected.\textsuperscript{13}

Causes of diastolic dysfunction are often related to processes that can cause the left ventricle to stiffen. As a consequence, it produces difficulty for the blood to enter into the left atrium, leading to back flow of blood to the lungs resulting in pulmonary edema. Hypertension, especially in chronic cases, has been pointed out as one of the most common causes of left ventricular stiffness. There is also a link to aortic stenosis, diabetes mellitus, and cardiomyopathies. In some patients, a history of constrictive pericarditis and conditions such as Amyloidosis and Sarcoidosis has also been pointed out as possible causes of diastolic dysfunction.\textsuperscript{12,15}

From a prognostic point of view, low output congestive heart failure carries a relatively poor prognosis in comparison to the high output heart failure since here the problem is with the heart muscles itself. Evaluation of the functioning state of the patient is important to determining the prognosis of the case.

**Left-sided congestive heart failure**

Traditionally, the literature on congestive heart failure lists two major classifications of heart failure most of the time: the right-sided, or
systemic failure, and the left-sided, or pulmonary edema heart failure. In one of these classifications, the left-sided heart failure is mainly characterized by problems with the respiratory system. Signs such as tachypnea, or an increase in the respiratory rate is one of the most common assessment findings in these patients. Moreover, there is also dyspnea, especially felt upon exertion. Crackles or rales, a sign indicating presence of fluid in the pleural cavity, are also heard upon auscultation. These are usually present initially at the bases of the lungs, and then progress to other parts of the pleural cavity as the condition worsens.

Apart from the aforementioned symptoms, patients are also seen to develop cyanosis, which is indicative of oxygen deficiency on a chronic basis. Left-sided congestive heart failure leads to the development of pulmonary edema, or the accumulation of fluid in the lung parenchyma.\textsuperscript{16,17} Patients with left-sided failure, especially involving the left ventricle usually reveal displacement of the apical beat of the heart upon auscultation. This is especially true to those who have developed cardiomegaly as a complication of longstanding left ventricular dysfunction.\textsuperscript{18} Furthermore, increased blood flow and increased pressures within the pericardial cavity results in the development of gallops (extra heart beats). In individuals with valvular disorders coexisting with congestive heart failure, cardiac murmurs become highly audible upon auscultation.\textsuperscript{16}

Left-sided congestive heart failure can also be due to backward failure, in which the vessels leading to the pulmonary circulation is congested, resulting in predominantly respiratory symptoms. The terms \textit{backward} and \textit{forward} failure have been used in recent years, which are also better known as \textit{systolic heart failure} and \textit{diastolic heart failure}.\textsuperscript{78} According to a definition, backward failure of the left ventricle is the condition in which
the left ventricle is able to pump the blood at a sufficient rate only when
the ventricular filling pressure is abnormally high. That is when the
preload is more.\textsuperscript{79}

Backward heart failure occurs due to passive engorgement of the
systemic venous system as a result of dysfunction in a ventricle and
subsequent pressure increase behind it.\textsuperscript{80} It can originate from either the
left ventricle or the left atrium. Sometimes, the problem can stem from
both chambers of the left side of the heart. When both chambers of the
left side of the heart are affected the dyspnea experienced by the patient
can occur upon exertion in mild to moderate cases, but severe cases can
lead to the development of dyspnea even at rest. Orthopnea can also be
seen among these patients, which requires them to sit up while sleeping
so as not to feel short of breath. Additionally, there are patients that
report experiencing \textit{paroxysmal nocturnal dyspnea} or the attack of severe
breathlessness at night, most especially during the first few hours after
sleeping.\textsuperscript{10,16,18}

As the problem at the left side of the heart worsens, the patient usually
complains of activity intolerance and easy fatigability due to poor oxygen
tissue perfusion. Others still may exhibit inspiratory wheezes that worsen
with the progression of left-sided failure. However, when left-sided heart
failure affects the forward functioning of the left ventricle, signs and
symptoms indicating a deficiency in the systemic circulation occurs. These
include confusion or decreasing level of consciousness, dizziness and cold
and clammy extremities when the patient is at rest.\textsuperscript{14,16,19}

Left-sided heart failure is usually managed by treating the underlying
cause. When there is no structural defect involved in the cardiac
musculature, the patient usually responds well to medical management.\textsuperscript{19} Because one of the major problems in this condition is the increased venous return to the heart, positioning the patient upright is done to take advantage of gravity to relieve the congestion. Moreover, the administration of diuretics, such as furosemide, is done to decrease circulating fluid volume and relieve the congestion. Other patients are also given vasodilators such as nitroglycerine to regulate the blood pressure and relieve the congestion of the vessels.\textsuperscript{16,18,19}

The use of inotropic agents, such as digoxin, may be implemented to promote increased contractile power of the left side of the heart and to ensure adequate perfusion. Surgical interventions may also be required to be done in some patients, especially in severe cases or in those who are experiencing cardiogenic shock. For these patients, the insertion of intra-aortic balloon pumps are initiated as a temporary form of treatment to ensure that there will be sufficient cardiac output until the problem is either corrected or properly managed.\textsuperscript{10,14,18,19}

Respiratory syndrome associated with left-sided heart failure is usually treated with BIPAP (bilevel positive airway pressure) or CPAP (continuous positive airway pressure) to reduce the necessity of putting the patient under mechanical ventilation. This is especially true when one of the goals of therapy is to maintain and improve respiratory functioning and decrease the risk of complications associated with mechanical ventilation.\textsuperscript{17,19}

\textbf{Right-sided congestive heart failure}

Although the term heart failure mostly refers to left ventricular failure, heart failure may include both sides of the ventricles (right and left...
ventricle) or may be limited to the right ventricle alone. Right-sided heart failure may occur alone, sparing the left side of the heart, when it is associated with chronic obstructive pulmonary disease. When severely elevated pulmonary arterial (PA) pressure results in right ventricular (RV) failure, it is known as cor pulmonale.\textsuperscript{20,21}

The right-sided heart failure occurs as a result of various mechanisms. The heart pumps the deoxygenated blood through the right atrium into the right ventricle. The right ventricle will then pump out the blood from heart into the lungs for oxygenation. Usually the right-sided heart failure (RV HF) occurs as a consequence of left sided failure (LV HF). When the left sided ventricle fails to pump adequately, the fluid pressure gets elevated and results in back pressure to the lungs; this fluid, which is transferred back through the lungs, leads to damage to the right-sided heart. When this condition persists for a longer period of time, the right-sided heart fails to pump further and blood gets congested and pooled back to the body’s venous system. This results in fluid redistribution in the peripheral circulation especially the extremities.\textsuperscript{22}

Another mechanism through which right sided heart failure occurs is when there is excessive resistance to the blood flow from the right side of the heart structures, such as the right ventricle, right atrium or pulmonary artery. It can also occur due to improper functioning of the tricuspid valve. This process also results in the congestion of fluid and pressure rises in the veins that empty into the right side of the heart. Consequently, the high pressure can also build up in the liver (liver congestion) and the veins of the legs. As the liver becomes enlarged (hepatomegaly) there may also be pain associated with it. Additionally, congestion in the extremities produces signs of swelling in the ankles or legs.\textsuperscript{23}
There are several causes responsible for right side heart failure, such as: RV failure with normal after load, RV infarction, RV failure secondary to increased after load, pulmonary embolus, mitral valve disease with pulmonary hypertension, congenital heart disease, acute respiratory distress syndrome (ARDS), obstructive sleep apnea, increased after load complicating cardiac surgery, inflammatory effects of cardio-pulmonary bypass (CPB), protamine use, increased after load complicating thoracic surgery, extensive lung resection, left ventricular assist device, RV failure secondary to volume overload, atrial septal defect, and ventricular septal defect. These conditions can be broadly divided into the following categories:

1. Intrinsic RV failure in the absence of pulmonary hypertension (usually, RV infarction);
2. RV failure secondary to increased RV after load; and,
3. RV failure because of volume overload.

The main presenting symptoms of right-sided heart failure are edema and nocturia (excessive urination at night caused by fluid redistribution while a person is lying down). There are different varieties of edema that can be present, such as dependent edema (edema that travels by gravity to the lowest portions of the body), edema of hepatic region that results in enlargement of the liver, edema of a serous cavity (ascites), and occasionally edema of the skin or soft tissue. Clinical signs are inclusive of peripheral edema, such as, right-sided third heart sound, increased split of the second heart sound, jugular venous distension, systolic murmur of tricuspid regurgitation, pulmonary embolus, deep vein thrombosis, increased D-dimers, and type I respiratory failure. Since congestive heart failure leads to excessive retention of fluids in areas of the body, the kidneys may also be affected and not able to excrete extra sodium.
and water leading to kidney failure. The sodium retention can become exacerbated, and develop into excessive fluid retention and aggravated systemic congestion leading to overall worsened symptoms of congestive heart failure.

Right ventricular failure can also occur with normal RV afterload as a consequence of myocardial infarction. The electrocardiogram (ECG) shows abnormalities, such as, right axis deviation, right ventricular hypertrophy (RVH), and right bundle branch block (RBBB). Chest x-ray may reveal enlargement of the main pulmonary artery (PA), oligemia of a pulmonary lobe (Westermark’s sign), and a distended azygous vein; additionally, echocardiography may show dilated, hypertrophic, or poorly contractile RV, tricuspid regurgitation and septal shift.

Poor blood supply to the right side of the myocardium causes right ventricular infarction leading to heart failure, shock, cardiac arrhythmias and death in the absence of pressure overload independent of left ventricular damage. Under these circumstances, if the right ventricle is bypassed (Fontan procedure), the circulation can be maintained but when pathological a hypcontractile right ventricle is present it plays an active role in compromising the overall status of circulation.

A common classification system, established by the New York Heart Association, Class I, II, III, IV, for heart failure is based upon the patient’s subjective report of functional activity limitations as compared to their ordinary level of physical activity. These are described below.
Class I:
4.0 – 6.0 cal/min, seen in a patient with cardiac disease but without resulting limitations of physical activity. Ordinary physical activity does not cause undue fatigue, palpitation, dyspnea, or anginal pain.

Class II:
3.0 – 4.0 cal/min, seen in a patient with cardiac disease resulting in slight limitation of physical activity. They are comfortable at rest, and ordinary physical activity results in fatigue, palpitation, dyspnea or anginal pain.

Class III:
2.0 – 3.0 cal/min, seen in a patient with marked limitation of physical activity. Less than ordinary physical activity causes fatigue, palpitation, dyspnea or anginal pain.

Class IV:
1.0 – 2.0 cal/min, seen in a patient with an inability to carry on any physical activity without discomfort. Anginal pain is also present at rest. If any physical activity is undertaken, discomfort gets increased.

**Acute versus chronic congestive heart failure**

Many different terms and phrases are used to describe patients with congestive heart failure depending upon the HF classification being used. Congestive heart failure is classified based upon the onset and the duration of symptoms, such as those associated with acute onset and belonging to a chronic condition. Physicians may use the general
classification of acute versus chronic CHF with a slight difference. The term acute heart failure may be a little confusing to patients because some clinicians use this term to describe severity while others use it to describe decompensated and recent- or new-onset heart failure. The true definition of the word *acute* refers to an indication of time (or onset of a disease state) rather than to the severity of a disease.

When using words such as acute, advanced, and decompensated clinicians should avoid using them interchangeably while discussing heart failure with patients. There is a clear distinction between new-onset HF, transient HF, and chronic HF. The term *new-onset heart failure* is self-explanatory and refers to when the first incidence of HF is presented. The term *transient heart failure* refers to symptomatic heart failure over a limited time period, even though long-term treatment may be indicated. The term *worsening heart failure* (or chronic HF) is the most common form of heart failure, which needs hospitalization and accounting for 80% of cases.²⁷

The European Society of Cardiology has provided clear guidelines for the diagnosis and treatment of acute heart failure. According to these guidelines the patients with heart failure are classified into 1 of 6 groups on the basis of typical clinical and hemodynamic characteristics.²⁸ The guidelines divide acute heart failure into the following groups of patients:²⁸,²⁹

I. Acute decompensated heart failure
II. Acute heart failure with hypertension/hypertensive crisis
III. Acute heart failure with pulmonary edema
IVa. Cardiogenic shock or low output syndrome
IVb. Severe cardiogenic shock
V. High output failure
VI. Right sided heart failure

The first three groups of patients comprise over 90% of acute heart failure (AHF) presentations. The patients with acute decompensated heart failure (ADHF) typically present with signs and symptoms of mild to moderate levels of congestion. Patients with hypertensive acute heart failure have relatively preserved left ventricular (LV) systolic function with (LV) ejection fraction >0.40, elevated blood pressure and pulmonary edema.

Patients having acute heart failure with pulmonary edema characteristically present with predominated symptoms of severe respiratory distress, orthopnea, signs of pulmonary edema, and hypoxemia (the oxygen saturation is usually <90% on room air). Patients having low-output syndrome present with symptoms resulting from heart failure and tissue hypo-perfusion, and they display a variety of symptoms ranging from a low-output state to cardiogenic shock. High-output failure is a less common cause of acute heart failure and usually presents with warm extremities, pulmonary congestion, along with a combination of low blood pressure, high cardiac output and generally an elevated heart rate.28,29,30

Stevenson and colleagues have also provided a system of classification for acute decompensated heart failure. Unlike the European Society of Cardiology system, this classification system focuses on the severity of the disease rather than the cause of heart failure. In this system patients are classified on the basis of the clinical presence or absence of hypo-perfusion and of congestion at rest.31
On the other hand, the term chronic heart failure is used to describe the prolonged nature of heart failure. Chronic heart failure usually occurs as a result of the long-term effects of various system illnesses, such as, hypertension, diabetes mellitus, sarcoidosis, amyloidosi, valvular heart disease, cardiomyopathy, and recurrent infection. Acute heart failure may result in further damage to the heart muscles leading to a chronic state of heart failure. Often, chronic heart failure is naturally compensated for in individual cases, requiring long-term treatment with multiple lifestyle modifications to achieve a stable state.

Chronic heart failure leads to long-standing hypo-perfusion of tissue with long-term consequences of multiple system hypo-perfusion. The management of acute and chronic heart failure remains different and are totally dependent on the short and long term goals of treatment. The primary goal of treating acute heart failure is to achieve a hemodynamically stable state through emergency interventions. While the management goal of chronic heart failure is to maintain a stable state and to prevent any episodes of acute heart failure.

**Biventricular heart failure**

As the name suggests, biventricular hear failure refers to the non-functioning of both sides of the heart muscle. In this type of heart failure both (left and right) sides of the ventricles are involved. Biventricular heart failure occurs due to systolic heart failure. It is a more severe degree of heart failure than the independent one-sided (left or right sided) heart failure. As discussed earlier, the patients with right-sided heart failure have main presenting symptoms of systemic congestion whereas the patients with left sided heart failure have predominant pulmonary congestion. In the biventricular type of heart failure, since both of the ventricles are malfunctioning, both pulmonary as well as
systemic congestion is involved. Biventricular failure occurs due to the whole myocardium being affected; such as, in cases of massive myocardial infarction (MI) affecting the anterior and posterior walls of the heart, cardiac arrhythmias, chronic left ventricular failure causing right ventricular failure, viral myocarditis and Chagas disease.\textsuperscript{3}

The presenting symptoms of the patient with biventricular heart failure remain very similar to that of right-sided heart failure due to the fluid retention. Usually the patient presents with swelling of legs, increased abdominal girth, weight gain due to fluid retention, shortness of breath, and fatigue. Biventricular failure classically presents with dyspnea, dependent edema, jugular venous distension, pulmonary vascular congestion and bilateral reduced contractility that differentiates the biventricular from the right-sided heart failure. The presence of dyspnea along with dependent edema confirms the heart failure as the biventricular category.\textsuperscript{72}

On examination, there is a mixture of signs involving left as well as right-sided heart failure. Pulmonary signs of fluid retention are evident on auscultation. On pulmonary examination, crackles (rales or crepitation) are found which is suggestive of pulmonary edema. Pleural effusion is a very common x-ray finding in the patient with biventricular failure.\textsuperscript{20} Extremities tend to be swollen and show signs of pedal edema. Cardiac cachexia (severe weight loss) is also seen in severe cases of biventricular failure, which is suggestive of a poor prognosis and high mortality.\textsuperscript{3,20} The patient often requires resynchronization of the heart through biventricular pacing, which stimulates both sides of the ventricles to improve the coordination of ventricular contraction.\textsuperscript{20}
From a prognostic standpoint, the biventricular failure carries a higher risk of morbidity and mortality. Global damage to the heart leads to compromise in the systemic circulation, and management is difficult due to other systems becoming involved as a result of chronic hypo-perfusion, such as in cases of renal failure, muscle wasting, metabolic acidosis and hypoxemia.

**Causes Of Congestive Heart Failure**

The prior section provided an overview of congestive heart failure outcomes and classification systems. This section discusses a variety of factors that predispose individuals to congestive heart failure. The conditions in which either the preload and/or afterload are increased or those in which the myocardial contractility is affected cause congestive cardiac failure. The most common cause of heart failure is coronary artery disease (CAD), also known as ischemic heart disease (IHD). Other common causes are hypertension, valvular heart diseases, alcohol, infective diseases, diabetes mellitus, and many systemic diseases.

The etiology of congestive heart failure plays a significant role in deciding the prognosis of the disease. The outcome of the disease largely depends on the cause that led to cardiac failure. For example, in one study, the frequency of sudden death was found to be the highest in patients with CHF caused by dilated cardiomyopathy (25%), followed by those with CHF caused by valvular heart disease (18%) and those with CHF caused by ischemic heart disease (17.5%).

It is very important to know the cause of congestive heart failure in order to know the prognosis as well as to decide the course of treatment. The course of treatment in cases of hypertensive congestive heart failure will
aim at controlling and preventing a further rise in blood pressure, whereas, in cases of valvular heart disease the aim will be to correct the malfunctioning of the valve medically or surgically. Thus, the treatment course will be different for heart failures due to different causes. The following section provides an overview of the common causes of CHF.

**Cardiac arrhythmias**

The heart has its own special electrogenic system for generation and conduction of electrical impulses. This specialized excitatory and conductive system of the heart consists of the following parts: sinoatrial node (SA node), atrio-ventricular node (AV node) and bundle of HIS or the purkinje fibres. These are also responsible for maintaining the rhythm of the heart. Sympathetic and parasympathetic systems also play a regulatory role.

In situations of arrhythmia occurrence, as the name suggests, the rhythm of the heart becomes abnormal. Abnormal rhythm arises due to a change in the normal sequence of electrical impulses. The abnormal electrical impulse may cause the heart to beat too slowly, too fast or irregularly.

Arrhythmia is caused by 5 main factors. The first factor is when the rhythm of the pacemaker becomes abnormal, and arrhythmia occurs; the second factor is when the shift of the pacemaker from the sinus node to another location in the heart occurs; the third factor is when the spread of the impulse is blocked at some point in the heart; the fourth factor is when the impulse transmits through abnormal pathways in the heart; and, lastly, due to the generation of spurious impulses at any part of the heart. These are all factors that may cause arrhythmias. Abnormal sinus rhythms are tachycardia, bradycardia and sinus arrhythmia. Abnormal
rhythms resulting from impulse conduction block are sinoatrial block and atrioventricular block.

Premature ventricular contractions and premature atrial contractions also contribute to arrhythmias.\textsuperscript{33} Ventricular fibrillation is the most serious of all arrhythmias and causes death within minutes. Atrial fibrillation is another cause of irregular impulse, which is not a fatal rhythm but often requires treatment to prevent an adverse outcome. Studies have found that more than half of the congestive heart failure-related deaths are sudden, and presumably due to ventricular arrhythmias. The prevalence of ventricular tachycardia has also been found to be quite high (approximately 54\%). Some new studies now suggest that ventricular arrhythmias may independently influence prognosis in patients with CHF.\textsuperscript{33} In arrhythmias due to atrial fibrillation tachycardia occurs. This does not allow proper filling of the heart and hence resulting in reduced cardiac output. It also puts backpressure on the vessels causing congestion or pooling of blood.

In arrhythmias due to tachycardia myopathy, along with the aforesaid cause, relentless beating of the heart at a fast pace does not allow the heart to rest. And hence the muscles get fatigued. In arrhythmias due to complete heart block, even though the stroke volume remains the same, bradycardia reduces the cardiac output. This gradually leads to cardiac failure.\textsuperscript{34}

**Myocardial infarction**

Myocardial infarction, commonly referred to by non-medical laypersons as a heart attack, is defined as an irreversible loss of muscle fibers due to necrosis secondary to prolonged ischemia. After the heart becomes
damaged due to sudden myocardial infarction, mechanisms of the body jump into action to restore normal function. Collateral blood supply develops and it saves the partially damaged portions from further damage and restores the function of the areas where reversible damage has occurred. On the other hand, the healthy tissue takes over the functions of the lost areas. The tissue hypertrophies and makes up for the damage that has occurred.\textsuperscript{6}

Over time, patients with a history of myocardial infarction develop heart failure due to myocardial necrosis with consequent ventricular remodeling. The damaged area weakens and the left ventricular wall thins out in the area of infarction. Hence, the ventricular cavity dilates. Compensatory hypertrophy also occurs. Aldosterone (a steroid hormone) lays down an adverse effect on the ventricular remodeling as it produces left ventricular dysfunction and fibrosis. Endothelial walls become damaged as a result. Left ventricular dysfunction is the contributing factor in the progression of heart failure; and, left ventricular fibrosis contributes to arrhythmia.\textsuperscript{35}

All of these aforementioned compensatory mechanisms initially prove to be beneficial as they help to maintain a normal ejection fraction; however, over time as the functions of the heart deteriorates and as more and more heart cells (myocytes) die, the patient’s condition will evolve to heart failure.

**Cardiomyopathy**

Cardiomyopathy is a condition in which diseases of the myocardium damage the musculature of the heart. The heart musculature may
elongate, thicken or become rigid. There may be fibrosis and scarring. Such conditions are outlined in the section below.

*Ischemic cardiomyopathy*

Coronary heart disease is a disease in which plaque builds up inside the coronary arteries impairing the blood flow to the heart musculature resulting in ischemia. The flow of oxygen rich blood is hampered and the muscles weaken over time. The muscles cannot then pump enough blood needed in various areas of the body. Under stressful conditions vasodilation of the vessels of the heart occurs; but the vessels where the plaque is situated are unable to dilate as needed leading to further damage to the ischemic area. This results in a vicious cycle that ultimately leads to heart failure.

Coronary artery disease is the major contributor to progression of left ventricular systolic dysfunction, which causes heart failure. In coronary heart disease, the ischemic segments become akinetic or dyskinetic; the segments cease contracting or contract poorly. This negatively impacts healthy body tissues that rely upon the heart to circulate blood by hampering their normal function. Coronary artery disease leads to a distortion of the normal pattern of heart contraction and relaxation, which is also known as segmental dysfunction of the ventricle.

In the past two decades, it has been found that the leading cause of heart failure is not hypertension or valvular heart disease but is coronary artery disease or ischemic heart disease, which has emerged as the leading cause of chronic heart failure. In study trials reported by the New England Journal of Medicine over the past 10 years, coronary artery disease was the underlying cause of heart failure in almost 70% of patients.
Hypertrophic cardiomyopathy

The importance of ventricular hypertrophy as a powerful predictor of the prognosis of heart failure, irrespective of the blood pressure and other cardiovascular risk factors, is widely recognized. Left ventricular hypertrophy is the most common hypertrophy of the heart and is proportional to the systolic blood pressure in terms of its overall effect upon heart function. Increased blood pressure causes increased peripheral resistance and hence more afterload. The muscle mass of the ventricle undergoes hypertrophy to compensate for the increased workload.

Other factors that play an important role in the pathogenesis of ventricular hypertrophy are age, sex, race, body mass index and stimulation of the renin-angiotensin-aldosterone and sympathetic nervous systems. Changes in myocardial architecture occur as a result of these contributing factors. The heart’s muscle fibers (myocytes) hypertrophy, and interstitial fibrosis and thickening of intra-myocardial coronary arteries occur. Early diastolic dysfunction and late systolic dysfunction also occur leading to congestive heart failure.38

Cardiac hypertrophy is of two types: concentric and eccentric hypertrophy. Concentric hypertrophy, in which the increased mass is not in proportion to the chamber volume, occurs when there is pressure overload. To overcome the wall stress and the increased systolic pressure, new muscle fibers (myofibrils) are laid parallel to the old ones. This causes wall thickening and hence is named concentric hypertrophy.20

On the contrary, in eccentric hypertrophy there is a great increase in the cavity size. This occurs when there is volume overload and the heart
fibers are laid down in an irregular, series addition. The result is heart chamber enlargement due to increased heart preload, which results in an eccentric, much larger, kind of hypertrophy.\textsuperscript{20}

Cardiac wall hypertrophy is a compensatory mechanism of the heart to meet the increased workload. Initially it restores the elevated wall stress to normal. However, when it proves to be insufficient in restoring the heart’s functionality then heart failure develops. Severe hypertrophy also impairs ventricular filling during diastole and it may cause myocardial ischemia. The musculature of the heart is supplied with blood during diastole when the muscles are relaxing. The increased demand due to increased muscle mass and the improper filling of blood vessels contribute to myocardial ischemia.\textsuperscript{20}

\textit{Dilated cardiomyopathy}

Today, dilated cardiomyopathy has emerged as the third most common cause of heart failure.\textsuperscript{39} Dilated cardiomyopathy is a progressive disease of the heart muscle. Contrary to the hypertrophy where the muscle mass increases, in dilated cardiomyopathy the muscle mass does not increase. Only the ventricular chamber enlarges while the left ventricular wall thickness remains the same. It is characterized by contractile dysfunction. Initially, the dilatation allows the heart to generate a high stroke volume but as the condition of the heart worsens, the contractility is impaired. The dilatation that occurs increases the heart wall stress and thereby reduces contractility (also known as Laplace’s Law or the description of blood flow).

Hypertrophy and dilatation of the heart occur in response to low cardiac output. The reason for dilatation is explained by the Frank-Starling Law,
which states that myocardial force is directly proportional to the muscle length. The myocardial force at end-diastole compared with end-systole increases as muscle length increases, thereby generating a greater amount of force as the muscle is stretched. Prolonged stretching, however, leads to the loss of elasticity, permanent lengthening and failure of the heart muscle contractile unit. Thus, the heart muscle fibers are unable to contract enough to pump the maximum blood out of the heart. Dilated cardiomyopathy is characterized by ventricular chamber enlargement and contractile dysfunction with normal left ventricular (LV) wall thickness. The right ventricle may also be dilated and dysfunctional leading to a progressive heart failure.

**Valvular heart disease**

Any abnormality in the heart valves hampers the normal blood flow in the heart. The two most common forms of valvular heart disease are: *stenosis* and *regurgitation*. In stenosis, the valvular orifice becomes narrow and the blood does not flow smoothly. Due to this hindrance the heart has to work hard to pump blood more efficiently. Examples of valve stenosis are mitral valve stenosis, aortic stenosis, tricuspid valve stenosis and pulmonary valve stenosis. The most common valvular stenosis is aortic stenosis.

In patients with valvular aortic stenosis, the severity of stenosis increases over time. The left ventricle adapts to the obstruction by concentric hypertrophy (characterized by wall thickening while maintaining a normal left ventricle chamber size). And, when this compensatory mechanism fails, heart failure occurs.
On the other hand, regurgitation occurs due to valve prolapse. Any infection in the valves such as myocarditis or endocarditis also causes damage to the valves and hence their efficiency. Globally, rheumatic heart disease remains the most common cause of aortic regurgitation. Coronary heart disease and hypertension also cause the valves to become incompetent over a long span of time. The incompetent valves are unable to prevent the back flow of blood and the blood starts leaking into the previous heart chamber causing regurgitation. Back pressure results, which leads to congestion of blood in the blood vessels. The various types of regurgitation are tricuspid regurgitation, pulmonary regurgitation, mitral regurgitation or aortic regurgitation; and, 13% of the cases of congestive heart failure are caused by valvular heart disease.

In cases of aortic valve prolapse, during systole the heart pumps blood into the aorta and during diastole, when the ventricle is relaxing, the blood starts leaking back into the left ventricle due to valvular insufficiency. Severe valvular regurgitation causes myocardial dilatation to occur and an increase in preload and afterload. The volume overload in severe aortic regurgitation contributes to an increase in preload, and afterload is also increased because the elevated end diastolic volume increases left ventricle wall stress. There is an increase in stroke volume as a result of blood being pumped into a highly resistant aorta with corresponding development of systolic hypertension. This in turn further increases the afterload.

Preload and afterload combined together lead to progressive dilatation of the left ventricle. Dilatation increases the wall stress and decreases the shortening of the muscle fibers, leading to chronic heart failure. Similarly, in cases of mitral valve regurgitation, the left ventricle develops compensatory changes. Left ventricle dilatation and eccentric hypertrophy
(as a result of volume overload) also occurs, which are all contributing causes of congestive heart failure.\(^{40}\)

**Hypertension**

Hypertension is the most common risk factor for congestive heart failure. A study revealed that hypertension had a high risk for CHF, accounting for 39\% of cases in men and 59\% in women. Kannel and colleagues demonstrated that hypertensive men had a nearly 8-fold risk of developing CHF compared with normotensive men.\(^{41}\) Similarly, hypertensive women had a 4-fold risk compared with normotensive women. The prognosis of hypertensive CHF was poor; only 24\% of men and 31\% of women survived 5 years.

Prevention and control of high blood pressure brings down the number of cases of heart failure and mortality.\(^{42}\) Hypertension increases afterload, which results in the heart needing to pump blood with greater force. To review, afterload is the volume and pressure of blood in the ventricle during systole;\(^{34}\) or, it can also be defined as the tension that the muscle is required to develop during contraction. The aortic pressure determines afterload; and, the peripheral vascular resistance in turn determines the aortic pressure.\(^{20}\) The increase of the vascular resistance is directly proportional to the increase of the aortic pressure and hence the afterload. Hypertension occurs when either prolonged sympathetic over activity causes vasoconstriction or the vessels harden due to arteriosclerosis resulting in increased peripheral resistance.

Over time, hemodynamic overload causes compensatory hypertrophy of the heart muscle to occur, and the hypertrophy that occurs is of a concentric type.\(^{34}\) Compensatory hypertrophy restores the previously
elevated ventricular wall stress. A high systolic pressure is generated which helps to maintain normal output and helps cater to the needs of the body. If the condition of the heart further deteriorates, and the hypertrophy proves to be insufficient to meet the relatively increasing demand and decreasing cardiac efficiency, then further heart failure with the ventricular dilatation occurs.

A vicious cycle starts when the heart’s compensatory mechanisms start to occur. In an intact heart with acute cardiac failure these mechanisms are of utmost importance to help restore the blood supply to vital parts of the body and to return the heart functions to normalcy. These are the lifesaving mechanisms that play a pivotal role in saving the life of a patient in acute cardiac failure. However, in cases of chronic heart failure these mechanisms can contribute to the decline of heart function and further deteriorate a patient’s condition.20

When the heart fails to supply enough blood to body tissues there is negative feedback that causes the sympathetic nervous system to activate. In hypertensive cardiac failure this causes further increase in the afterload by raising vascular resistance. It may further cause arrhythmias and damage myocytes. The renin-angiotensinogen-aldosterone system also activates in response to decline in the cardiac output. Angiotensin 2 causes excess vasoconstriction and aldosterone causes retention of salt and water, which further increases the afterload. Endothelin 1 is also increased in heart failure and causes excessive vasoconstriction. In hypertensive heart failure, all the factors mentioned above combine to cause a devastating effect on the patient and the prognosis in such cases is very poor.20
Anemia

Anemia causes high output cardiac failure. Due to the increased demand in anemia the heart has to persistently work hard, manifest a high ejection fraction or stroke volume, and after some time all of the compensatory mechanisms fail to keep up with the demands of body and the heart fails. Studies have shown that anemia is an independent risk factor for death in CHF, and that correction of anemia through transfusions can correct CHF in a patient. Anemia in heart failure almost doubles the rate of mortality.

Anemia causes hypoxia in the tissues due to poor oxygen supply. This causes vasodilatation. Vasodilatation in turn lowers the blood pressure. As suggested earlier, low blood pressure activates the sympathetic nervous system. This brings about vasoconstriction and tachycardia. The vasoconstriction also causes the glomerular filtration rate (GFR) to lower and thus activating the renin angiotensinogen aldosterone system. This further contributes to vasoconstriction and fluid and salt retention. Additionally, extravascular and intravascular fluid increases. The increased blood volume puts a high workload on the heart by increasing both the preload and the afterload. As raised earlier, the heart undergoes remodeling by ventricular dilatation and hypertrophy, and myocardial injury and cardiac fibrosis develop, which finally develops in heart failure.

Due to renal insufficiency, erythropoietin production lowers and anemia worsens. As CHF worsens there are certain cytokines released, such as TNF alpha, which leads to a vicious cycle and worsening of anemia.45
Beriberi

Beriberi is a disease caused by deficiency of vitamin B1. It causes right heart cardiomyopathy and high output failure. Vasodilation occurs thereby reducing the peripheral resistance. This increases the venous return and, hence, the cardiac stroke volume. Initially, the blood pressure is maintained at its normal level. The kidneys and the skin try to maintain the blood pressure through vasoconstriction. However, later on as the compensatory mechanisms fail, the blood pressure starts falling to critical levels and ultimately leads to death. Beri Beri also presents with arrhythmias and there is further damage to the heart.46

Beriberi is of two types: dry beriberi and wet beriberi. The wet beriberi is the type that has the cardiac involvement. The cardiac failure occurs in the three following three steps. Firstly, peripheral vasodilatation occurs and a high cardiac output state ensues. The renin angiotensinogen aldosterone system activates, and results in vasoconstriction and fluid and salt retention in the body. With the increasing vasodilatation, density of the blood decreases due to dilution and the kidneys respond by conserving even more salt. When the salt is conserved, it causes fluid retention as well. The fluid starts to collect in the dependent parts of the body.

Due to the fluid overload in the body, the heart works more than its capacity and cardiac injury occurs. Over time, the cardiac injury resulting in cases of wet beriberi develops into cardiac failure.

Another form of wet beriberi that is more severe and rapid is acute fulminant cardiovascular beriberi, or Shoshin beriberi. Injury to the heart musculature occurs initially and then results in failure of the heart to
pump blood to meet the demands of the body. This is considered a severe form of beriberi because if no treatment is provided then death follows soon.47

**Thyrotoxicosis**

Thyrotoxicosis is a hyper metabolic state that involves excess synthesis and secretion of thyroid hormones by the thyroid gland. In a study, Sandler and Wilson elicited cardiac symptoms in 33% of patients of hyperthyroidism; of these, 57% had pre-existing ischemic, hypertensive or valvular heart disease. Due to hyperthyroidism there is a hyper metabolic state that develops and the demand for energy increases. The energy expenditure is also high. The heart undergoes adaptations to meet the high demand. An increase in mRNA coding for contractile elements and for the sarcoplasmic reticulum occurs. Left ventricular hypertrophy is the eventual result for patients with thyrotoxicosis.

Although direct evidence of sympathetic over activity has not been found, it is postulated that in thyrotoxicosis there is increased sensitivity to catecholamines. In thyrotoxicosis, increased numbers of beta adrenoceptors and increased cAMP concentration in response to insulin induced hypoglycemia is suggestive of an increased sensitivity to the sympathetic system.

Atrial tachyarrhythmia is another factor found to occur in hyperthyroidism and may be responsible for precipitating heart failure. Premature atrial depolarisations, atrial fibrillation and atrial flutter all occur in the state of hyperthyroidism. A lowering of the threshold of atrial depolarization might be the cause of arrhythmias. Additionally, an increased incidence of mitral valve prolapse in hyperthyroidism has been reported.48
**Atriovenous fistula**

Atriovenous (AV) fistula is an abnormal connection between the vein and the artery causing the mixing of the venous and arterial blood. It may be caused as a result of surgery, trauma, and rupture of an arterial aneurysm or may be present congenitally since birth. It is more commonly the result of an artificially created arteriovenous fistula such as is performed for hemodialysis.

Often patients presenting with this kind of heart failure will have a surgical history where an AV fistula has been performed. This causes high output cardiac failure. Since the pressure of the arterial blood is high, a large amount of blood is shunted to the vein causing a fall in peripheral resistance and, hence, the blood pressure; and, there is an increase in venous return volume.

A large volume of venous blood causes an increase in cardiac preload. And the decrease in the arterial supply to the tissues causes there to be an increase in blood demand (in response to an increase in cardiac output). Over time, the heart tries to compensate by developing hypertrophy and dilatation. Cardiomyopathy develops and eventually heart failure results. Physical manifestations include tachycardia, elevated blood pressure, hyperkinetic precordium, and jugular venous distension. Generally, the AV fistula is quite large and is usually located in the upper arm, closer to the heart.49

**Cor pulmonale**

Cor pulmonale is the alteration in the structure and function of the right ventricle caused by a disorder of the respiratory system. Usually, a
Pulmonary vasoconstriction occurs as a physiologic response to alveolar hypoxia. Vasoconstriction results in pulmonary hypertension. Various parenchymatous diseases, such as interstitial lung disease or alveolar diseases, for example, emphysema, decrease the vascular bed and there is obstruction to the blood flow that occurs. The obstruction increases resistance and, hence, hypertension. Blood disorders such as sickle cell anemia and polycythemia increase the viscosity of the blood. This also results in an increase in the blood pressure.

Idiopathic pulmonary hypertension is another common cause that is prevalent. Initially the right ventricle maintains high output through hypertrophy and dilatation. However, over time there is damage to the myocardium that occurs and the ventricle is no longer able to maintain adequate blood supply leading to a decline in blood circulating to the lungs. This further causes a decline in the return of blood to the left atrium and ultimately the left ventricle. Thus, the left ventricle is only able to pump insufficient blood.

The artery that supplies the musculature of the right ventricle also receives and supplies less blood, which causes further damage to the
right ventricular musculature. Thus, the ejection fraction of the right ventricle is further lowered and, as raised earlier, this leads to a vicious cycle where heart failure ensues. The decreased cardiac preload and available blood supply to the musculature of the left ventricle leads to damage of the left ventricle and, hence, to an irreversible condition of both left- and right-sided cardiac failure.50

**Paget’s disease of bone**

Paget’s disease is the disease of bones in which abnormal destruction and remodeling of bones occur. It is characterized by abnormally high resorption of bone and then followed by bone formation. This occurs in a highly disorganized fashion resulting in deformity. The resultant bone is less compact, more vascular, larger and weaker. This new bone becomes more prone to fracture as compared to the previous healthy bone. The disease is idiopathic and the cause of the disease remains largely unknown.

In patients with Paget’s disease bones extensively affected lead to increased bony vascularity. This increased vascularity demands a high supply of blood, which is a condition that may lead to high output cardiac failure.51

**Rheumatic heart disease**

Rheumatic heart disease is inflammation occurring in the heart in reaction to cell mediated and humoral autoimmune response to infection with groups A hemolytic streptococci. It usually occurs 1–3 weeks after the onset of streptococcal pharyngitis. In the acute stage it causes pericarditis, myocarditis and endocarditis. However, when the disease
progresses to become chronic, fibrosis in the valves develop and it ultimately results in valvular stenosis or insufficiency.

The valves most commonly affected in rheumatic heart disease are mitral, aortic, tricuspid and pulmonary valves. In the long run, the disease causes thickening and fibrosis of the valves. The valve orifice becomes stenosed. Less commonly, valvular insufficiency may also occur, which leads to cardiac failure.\textsuperscript{52}

**Infective endocarditis**

Infective endocarditis is defined as inflammation caused by infection of the endocardial surface of the heart. The pathogens responsible for the infection are streptococcus, staphylococcus and cornaebacterium. Certain fungi such as candida albicans are also responsible for endocardial infection.

Infection affects one or more heart valves, the endocardium of the walls, or a septal defect. In infective endocarditis, bacteria adhere to the low-pressure areas of the heart and invade and damage the leaflets of heart valves. The most commonly affected valve is the mitral valve, followed by the aortic valve, then the tricuspid valve and, rarely, the pulmonary valve. If left untreated, infective myocarditis causes severe valvular insufficiency and leads to intractable congestive cardiac failure.\textsuperscript{53}

**Myocarditis and pericarditis**

As explained above, myocarditis is inflammation of the myocardium or the muscle mass of the heart. It is caused by infections of bacterial and viral origins of which viral causes predominate. It is also caused by non-
infective causes. Additionally, the cause of infection may be due to autoimmune reactions of the body and to toxic exposures of the myocardium. Inflammatory mediators accumulate in the myocardium leading to myocyte injury. It causes necrosis and degeneration of the adjacent myocytes, and causes inflammatory cardiomyopathy and leads to cardiac dysfunction.

Pericarditis is inflammation of the sac like layer that covers the heart known as the pericardium. The inflamed pericardium disrupts the normal rhythm of the heart and may predispose to or aggravate cardiac failure if the pericarditis is severe and persistent.73

**Congenital heart diseases**

There are various congenital diseases that are present since birth and that alter the normal blood pumping mechanism. Examples of congenital heart disease are outlined below.

**Septal defects**

Septal defects occur when there are defects in the walls that separate the chambers of the heart, and the path of blood flow becomes altered. For example, atrial septal defect (ASD) is the defect or hole in the septum between the two atria. This causes the mixing of oxygenated and deoxygenated blood increasing the workload on the heart to cater to the increasing demands of the body tissues.

Ventricular septal defect (VSD) is the defect in the wall that separates the two ventricles. Mixing of the blood takes place and the aorta is not
supplied with an adequate amount of oxygenated blood. Also, the volume overload in the right ventricle causes backpressure effects on the right atrium.

*Patent ductus arteriosis*

Patent ductus arteriosis (PDA) is another common congenital heart disease commonly found. This condition occurs when a blood vessel connecting the aorta and pulmonary vessel necessary for pulmonary circulation during the fetal life remains patent and does not obliterate. This patent blood vessel forms a connection between the aorta and pulmonary artery. This causes the oxygenated blood to mix with the deoxygenated blood of the pulmonary artery thus increasing the workload of the heart. It also contributes to increased volume in the lung arteries and thus increases the pulmonary pressure.\(^{54}\)

*Valvular defects*

Other congenital defects include defects in the valves. The types of defects that are present are stenosis, atresia and regurgitation. Most commonly found is pulmonary valve stenosis. Another complex congenital heart disease is Tetralogy of Fallot. It is a combination of the four defects namely, pulmonary valve stenosis, a large ventricular septal defect, aorta stenosis (situated near to the ventricular septal defect) and right ventricular hypertrophy. Tetralogy of Fallot also leads to heart failure if left uncured.

Many types of congenital heart diseases increase the workload of the heart causing the heart to work harder. Over the course of time this causes heart failure.\(^{54}\)
Aneurysm of aorta

The walls of the aorta may weaken and subsequently result in ballooning of the aorta. It may occur at any part of aorta, such as the root of the aorta, the aortic arch or the descending aorta. The greatest risk that aneurysm of the aorta poses is rupture or dissection which may very instantly cause death.

In the pathogenesis of heart failure, this condition leads to dilation of the aortic valve. Dilation causes aortic valve regurgitation and subsequently congestive heart failure ensues.\textsuperscript{74}

Toxic exposures

Toxic exposure is an important part of the differential and etiology of congestive heart failure. This section discusses various toxic conditions for the health team to consider where the heart is affected.

Alcoholic cardiomyopathy

Alcohol leads to cardiac failure through cardiomyopathy. There are studies pointing towards the fact that alcohol has a direct toxic effect on the myocardium. It is a myocardial depressant. The mechanisms through which the alcohol is thought to have toxic effects on myocardium are raised below.

Inhibition of the protein synthesis occurs. Alcohol intoxication inhibits oxidative phosphorylation and causes fatty acid ester accumulation. It decreases the immunity of the myocardial cells by causing free radical damage and disrupts the cell membrane structure.
Alcohol toxicity causes coronary vasospasm and activation of the renin angiotensinogen aldosterone system. It contributes to cardiac arrhythmias and hypertension.\textsuperscript{55} In a study on test animals, left ventricular performance became depressed, and arrythmias were observed.\textsuperscript{56}

\textit{Cocaine toxicity}

Cocaine is notorious for creating adverse effects on almost every system of the body. It has a very adverse effect on the heart. Tachydysrhythmias is the most common adverse effect. It also causes chronic accelerated atherosclerosis and myocardial infarction.

The effect of cocaine is increased levels of catecholamines circulating in the blood. The catecholamines may reach toxic levels in the blood. Cocaine inhibits the reuptake of catecholamines at the presynaptic terminals. Thus, the sustained presence of catecholamines results in the manifestation of sympathetic activity. Symptoms include tachycardia, hypertension, vasoconstriction and those related to increased myocardial oxygen consumption. The anesthetic effects of cocaine impair impulse conduction further contributing to dysrhythmias.

Long-term use of cocaine causes accumulation of catecholamines in the left ventricle resulting in an increased risk of arrhythmia by inhibiting the production of catecholamines due to negative feedback sent by accumulating catecholamines. Cocaine has also found to cause cardiomegaly. It causes myocarditis and dilated cardiomyopathy.
Cocaine has direct cardiotoxic effects as that of quinidine. It causes intraventricular conduction delay, and may result in bradycardia, hypotension, decreased contractility and dysrhythmia. Cocaine has membrane-stabilizing effects that may result in sudden asystole and, hence, cardiac arrest.

Individuals that use cocaine also are predisposed to coronary heart disease. Cocaine is found to promote atherosclerosis. It increases the production of the potent vasoconstrictor endothelin. It also further contributes to vasospasm by decreasing production of a powerful vasodilator, nitrous oxide. Thus, patients having atheromatous plaques in their hearts may experience devastating effects. Cocaine in many ways damages the heart function and leads to heart failure.57

**Toxic drug induced myocarditis**

Inflammation of the myocardium may be caused by drugs that have been used as part of a medical treatment over a long period of time. It causes the death of muscle fibers, and the fibrous tissue replaces the dead tissue. Drug induced myocarditis finally results in dilated cardiomyopathy. Examples of such medications are phenothiazides and clozapine. Toxic myocarditis causes irreparable damage to the heart, and becomes irreversible. Hence, in most of the cases it leads to death due to cardiac failure.58

Myocyte dysfunction as a result of toxic drug induced myocarditis occurs through the following two mechanisms: *acute inflammatory activation* in which cytokines cause depression of the myocyte function; and, *the direct toxic effects* of the drug, which causes impairment of intra-cellular energetics. These effects are caused by abnormalities of mitochondrial
function and myocytic calcium processing.\textsuperscript{59} For example, the chemotherapeutic drugs have been found to be cardiotoxic and are potential causes for heart failure. Examples include Anthracyclines, cyclophosphamide, taxanes and 5-fluorouracil.

**Symptoms Of Congestive Heart Failure**

The symptoms of congestive heart failure and their presentation will be covered in this section. As mentioned in the prior section, CHF is a disease with characteristic features, such as dyspnea, fatigue and signs of volume overload. CHF signs and symptoms are mostly attributed to systolic or diastolic dysfunction and to ventricular enlargement.\textsuperscript{14} The New York Heart Association provides a symptom classification system of heart failure, which is briefly outlined below.\textsuperscript{3}

- **Class A:** high risk heart failure but without any structural manifestation of heart disease or symptoms;
- **Class B:** structural heart failure but without any signs or symptoms;
- **Class C:** structural heart failure with symptoms of heart failure;
- **Class D:** refractory heart failure needing specific medical interventions.

**Shortness of breath**

Dyspnea occurs commonly in cases of left ventricular failure. Shortness of breath, reflecting pulmonary congestion and fatigue, are demonstrative of a low amount of carbon dioxide. Dyspnea in heart failure generally occurs during exertion and can be relieved by taking some rest. With the progression of heart failure, dyspnea may occur both during the day or night and may even lead to nocturnal coughing in many cases.
Orthopnea often occurs with the progression of congestive heart failure; it is the shortness of breath or dyspnea occurring after lying down and can be relieved by encouraging the patient to sit straight up. Dyspnea is one of the most debilitating conditions in heart failure. A condition called paroxysmal nocturnal dyspnea occurs during the night and is relieved only when the patient sits up for at least a period 15 to 20 minutes.³

In severe cases of heart failure, Cheyne-Stokes respiration occurs which can be described as a periodic cycle of breathing. In this case hyperpnea (increased breathing) and apnea (no breathing) occurs in a cyclic pattern and can occur at any time during the day or night. This syndrome can awaken the patient during nighttime sleeping. Paroxysmal nocturnal dyspnea is associated with congestion in the lungs while Cheyne-Stokes respiration is indicative of a low amount of carbon dioxide.³,²⁰

Sleep apnea or breathing disorders, related to the worsening of heart failure, occur commonly in patients with CHF. Patients with high output heart failure also show signs of breathlessness at rest or on exertion. The typical signs of tachypnea are present in patients of high output heart failure.⁷ Hypoxemia along with a highly reduced cerebral blood flow can lead to chronic irritability and impairment of the patient’s mental performance.³,²⁰ In cases of left-sided heart failure the left ventricular dysfunction may present with dyspnea only upon exertion, pulmonary edema seen with shortness of breath, dry cough which may sometimes present with pink, frothy sputum, pallor or even cyanosis with cold clammy skin and normal or raised blood pressure.²⁹

Doppler tissue studies have been done to demonstrate the effect of increased left ventricular filling patterns but it cannot offer possible
reasons for the symptom of dyspnea in elderly patients, especially women. The factors causing dyspnea in elderly women have been cited to be related to older age, poor perception of health, anxiety and depression, impairment of daily functions, low levels of happiness or other muscular disabilities. However, there may be some other factors at play as well that are considered to be related to a more advanced stage of diastolic dysfunction, with modifications in secondary structure and hemodynamics, in addition to co morbid conditions, such as, left ventricular hypertrophy or pulmonary arterial hypertension.\textsuperscript{12} Patients with heart failure are highly prone to comorbid pulmonary complications, such as, obstructive sleep apnea.\textsuperscript{60} In fact, any adult patient that presents with dyspnea or respiratory failure should be considered for a differential diagnosis of congestive heart failure.

**Leg swelling**

Swelling of the legs along with fatigue is a commonly occurring condition in right ventricular failure. Abdominal bloating or anorexia is also seen in patients with right ventricular failure.\textsuperscript{3} There can be discomfort or pain associated with swelling because the affected liver and the abdominal congestion causes abdominal discomfort (usually in the upper right quadrant).

Peripheral edema that develops has been found to be due to hemodynamic instability in patients with decompensated heart failure. The lower extremity edema has detrimental effects on the integrity of the skin, which may eventually lead to the breakdown of skin. The different causes that are attributed to peripheral edema in patients with heart failure are: left ventricular heart failure, right ventricular heart failure, medication usage, compensatory changes to influence the hydrostatic pressure, and autoregulation in the vasculature of cardiovascular system.
Nutritional deficiency in patients with chronic heart failure is important to consider because it increases the level of albumin in the serum, which also leads to decrease in osmotic pressure and ultimately causing peripheral edema. A reduced level of albumin causes a lowering in the colloid osmotic-oncotic pressure to almost below hydrostatic pressure, which in turn causes fluids to leak from the vasculature into the interstitial spaces. Other symptoms of heart failure, such as shortness of breath, also contributes to nutritional and albumin deficiency, thus, leading to peripheral edema.61

Pleural effusion and peripheral edema have been found to be present in patients with high output heart failure.7 The cardiogenic pulmonary edema occurs because of the interplay of a variety of reasons, including dysfunction of the left ventricle, pulmonary capillary endothelium dysfunction, intravascular and osmotic/oncotic pressures and right side of the heart.16 A series of humoral and neurohumoral mechanisms (focusing on angiotensin) are triggered in congestive heart failure, which stimulate the sodium and water reabsorption by the kidneys and expansion of the extracellular fluid. These effects along with an increase in the venous capillary pressure and reduced plasma oncotic pressure lead to an extravasation of fluid and formation of edema.62

In right ventricle heart failure, the ventricle cannot pump blood into the pulmonary circulation causing the blood to go back to the right atrium, superior vena cava and peripheral tissues resulting in edema of distal peripheral tissues (below the level of the heart), including the liver and lower extremities.61 Right-sided heart failure generally presents with fatigue, pitting ankle edema, tenderness in the upper abdomen, shortness of breath, which may be due to pulmonary effusion, and a distension of the abdomen.29 In left ventricular failure, cardiac output is reduced, which
in turn triggers stimulation of the sympathetic nervous system and neurohumeral compensation, which ultimately causes edema. Activation of the renin-angiotensin system, release of aldosterone vasoconstriction of peripheral vessels and retention of sodium and water are all involved in the mechanism of causing edema and associated swelling of the legs and ankles.

The use of certain medications such as calcium channel blockers, beta-blockers, insulin, glyburide, and metformin are also associated with the development of peripheral edema. Edema leads to more complications in patients. Patients with decompensated heart failure have a decreased level of exercise tolerance; attempts to move their edematous extremities is a further contributing factor to immobility. The skin of the edematous patient is predisposed to breakdown and tears over bony structures, and pressure ulcers are commonly formed on the heels of patients with edema of their lower extremities.61

**Exercise intolerance**

Individuals with congestive heart failure exhibit a reduced ability to perform activities involving dynamic movement of large skeletal muscles owing to the underlying symptoms of heart failure, mainly dyspnea and fatigue. Heart failure or any other cardiovascular disease has a restrictive effect on the ability of the heart to increase the cardiac output during exercise. In addition, an individual’s age, sex and skeletal muscle conditioning influence the level of physical intolerance encountered during exercise.61 The inability to perform aerobic exercise has been said to be a hallmark of heart failure. This is largely due to a reduced aerobic capacity primarily as a result of impairment of cardiac output and, hence, insufficient circulation of blood to active skeletal muscle.
The patients with heart failure have been observed to achieve less than 50% of their maximum cardiac output in comparison to healthy counterparts during their performance at peak exercise. Stroke volume is also known to decrease. Exercise intolerance manifests when there is an inability to increase cardiac output with a minimal increase in stroke volume combined with a reduced heart rate during periods of low heart workload, such as occurs during mild/moderate exercise. Patients with a dilated left ventricle and a decreased left ventricular systolic function experience a rise in the stroke volume during exercise, which remains very low mainly because the left ventricular preload and ejection fraction is affected. The impaired intrinsic contractility, decreased beta-adrenergic response, and increased systemic vascular resistance all lead to a failure to increase left ventricular systolic emptying. Additionally, a reduction in the sympathetic and renin-angiotensin systems contributes to the patient’s intolerance relative to exercise.63

Pathology of the skeletal muscles has been demonstrated to be one of the major factors responsible for exercise intolerance in patients with heart failure, and a cause of physical disability for them. Different hypothesis suggest there are links between abnormalities in enzymatic and histologic functionalities and exercise tolerance. The reduction in cardiac function, which may be related to measurement of peak oxygen consumption with cardiac output, has been correlated with a decrease in exercise tolerance. It has been observed that abnormalities in the peripheral skeletal muscle function hinder changes in cardiac output and improvement in exercise tolerance.

Histologic and biochemical changes occur in the skeletal muscles in patients with cardiac heart failure contributing to exercise intolerance. Muscle fiber atrophy, changes in the capillary density, reduction in the
maximal strength of cardiac muscles and a reduction in the electromyographic activity are all linked with exercise intolerance in individuals with heart failure. Cardiac cachexia is also shown to be a cause of exercise intolerance in patients with heart failure.\textsuperscript{64}

Glycolytic fibers (specifically of skeletal muscles) are found to increase in patients with heart failure and in proportion to their ability to have oxygen supplied to their heart muscle fibers. The inability of oxygen to reach the capillary bed becomes altered, which in turn negatively affects the capacity of a patient to tolerate physical exertion. The capillary density or the endothelial cells per muscle fiber has been shown as reduced in men with congestive heart failure, and manifests itself as an inability to tolerate exertion. Changes have also been shown in the glycolytic and oxidative pathways in patients with congestive heart failure affecting their tolerance to exercise.

There is a marked change in the activity of oxidative enzymes such as succinate dehydrogenase, citrate synthetase, and beta-hydroxyacyl CoA dehydrogenase, causing an intolerance to exercise. It can be said that a collective alteration in the morphologic, histologic and enzymatic functions of the skeletal muscles affects the stamina to exercise.\textsuperscript{64} An elevation in the cytokines and tumor necrosis factor-alpha also promotes muscle wasting and forms the immunological basis of exercise intolerance in patients with congestive heart failure.\textsuperscript{64}

**Wheezing**

Shortness of breath and wheezing is the most commonly occurring symptom in patients with congestive heart failure. The accumulation of the fluid in the lungs is the main cause of the persistent cough or
wheezing that occurs. It also produces phlegm, which may sometimes be tinged with blood.65 The blood that collects in the pulmonary veins due to decreased cardiac output causes fluid to leak into the lungs. This buildup of fluid in the lungs is the reason for the persistent cough or wheezing.65

Wheezing is a continuous and adventitious sound of breath, which is produced when air flows through a narrowed airway. Wheezes are high-pitched, continuous, musical sounds (different from rales) and have a dominant frequency of 400 Hz or more.66 Congestive heart failure has been found to be associated with wheezes.

In case of left ventricular failure, there is an increase in the pulmonary pressure, which causes the serum to outflow into the pulmonary vessels and eventually into the interstitial spaces. With an increase in the pressure in the interstitial spaces, the bronchioles undergo constriction, causing the air that passes through them to produce a wheezing sound. This may also be accompanied with a leakage of fluid from pulmonary capillaries into the alveolar sacs. This condition is not favorable for an efficient exchange of oxygen into the blood and results in hypoxemia. The presence of wheezing along with jugular neck distention and hypoxemia is indicative of congestive heart failure.67

**Pulmonary crackles**

Crackles in the lungs can be described as explosive breath sounds are known as crackles, and generally associated with heart failure and pulmonary compromise. Crackles represent an abrupt opening or closing of a single airway and occur at similar inspired volumes and transpulmonary pressures during consecutive inspirations. The crackling sound in heart failure is a result of the opening of airways that have been
constricted because of peribronchial edema. The crackle in heart failure has been described as *slight crepitus mucous rattle*; such crackles in heart failure patients were studied to have medium crackles in 65% and dry rales in 88% of patients. With patient recovery and progress, crackles will disappear.\(^{68}\)

The identification of crackles in patients with congestive heart failure is an important respiratory examination leading to the diagnosis of CHF. The fluid in the small airways is the main cause of crackles (also described as rales). The discontinuous brief sound typical of crackles is heard during inspiration or expiration. Crackles can be associated with pulmonary edema in the case of heart failure when the crackle sound does not clear up, even after encouraging the patient to cough. Crackles that are audible in patients with heart failure are fine or may be coarse in nature; coarse crackles tend to be loud, of low pitch and last for a longer duration than fine crackles. When crackles are heard in asymptomatic individuals, the follow up diagnosis may lead to the presence of congestive heart failure.

Adventitious sounds associated with heart failure are distinct and rales are studied to be present in one out of four patients with left heart congestion because of the systolic heart failure.\(^{69}\) Crackles are dependent on the *timing* of the sound of inspiration and expiration, such as whether the sound is a late inspiratory crackle or an early inspiratory crackle.\(^{66}\)

Congestive heart failure is manifested by the presence of acute dyspnea with the presence of audible crackles. During auscultation, clinicians may record fine inspiratory crackles for patients with congestive heart failure although there may also be audible medium or coarse crackles heard. The crackles heard in patients with congestive heart failure occur at all times
but are mostly heard late in inspiration. It should be mentioned, however, that the crackles heard in congestive heart failure are not the specific findings of the disease. Patients with heart failure may be assessed to have an examination where an audible pitch of pulmonary crackles progressively increases during inspiration and decreases during expiration. It has been proven clinically that the coarser crackles, which are low pitched, have the tendency to occur early in inspiration while the finer, high-pitched crackles, occur late in inspiration.

Pulmonary congestion is what causes the audible sound of pulmonary crackles; and, wheezing has been reported to be dominantly present in the acute or sub-acute form of congestive heart failure. Research studies report that only 20% of patients with chronic congestive heart failure manifest pulmonary crackles.

**Cyanosis**

In left ventricular failure, cyanosis may be seen as a sign of the disease. If severe hypoxemia is present, it leads to the condition of central cyanosis, which is known to affect all of the body along with the tongue and mucous membranes. It is a result of severe hypoxemia when the circulating blood flow is reduced resulting in a lack of oxygen. Sometimes peripheral cyanosis may also occur, which is evidenced in changes in the coloration of the lips, fingers and toes in patients with heart failure.

Acute heart failure may also be associated with a reduction in the tissue perfusion at rest, and manifests with weakness, confusion, drowsiness, paleness (with peripheral cyanosis), cold clammy skin, low blood pressure, filliform pulse and oliguria. All of these symptoms are representative of cardiogenic shock.
Cyanosis has been defined as a bluish coloration of the skin due to reduction in the hemoglobin and formation of abnormal chemicals such as methemoglobin and sulphahemoglobin. It is more pronounced in the lips, nail beds and ears. Cyanosis may be one of two types: peripheral and central. The reduced cardiac output, as is seen in congestive heart failure, is responsible for cyanosis of a cardiac origin.

Cyanosis occurs when the right ventricular failure makes it difficult for the heart to pump blood. The low oxygen level in the blood is what manifests as symptomatic cyanosis. Cyanosis, of cardiac origin, develops slowly. Congestive heart failure is one of the primary causes of cyanosis. When cyanosis is accompanied with difficulty in breathing, fatigue or coughing with dark mucus, it is indicative of the presence of heart failure. Oxygenation has been cited to be a critical issue in congestive heart failure in the presence of hypoxemia, and the problem with inadequate oxygenation is that it will deteriorate the cardiac function.

Bronchoconstriction and the filling of the alveolar sacs with interstitial fluid due to an increased interstitial pressure further impairs the oxygenation of red blood cells and other vital organs leading to cyanosis. Aggressive restoration of oxygen is one of the major criteria in managing congestive heart failure.

**General symptoms of heart failure**

Some other general symptoms of heart failure include the presence of cool fingers and toes, nocturia, and reduction in daytime micturition (fainting shortly after or during urination). Light-headedness associated with posture may also be seen. Severe biventricular failure can be diagnosed in correlation with the wasting of skeletal muscle, which is
indicative of an increase in cytokine catabolism. Weight loss or cardiac cachexia is also identified with high mortality rates in CHF. Elderly patients complain of atypical symptoms, such as confusion, delirium, a tendency to fall, sudden decline in functions, sleep disturbance and urinary incontinence that occurs during the night.³

Patients with acute congestive heart failure tend to present with a clinical picture of central or peripheral cyanosis and reduced oxygen saturation.¹⁹ Additionally, in patients with CHF, there exists high output heart failure where warm peripheries (versus cold peripheries) has been shown to exist because of low systemic vascular resistance and peripheral vasodilatation.⁷

Other systemic diseases

The following sections discuss various systemic disease processes that impact heart function and can lead to congestive heart failure.

Diabetes mellitus

Diabetes mellitus is a disease in which there is a relative or absolute deficiency of insulin. In relative deficiency, there is ample insulin but the body is unable to utilize the insulin. And in absolute deficiency there is deficient production of the insulin hormone from the pancreas. Insulin helps the cells take up sugar for their metabolism. When the deficiency of insulin takes place, the cells are unable to utilize glucose. Hence the sugar remains circulating in the blood in large quantities. In diabetes, the body doesn’t make enough insulin or doesn’t use insulin properly. Over time, high blood sugar levels can damage and weaken the heart muscle and the blood vessels around the heart, which leads to heart failure.
Diabetes both predisposes the patient to heart failure and contributes to the progression of heart failure. Hyperglycemia causes an adverse effect on endothelial function and redox (free radical or oxidative stress) state. Excess circulating glucose and fatty acids affect cardiomyocyte ultrastructure, myocardial energy metabolism, vascular and myocardial compliance, thrombosis, intracellular signaling and gene expression. Diabetes accelerates the coronary atherosclerosis and cardiomyopathy. One study has suggested a characteristic pattern of concentric left ventricular hypertrophy with mildly reduced systolic performance that can develop in such cases.\textsuperscript{43}

Patients with type 2-diabetes represent 10\%-15\% of the total population of patients with acute coronary syndromes. Under normal conditions, glucose is not utilized by the myocardium for its metabolism; and, fatty acids are preferred over glucose. However, under stressful conditions both the glucose and fatty acids compete within myocardial cells to be the substrate for metabolism. Additionally, glucose is utilized through glycolysis. In type 2-diabetes glucose is not used properly, as the insulin resistance is high. The heart cells become weak and ischemia develops, which causes further deterioration of the heart’s function.\textsuperscript{44}

Coronary endothelium regulates the coronary blood flow through certain factors. In the healthy heart, these factors help the heart cope with increased demands placed upon it under stressful conditions. When hyperglycemia occurs, the endothelium becomes damaged causing impairment in the heart’s compensatory mechanism. Certain chemical changes take place and bring about the activation of endothelial protein kinase C. This protein kinase is thought to reduce the activity of the rate-limiting enzyme endothelial nitric oxide synthase in endothelial nitric
oxide synthesis and increase release of endothelin-1. Endothelin-1 is a strong vasoconstrictor.\textsuperscript{44}

Hyperglycemia also results in the production of reactive oxygen species (ROS). When produced in excess, ROS interferes with cardiomyocyte membrane transport, mitochondrial electron transport, and nuclear transcription. It leads to contractile dysfunction. These are all factors that contribute to cardiac failure.\textsuperscript{44}

\textbf{Diagnosis Of Congestive Heart Failure}

\textit{Heart failure} is described as the inability of the myocardium of the heart to deliver adequate oxygenated blood to the tissues to cope with the requirements of rest or exercise. The initial diagnostic evaluation of heart failure comprises the following four features:\textsuperscript{72}

\begin{itemize}
  \item [\textit{i})] A physical examination
  \item [\textit{ii})] A chest radiograph
  \item [\textit{iii})] An electrocardiogram
\end{itemize}

\textbf{Physical examination}

The physical examination is of prime importance in diagnosing the congestive heart failure. It comprises the steps as discussed in the sections to follow. The patient with congestive heart failure in an early stage will not show remarkable physical findings, but a careful and thorough examination will reveal key findings of congestive heart failure.
General appearance and vital signs

In mild to moderate stages of congestive heart failure the patient will not appear in any distress. The patient will feel uncomfortable after lying down in a supine position for a few minutes. Otherwise, the patient will appear fine. In later to advanced stages of heart failure the patient will likely present to the hospital emergency department in a severely dyspneic state, and be unable to lie down. The patient will feel improved when supported to sit in an upright position.

Patients typically present with a high pulse rate, more than 120 per minute (sinus tachycardia), and tachypnea. The appearance of the patient will be exhausted with labored breathing. In early heart failure the blood pressure may sharply rise due to vasoconstriction, a compensatory mechanism, in the peripheral circulation. This will lead to cold extremities and cyanosis of the nails and lips due to increased adrenergic activity. In severe cases of heart failure, the patient will appear with severe dyspnea and cyanosis. Often, there is the appearance of agitation and confusion due to cerebral hypoperfusion. Central cyanosis is often evident and suggestive of severe hypoxemia; it will affect the entire body and also normally warm areas of the body, such as the tongue and mucous membranes.

Often individuals with congestive heart failure appear puffy and have signs of non-tender pedal edema (leg and ankle) especially with right ventricular failure. Patients with advanced heart failure will present with a large belly suggestive of ascites. Swelling of the skin and soft tissue will give a person a swollen look. Patients might also have signs of icterus, cyanosis, and a malar (plum-red) flush in severe heart failure. General examination may reveal some signs of systemic illness like anemia,
hypothyroidism, sarcoidosis, alcoholism, atrial fibrillation, and hemochromatosis. In cases of severe heart failure patients might appear to have marked weight loss or cardiac cachexia. It is usually as sign of a poor prognostic outcome.\textsuperscript{3,10,75}

*Cardiac examination*

Cardiac findings are support in the diagnosis of congestive heart failure. These findings, however, do not provide important information regarding the severity of heart failure.\textsuperscript{20}

In cases of cardiomegaly, the point of maximal impulse (PMI) is displaced lateral to the midclavicular line and/or below the 5th intercostal space. PMI is palpable in two interspaces. In some cases the third heart sound (S3) is audible and palpable in the apex region. This is known as a gallop rhythm and is suggestive of severe hemodynamic compromise. The S4 heart sound is present in patients with diastolic heart failure.\textsuperscript{10,20,75} The left ventricular systolic dysfunction, sustained, diffuse and displaced, impulse is found.\textsuperscript{3} In addition to these findings murmurs for mitral and tricuspid valve regurgitation are often noted in advanced heart failure.\textsuperscript{3,20} A pan systolic murmur is found at the apex of the heart in cases of mitral stenosis or regurgitation.\textsuperscript{3}

*Pulmonary examination*

On auscultation, the most common finding on pulmonary examination is pulmonary crackles, rales and crepitation, which is due to fluid accumulation in the alveolar spaces of the lungs resulting in pulmonary edema, especially found in basilar region. In the absence of any other respiratory diseases, rales are diagnostic of heart failure. Rales may be
absent in cases of chronic HF due to increased lymphatic drainage of alveolar spaces. In addition, expiratory wheezing is known as cardiac asthma in cases of heart failure.\textsuperscript{3,20,75} On percussion, dullness is found with diminished breath sounds at the bases of the lungs.\textsuperscript{3}

\textit{Abdominal examination}

Abdominal findings are very significant in diagnosing heart failure. Hepatomegaly is a useful indicator and it is often tender and pulsatile during cardiac systole if tricuspid regurgitation is present. Accumulation of fluid in the peritoneal cavity or ascites is usually a late finding that occurs due to chronically increased pressure in the hepatic veins. Jaundice is noted in later stages of heart failure with increase in both direct and indirect bilirubin levels.\textsuperscript{3,10,20,75}

\textit{Extremity examination}

Edema in the extremity region is the cardinal sign of heart failure that is often an initial alarming sign for patients that lead them to seek care from a physician even in the absence of any significant distress. The edema is usually bilaterally symmetrical occurring mainly in the dependent areas of the body, such as, in the ankles and pre-tibial region in mobile patients. In bedridden patients, edema is found in the sacral region and scrotum,\textsuperscript{3,10,20,75} and the edema is pitting in nature and nontender. In advanced stages, generalized edema is found which is known as anasarca. Edema may seem asymmetric in some cases of CHF with patients lying predominantly on one side.\textsuperscript{3}
Jugular vein examination

Jugular vein examination is useful in estimating right atrial pressure. In the early stage of heart failure, jugular venous pressure is often recorded as normal but becomes elevated with continuous pressure on the abdomen.\(^3\) It is suggestive of positive hepatojugular reflex, which is observed with firm pressure applied over the liver during abdominal examination. On a central venous pressure manometer reading there is a prominent v wave seen that represents the jugular venous pulse in cases of tricuspid regurgitation.\(^3,75\) Systemic venous hypertension is clinically presented by distension of the jugular veins. In persons with a normal jugular venous pressure there is a decrease in pressure with respiration but in cases of heart failure the pressure increases with respiration, which is known as Kussmaul sign.\(^75\)

Left-sided versus right-sided heart failure

Points for differentiation in left-sided or right-sided heart failure are outlined below.\(^10\)

Left sided heart failure findings:

- S3 gallop
- Cardiac murmur (Aortic stenosis, mitral regurgitation or aortic regurgitation)
- Paradoxic splitting of S2
- Pulmonary rales
- Tachypnea

Right-sided heart failure findings:

- Peripheral edema
- Ascites
- Hepatomegaly
- Hepatojugular reflex
- Peri-oral and peripheral cyanosis
- Jugular venous distension

The diagnostic tests for congestive heart failure comprise electrocardiography, chest radiography, echocardiography, angiography and other procedures such as MRI (magnetic resonance imaging) and CT (ultrafast or cine computed tomography).

An electrocardiogram is recommended in all patients who are suspected to have heart failure. The most common findings of the ECG are atrial and ventricular arrhythmias. On the ECG, electrical conduction disturbance with a correlating low voltage pattern is also suggestive of the presence of amyloidosis.

Echocardiography, which provides transthoracic-two dimensional images along with Doppler flow studies, has been highly recommended in all cases of suspected heart failure. When the two-dimensional echocardiogram is not acceptable, transesophageal echocardiography is recommended which offers high quality images; however, this is an invasive technique and only used in specific cases.

**Chest X-ray**

Chest x-ray (PA view) is advised in suspected cases of congestive heart failure. There is a difference seen in the findings of right heart failure and in left heart failure.
Due to left sided heart failure, a rise in pulmonary pressure occurs. Under normal circumstances, and with an erect posture, the bases of the lungs are better perfused. Hence, the vessels supplying the lungs appear prominent. When there is a rise in pulmonary pressure, the vascularity of the apices also becomes prominent and there is dilation of the pulmonary arteries. Prominent upper lobe vessels may be seen in the x-ray film as prominent opaque lines. As the pressure rises further, interstitial edema takes place and reticular shadowing of alveolar edema may be appreciated in the x-ray film.

Subsequently, intralobular septal thickening takes place, which can be seen as horizontal lines in the costophrenic angles, also known as ‘Kerley B’ lines. Enlarged hilar vessels may be appreciated. If congestive heart failure has progressed into later stages, hazy opacifications spreading from the hilar regions may be seen. Pleural effusions may be indicated in the chest x-ray. The cardiac silhouette may be enlarged and the ratio between the cardiac width and the width of the chest in the x-ray may be more than half. It occurs due to either cardiomegaly or cardiac dilatation.

From the radiological picture, assessment of the severity of pulmonary venous hypertension (PVH) can be done. With the pulmonary capillary pressure around 13-17 mm Hg, the vessels at the apices and bases are seen as equal in terms of size (early grade I PVH). When pressure rises to 18-23 mm Hg, the vessels that lead to the lower lobes constrict further and the vessels that lead to the upper lobes dilate. Evidence of interstitial edema, with ill-defined vessels and Kerley B lines suggest grade II PVH. Lateral and posterior costophrenic angles may be blunted indicating the presence of pleural fluid. When pulmonary capillary pressure exceeds 25 mm Hg, large pleural effusions, and alveolar edema in a perihilar and lower-lobe distribution suggest grade III PVH.77
Echocardiogram and ECG

An electrocardiogram confirms the presence of heart failure. If the chest radiograph or electrocardiogram is normal, heart failure can be easily ruled out.\textsuperscript{72}

An electrocardiogram (ECG or EKG) and an echocardiogram (echo) are diagnostic tests that are done to check the heart function and detect any abnormalities in its regular functioning. Electrocardiogram or ECG is defined as a simple and painless technique intended to record the electrical activity of the heart. It gives a clear picture of how the heart works, the rate, rhythm and the strength of the heartbeat, and the timing of the heart signals as the each traverses from the top to the bottom of the heart through each compartment. The EKG is a non-invasive test in which the heart rhythm appears in the form of wavy lines that can be interpreted to evaluate the function of the heart. It is intended to check irregularities in the heartbeat, any damage that may have been caused to the tissues and the muscles of the heart, any alterations in the thickness of the walls as well as chemical or electrolyte imbalances that may be present in the heart.\textsuperscript{82}

An echocardiogram is best defined as an ultrasound or a sonogram of the heart. In this procedure, the high frequency sonic waves are utilized to take an image of the internal organs. This is also a painless and non-invasive test. It has been utilized to detect the formation of blood clots in the vessels of the heart, presence of earlier heart attacks, tumors, infections, any abnormalities in the heart valves, and the efficiency of the heart in pumping blood to the tissues.\textsuperscript{82}
The echocardiogram provides single dimensional images of the heart, which are referred to as M-mode, accurate measurement of chambers of the heart as well as two-dimensional images of the beating heart along with the chambers, valves and main blood vessels leaving from the left and the right ventricles. It is also useful in the diagnosis of the fluid present in the pericardium. The use of an echocardiogram is a helpful test for physicians to distinguish between the severities of congestive heart failure and various other conditions, such as, congenital heart diseases, blood clots, tumors, valvular infection. An abnormally high pulmonary blood pressure can also be detected with the help of an echocardiogram. No extra preparation is needed for an echocardiogram and it can be easily done at the physician’s office.

Doppler echocardiography is the detection and measurement of the direction and the velocity of the blood flow while the echocardiography is being carried out. The images of the blood flow appear on the monitor as it traverses through or in and out of the heart. Doppler examination also records a series of audible signals for the blood flow. Whereas an echocardiogram provides M-mode and 2-D echo assessing the size, thickness and movement of the different structures in the heart, the doppler echocardiogram evaluates the blood flow through the heart.

In a doppler echocardiogram procedure, sticky patches or electrodes are placed on the chest and shoulders which are in turn connected to electrodes. These electrodes are useful in recording an electrocardiogram during the echocardiography test. Following this, a colorless gel is applied on the chest and the echo transducer is used to measure recordings from various regions of the chest and to obtain different views of the heart for a thorough examination. The images can be seen on a monitor and also
recorded on a photographic paper and videotape for future use by the physician.83

In congestive heart failure, a screening echocardiography has been found to be appropriate in selected patients suspected to be at an increased risk for developing systolic dysfunction. Patients with a strong family history of cardiomyopathy or those that have exposure to cardiotoxic drugs are good candidates for echocardiography.72

A reduction in the exercise tolerance or fluid retention manifests in the patient as dyspnea or fatigue, orthopnea, rales, an increased irregular venous pressure and edema. Cardiomegaly, pulmonary edema and pleural effusion are some of the radiographic findings in cases of congestive heart failure.72 Patients who exhibit dyspnea and are suspected for the presence of heart failure need a chest radiograph and an electrocardiogram. If the chest radiograph is normal, then the probability of heart failure becomes unlikely and pulmonary causes of dyspnea can be explored.

The electrocardiogram is a sensitive way of diagnosing heart failure. As mentioned, a normal electrocardiogram makes the chances of heart failure improbable. However, if both an electrocardiogram and a chest radiograph are normal then the unlikelihood of heart failure rises by almost 95 per cent.72

The role of an electrocardiogram in diagnosing congestive heart failure is important to explain to the patient undergoing testing. The small electrodes used in the electrocardiogram are beneficial in detecting the
presence of a heart attack, the thickening of the left ventricle and the abnormality of the heart rhythm. The most common ECG abnormalities in heart failure are found to be sinus tachycardia, sinus bradycardia, atrial tachycardia or flutter or fibrillation, ventricular arrhythmias, ischemia or infarction, left ventricular hypertrophy, atrioventricular block, and certain EKG waveforms that indicate conduction abnormalities, such as Q wave and QRS length over 120 milliseconds (ms) of a left bundle branch morphology depicting electrical or mechanical dysynchrony.

The echocardiogram is definitely considered to be the diagnostic standard for the identification of both systolic and diastolic heart failure. The presence of cardiomegaly or pulmonary vascular congestion on the chest radiograph is considered to be strongly suggestive of heart failure. The presence of anterior Q waves or left bundle branch morphology on the electrocardiogram indicates the raised probability of heart failure. Hence, it is suggested that patients with dyspnea and other suggestive abnormalities on the echocardiogram should undergo two-dimensional echocardiography with Doppler flow studies.

Currently, doppler echocardiography is the most important tool for the identification of abnormal diastolic function as well as a reduction in the early diastolic filling and ventricular compliance. These are indicative of a diastolic dysfunction. The Doppler echocardiogram is used to identify and distinguish between systolic and diastolic dysfunction, identify valvular stenosis or insufficiency, cardiomyopathy or pericardial disease. The cause of the heart failure is identified by echocardiogram, but there may be a number of underlying conditions that may enhance the severity of the condition, and, hence it is necessary that thorough evaluation of patients with confirmed heart failure be performed to identify other co-existing conditions.
Echocardiography is primarily beneficial as a widely available, non-invasive technique that is used in all patients suspected to have congestive heart failure. Doppler echocardiography is highly beneficial in providing a more accurate diagnosis and more appropriate management of the cardiovascular disease. Studies have claimed that patients in whom echocardiogram was done had better chances of survival. The American College of Cardiology (ACC)/American Heart Association (AHA) and the Agency for Health Care Policy and Research (AHCPR) guidelines recommend the use of echocardiography in the management of patients with suspected congestive heart failure.

The use of echocardiography has been found to be of critical significance in the assessment of congestive heart failure. Echocardiography is also beneficial in offering clues to the etiology of diastolic heart failure by providing an assessment of the valvular function, left ventricular mass, regional wall motion abnormalities and diastolic function. Hence, the evaluation, assessment and diagnosis provided by the echocardiography acts as a guide for further work up and treatment of congestive heart failure. The echocardiogram can also effectively assess primary or secondary valvular lesions in cases of congestive heart failure. The data that is obtained from the echocardiogram may also explain the etiology of heart failure, such as, hypertrophic cardiomyopathy, pericardial disease or amyloid or primary restrictive cardiomyopathy.

The evaluation of congestive heart failure by electrocardiogram helps to determine therapy in patients suffering from advanced cases of diastolic dysfunction with complications. The information collected from the echocardiography is also useful in determining the prognosis in patients with heart failure. It gives predictive values for left ventricular size, ejection fractions, pulmonary artery systolic pressure (as a measure of
the velocity of tricuspid regurgitation), light ventricular function and diastolic function.\textsuperscript{87}

**Blood tests**

The reasons for heart failure can be listed as myocardial, valvular, pericardial or systemic diseases either singly or in combination with each other. Laboratory evaluation is performed in addition to the patient history and physical examination are carried out to provide clues about the type of heart failure, the cause of heart failure, any existing co-morbidities and to distinguish it from respiratory disease, obesity and myocardial ischemia.\textsuperscript{72} Blood tests recommended for heart failure are further explained below.

The different tests recommended for the diagnosis of heart failure and identification of its causes include blood tests intended to check for anemia, thyroid problems, high cholesterol and a blood test for B-type natriuretic peptide (BNP), which is indicative of active heart failure.\textsuperscript{92} The blood tests that are commonly carried out include the analysis of electrolytes, such as potassium and sodium, albumin and creatinine since abnormal levels are linked to heart failure.\textsuperscript{93} Patients suspected to have heart failure are required to undergo blood testing as part of a complete diagnostic evaluation; this includes a complete blood count comprising of hemoglobin, leukocytes, and platelets, serum electrolytes, serum creatinine, estimated glomerular filtration rate, glucose, and liver function tests.\textsuperscript{27}

The abnormalities in the blood or electrolyte imbalances are suggested to be uncommon in mild to moderate (untreated) heart failure; however, some level of anemia, hyponatremia, hyperkalemia and reduction in the
renal function are observed in those patients that are being treated with diuretics and ace inhibitor (ACEI)/angiotensin receptor blocker (ARB)/aldosterone antagonist therapy. The diagnostic blood tests and their monitoring are recommended to be essential during the diagnosis, initiation of the therapy, titration of the doses, and follow-up phases in patients who are receiving drug therapy for heart failure.

Common laboratory test abnormalities indicative of a health-related problem or congestive heart failure are discussed below:

1. An increase in the serum creatinine levels is indicative of renal disease ACEI/ARB, aldosterone blockade.
2. Reduced hemoglobin levels or anemia is suggestive of a chronic condition of heart failure, hemodilution, loss of iron or its inadequate utilization, renal failure or presence of a chronic disease.
3. Hyponatraemia or sodium level below 135 mmol/L is suggestive of chronic heart failure, hemodilution, non-osmotic arginine vasopressin (AVP) release, and the use of diuretics. Hypernatremia in which the sodium levels are above 150 mmol/L are indicative of hyperglycemia and dehydration.
4. Hypokalemia (potassium levels below 3.5 mmol/L) suggest secondary hyperaldosteronism. Hyperkalemia (potassium levels over 5.5 mmol/L) show the presence of renal failure or the use of potassium supplements, and renin-angiotensin-aldosterone system blockers.
5. An increased level of brain natriuretic peptide (BNP) above 400 pg/ml is suggestive of increased ventricular wall stress while brain natriuretic peptide found to be below 100 pg/ml suggests normal wall stress.
6. High levels of albumin are indicative of dehydration and myeloma while low levels of albumin suggest poor nutrition as well as renal loss.

7. An increase in the transaminase levels in the blood depicts liver dysfunction, right heart failure or drug toxicity.

8. Elevated levels of troponin found in the blood indicate myocyte necrosis, prolonged ischemia, severe heart failure, myocarditis, sepsis, renal failure and pulmonary embolism.

Brain natriuretic peptide

The measurement of plasma levels of the brain natriuretic peptide has proven to be a useful and cost effective screening tool for the diagnosis of heart failure. It also brings down the need for taking up other expensive cardiac tests to diagnose it. Brain natriuretic peptide has been studied in the pathophysiology of congestive heart failure and has been identified to be a part of the adaptive response (hemodynamic changes) that occurs as a result of congestive heart failure.

The levels of brain natriuretic peptide circulating in the blood are increased to a considerable level in patients with chronic heart failure. Also, in patients suffering with left ventricular dysfunction there is also an increased level of brain natriuretic peptide found. Owing to these reasons, the plasma brain natriuretic peptide levels are measured as a means of clinical screening. These values also have a prognostic significance added to their diagnostic benefits.94

The determination of levels of natriuretic peptides and its usefulness as a biomarker in the diagnosis of heart failure as well as the management of chronic heart failure has been established through various research
studies. Levels of natriuretic peptides have been useful for the monitoring and adjusting of the drug treatment profile in patients, as well as in the staging of heart failure and in making treatment and discharge decisions.

Natriuretic peptide testing has been helpful in the identification of patients who are at risk for clinical events. If the concentration of natriuretic peptide is found to be high despite the optimal treatment provided, then it is suggestive of a poor prognosis.95

The increase in the level of the B-type natriuretic peptide occurs as a result of a response to the myocardial wall stress. Those patients that exhibit optimal left ventricular systolic function exhibit low levels of natriuretic peptide. There are various other conditions which are also related with an elevation in the level of the natriuretic peptide and these are left ventricular hypertrophy, tachycardia, right ventricular overload, myocardial ischemia, hypoxemia, renal dysfunction, advanced age, liver cirrhosis, sepsis and infection. Decrease in the natriuretic peptide levels has been found to occur in cases of obesity. It is useful in the treatment of heart failure.95

Natriuretic peptides are useful for the body in responding to heart failure by reducing the pressure on the lungs and increasing the flow of urine. The new blood tests for the diagnosis of heart failure are done for two kinds of natriuretic peptides: B-type natriuretic peptide and pro-BNP (N-terminal pro B-type natriuretic peptide). Patients with congestive heart failure have elevated levels of both of these natriuretic peptides. The measurements of these two types of peptides are used to distinguish between patients suffering with congestive heart failure and pulmonary problems.
Patients with normal levels of natriuretic are generally ruled out for the presence of heart failure. When the tests for the natriuretic peptides are taken over a period of few days, the results help to guide the medical therapy for the treatment of the heart failure. The changes in the levels of the two natriuretic peptides occur rapidly with respect to the degree of congestive heart failure present in a patient.96

**Troponins**

Troponin I or Troponin T is determined in patients suspected to have heart failure if there is evidence of an acute coronary syndrome. The increased level of cardiac troponins is indicative of myocyte necrosis. It may also suggest the need for the potential revascularization and an appropriate diagnostic work up. Another case where increased levels of troponins may be seen is acute myocarditis. There is mild incremental change in the levels of troponin in cases of severe heart failure and decompensation in patients. It is seen in patients where there has not been any evidence of myocardial ischemia because of acute coronary syndrome or a condition of sepsis. When present in high levels along with natriuretic peptide, troponin acts as a strong prognostic marker in heart failure.97

**Neurohormonal markers**

An increase in the levels of various neurohormonal markers such as norepinephrine, renin, aldosterone, endothelin, arginine vasopressin is also seen in cases of heart failure. The evaluation of the stimulation of neuroendocrine chemicals has been cited to be useful for research purposes even though it lacks any diagnostic or prognostic value.27
It is also known that atherosclerosis is one of the major causes of heart attacks and heart failure. Measurement of the levels of the total cholesterol along with low-density lipids, high-density lipids and triglycerides are important for assessing the risk factor. Added to this, the measurement of C-reactive protein is also significant since it is a marker for inflammation, which forms one of the components of atherosclerosis. Patients exhibiting increased levels of C-reactive protein are indicative of an increased cardiac risk.  

In the diagnosis of acute heart failure the blood tests that have been suggested above are inclusive of an initial evaluation with full blood count, measurement of sodium, potassium, urea, creatinine, glucose, albumin, hepatic enzymes and INR. Additionally, the prognosis of acute heart failure is shown by a reduced amount of sodium and increased urea and creatinine serum level in the blood. An increase in the cardiac troponin is also seen in patients with acute heart failure along with acute coronary syndrome.  

**Treatment**

The treatment of congestive heart failure is multifactorial and needs to be holistic in nature. This section covers the treatment plan and the related recommended lifestyle adjustments for patients with CHF.  

**Lifestyle changes**

Congestive heart failure is a lifelong condition that involves the application of a combination of treatments comprising of lifestyle changes, medication, devices or surgery, all of which work together to enhance the efficiency of the heart’s function. Simple and basic lifestyle changes made in the daily routine go a long way in managing heart failure and reducing
the risk of further complications. Cessation of smoking, healthy food habits and regular exercise are three key measures that will help the patient in efficiently managing congestive heart failure. Self-care can easily form the cornerstone of heart failure management and treatment. It has a definite effect on the patient’s symptoms, functional ability and sense of wellbeing, morbidity and prognosis of the disease. The aim of managing self-care is to maintain and enhance physical stability, avoid the behavior that may lead to the progression of the CHF and identify the early signs or worsening of the disease.

Health care education and counseling provided to the patients and their families by health care professionals play a major role in determining the promotion of self-management of the disease. The patient should be well informed about the etiology, signs, symptoms of heart failure, understanding the pharmacological treatment, modification of the risk factors, and recommendations for diet such as sodium restriction, for daily exercise, sexual activity, immunization, sleep and breathing disorders, and, in addition, of the importance of adherence to treatment protocols and of the psychosocial aspect of treatment as well as the prognosis of the treatment.

It is important that the health provider counsels the patient about the serious implications of heart failure and the prognosis of the disease, but they should also stress to the patient that he/she can continue to lead an active life and enjoy a reasonable quality of life. The patient should be counseled and educated about the outcomes of their disease, and informed about dietary restrictions, restrictions on alcohol and the significance of physical activity to reduce the hospitalization frequency due to congestive heart failure.
**Diet and nutrition**

Lifestyle factors comprising a healthy diet are an important means of successfully managing congestive heart failure. One of the key factors in reducing the risk and further worsening of the condition of heart failure is a healthy diet. The benefits of a healthy diet extracted from the current literature are discussed below. Incorporating a healthy diet as well as a healthy lifestyle can significantly cut down the risk of the following:

1. Heart diseases, heart attack and stroke;
2. Conditions leading to heart dysfunctions such as cholesterol, obesity, and high blood pressure;
3. Other diseases that may be chronic in nature such as type-2 diabetes, osteoporosis and some forms of cancer.

A healthy diet is important because circulating free fatty acid and glucose levels often exceed the normal range and chronic exposure of the heart to high concentration of free fatty acids circulating in the plasma may cause accumulation of the toxic lipid intermediates in the cardiomyocytes. Also, glucose and fatty acid oxidation can also give rise to the formation of harmful products, which can trigger events ultimately causing the dysfunction or death of the myocyte.

**Dietary sodium restriction**

All heart failure treatment guidelines suggest dietary sodium restriction as essential self-care behavior. This is due to the fact that sodium homeostasis physiology is changed in heart failure when compared with a normal population. An excessive sodium intake may lead to fluid retention, hence sodium restriction is recommended in all guidelines for heart failure management. The American College of Cardiology and American Heart Association has recommended 3,000 mg to 4,000 mg of
daily sodium intake\textsuperscript{146} while patients with volume overload should be restricted to 2,000 mg/d.\textsuperscript{147}

Restriction of sodium intake in the diet is associated with neurohormonal activation in heart failure. Animal studies have proven that diet restricted in sodium results in a reduction in the cardiac output and an increase in the vascular resistance because of the activation of the renin angiotensin aldosterone system.\textsuperscript{145} Sodium intake affects blood pressure control, the incidence of hypertension, left ventricular hypertrophy, cardiovascular disease and other risk factors associated with heart failure or the incidence of heart failure itself.\textsuperscript{145} To control sodium in the diet, alternate food seasoning options to satisfy taste preferences while reducing the elements that may be detrimental to cardiac health should be followed.\textsuperscript{145} Other recommended diet plans for individuals with congestive heart failure is further outlined below.\textsuperscript{143}

\textit{Fruits and vegetables}

Fruits and vegetables should be taken daily as part of the healthy diet. Fruits and vegetables are rich sources of fiber, vitamins and minerals. They are low sources of fat, calories, sodium and cholesterol. At least 5 servings of fruits and vegetables should be taken daily.

\textit{Grains}

Low fat breads, cereals, crackers, rice and pasta should be taken as a regular part of the diet. Vegetables rich in starch such as peas, potatoes, corn, winter squash and lima beans are also rich sources of B vitamins, iron and fiber. These foods are low in fat and cholesterol and hence good for patients with heart failure.
At least 6 or more servings should be taken daily. Grains and grain products are rich sources of fiber, vitamins, minerals and complex carbohydrates.

*Proteins*

Foods such as meat, poultry, seafood, dried peas, lentils, nuts and eggs have high B vitamins, iron and other vitamin and mineral content. At least 2 servings of fish should be taken weekly. Skinless turkey, chicken, fish or lean red meat should be used in the diet to keep the amount of saturated fats to a low level in the diet. Milk and dairy products are rich sources of protein, calcium, B vitamins-niacin and riboflavin and Vitamins A and D. Low-fat or non-fat cheese, yogurt or buttermilk should be used if dairy is consumed.

*Fats, oil and cholesterol*

The presence of high amounts of saturated fat allows the cholesterol to accumulate in the arteries thereby clogging or blocking the arteries, which increases the risk of heart attack, stroke and other cardiovascular problems. Food rich in saturated fats should not be used. Total fat intake should be limited to 25 to 35 % of total daily calories and saturated fat should be restricted to 10% of total daily calories.

Trans fatty acids are unhealthy which are formed following the hardening of vegetable oils. Trans fat foods can not only raise the LDL cholesterol, but also raise the HDL cholesterol. For this reason, fried foods, commercial baked food products, processed food and hard margarines should be avoided because they are rich in trans fatty acids.
**Herbs and spices**

Herbs or spices that contain sodium should be avoided. Lemon juice or fresh ground pepper should be used as seasoning on foods to enhance the flavor.

**Other recommendations**

While buying packaged foods, some of the information provided on the packaging should be properly read and followed. These include lists of nutrients, % daily value of food, daily value footnote, calories per gram and sodium content of the food.\(^{148}\)

Lean meat and poultry without skin should be taken and prepared without adding the saturated or trans-fat. Foods that contain partially hydrogenated vegetable oils should be avoided to reduce the trans-fat in the diet. Beverages and foods that have added sugar in them should be avoided. Select foods with little or no salt and also prepare food by using as little salt as possible. Alcohol should be taken in moderation (discussed in a later section).

All the patients who are symptomatic for congestive heart failure should be given recommendations for incorporating healthy and balanced food in their diet. In an attempt to prevent the retention of fluids the intake of sodium should be restricted in patients suffering from congestive heart failure. Patients should be provided a list of food to avoid that contains large quantities of salt.\(^{27}\) On the other hand, patients suffering from hyponatremia should limit their fluid intake up to 1.5 to 2 L/day; although, patients showing mild to moderate symptoms of congestive
heart failure are not expected to show any significant clinical benefits with the use of restriction in the fluid intake.\textsuperscript{27}

One of the most important things to consider is to maintain a healthy diet as part of the management of heart failure. The food that is eaten can have a considerable effect on controlling the obvious risk factors such as cholesterol, blood pressure, diabetes and obesity or being overweight. The diet should comprise a large quantity of nutrients inclusive of vitamins, whole fruits, whole grain and high fiber foods, fish, lean protein and fat free or low fat dairy products should be eaten regularly.\textsuperscript{101} Also, the food and the calories that are being consumed should be coordinated with regular physical activity to ensure appropriate utilization of calories by the patient.\textsuperscript{101}

**Weight control and monitoring**

Patients with congestive heart failure should be made aware that an increase in their body weight is related to a worsening of heart failure and fluid retention.\textsuperscript{102} Health providers should educate patients on ways to incorporate weight measurement into their daily routine. If a sudden weight increase of 2 Kg or more is seen in a period of 3 days, consideration should be given to an increase in the diuretic dose and the patient should be instructed to notify their health provider should this weight increase occur. The use of diuretics in such cases may also lead to volume depletion and so patients should be educated on that risk.\textsuperscript{27}

Good nutrition, controlled and balanced intake of calories and regular physical activity should be used as methods of managing obesity. Obesity leads to a number of risk factors such as high cholesterol, high blood pressure and insulin resistance, all of which work towards worsening the
condition of heart failure or other cardiovascular conditions. Patients with body mass index more than 30 Kg/m2 are considered to be obese, and it is recommended that efforts for weight reduction be considered to prevent further progression or worsening of heart failure, reduce heart failure symptoms and also improve the overall wellbeing of the patient.

**Cholesterol management**

Cholesterol deposits in the arteries is detrimental for cardiovascular health and can trigger a heart attack or stroke over time. To control the cholesterol and keep its levels down to optimal levels, a diet which is low in saturated fat, trans fat and cholesterol is recommended along with exercise. If the cholesterol values are not reduced through diet and exercise then medications may be needed.

The total cholesterol should be less than 200 mg/dL. Low-density lipid cholesterol which is responsible for causing damage should be below 100 mg/dL in patients that are at low risk of heart failure, below 130 mg/dL in those at an intermediate risk for heart failure and less than 100 mg/dL in those already with heart failure or diabetes. High-density lipid cholesterol is good for the body and should be 40 mg/dL or more in men and 50 mg/dL or more in women. The triglycerides should be less than 150 mg/dL.

**Smoking cessation**

Tobacco intake of any form both active and passive should be avoided. Smoking has been suggested to be a risk factor for many cardiovascular diseases. Some studies have suggested that complete cessation of smoking in patients with heart failure is related to a marked reduction in
the morbidity and mortality in patients. The patient should be given proper advice, support and motivation to stop smoking.

**Exercise and physical activity**

Patients suffering from heart failure are generally inactive and physical inactivity in turn leads to further progression of the disease. It is recommended that patients go for regular resistance or endurance physical training, which is initially done under supervision. This is found to be helpful in improving autonomic control by increasing the vagal tone and in bringing down the stimulation of symptomatic nervous system, and improving muscle strength, the ability to achieve vasodilatation, and endothelial dysfunction and reduced oxidative stress.

Exercise training to improve physical activity has been known to be highly beneficial in reducing the mortality and hospitalization in patients of heart failure. It has been suggested that cardiac rehabilitation programs form an effective treatment option for patients with heart failure; and, it has been recommended that all patients with heart failure undergo moderate daily activity.

All stable patients with chronic heart failure should be recommended to an exercise trainer. Exercise training can be given to all patients of heart failure. Regular exercise or an exercise program can be taken up at home, as outpatients as well as for patients that are hospitalized. Research evidence suggests that at least 30 minutes of daily physical activity on at least 5 days of the week cannot only help in reducing blood pressure, reduce cholesterol but also maintain health at optimal levels.
Alcohol intake

Alcohol is studied to have a negative inotropic effect and can be associated with an increase in the blood pressure and an increased risk of arrhythmias. Excessive use of alcohol is known to be detrimental to the health of individuals with heart failure. Those who are suspected of cardiomyopathy caused by an excessive use of alcohol should completely avoid alcohol. The recommendations on the limitation of alcohol are suggested to be up to 10 to 20 g/day of alcohol.\textsuperscript{27}

Sexual activity

There are various sexual problems associated with cardiovascular disease, medical treatment, and the psychological factors associated with cardiovascular disease include symptoms of fatigue and depression, which have been found in patients with congestive heart failure. Sexual activity has been found to influence the clinical status of patients presenting with mild to moderate symptoms. An increased risk of decompensation may also be triggered by sexual activity in patients with the NYHA class III-IV (classification system described earlier).

Patients should be educated on the recommendation to use sublingual nitroglycerine as a prophylactic treatment to prevent dyspnea or chest pain during sexual activity. Individual counseling is advocated for both male and female patients and their partners about sexual activity in the setting of congestive heart failure.\textsuperscript{107}

Travelling

Patients with symptoms of heart failure should avoid going to places with a high altitude, for example, above 1,500 miles or to places that are very
hot and humid. Air travel is preferable as compared with long journeys by some other means of transport. Also, the patient should discuss travel plans with their heart failure medical care team.27

**Blood pressure maintenance**

Patients with pre-hypertension should aim to reduce blood pressure to normal levels. The factors affecting the blood pressure are diet, physical inactivity, toxins, and psychosocial factors. All these factors should be modified to healthy levels to achieve optimum levels of blood pressure. The dietary factors raised earlier have been suggested to play a predominant role in keeping the blood pressure under check. Salt intake should be reduced, calorie deficit should be initiated to lose weight, consumption of alcohol should be reduced and potassium intake should be increased in CHF patients. Carbohydrates should be replaced with plant proteins or monosaturated fats to help in blood pressure reduction.107

**Blood glucose level**

Hyperglycemia or insulin resistance may lead to many cardiovascular complications comprising of congestive heart failure, stroke, peripheral vascular disease, cardiomyopathy and heart failure. Reducing calorie intake and increasing physical activity will help to improve insulin resistance and glucose control.108

**Immunization**

Patients with heart failure who have not shown any contraindications should be vaccinated for pneumococcal and annual influenza.27
**Sleep disorders**

Patients with heart failure are often found to experience sleep related breathing disorders, and a correction is needed to reduce the morbidity and mortality associated with it. Weight loss in clinically obese individuals, discontinuation of smoking, completely avoiding alcohol can help to reduce the risk of breathlessness. Added to this, a treatment with continuous airway pressure may also be considered to reduce the risks associated with sleep disorders.\(^{27}\)

**Angiography**

Coronary angiography is defined as the process by which X-ray images of the arteries of the heart (coronary arteries) are taken. The images obtained through coronary angiography are also known as a coronary angiogram.\(^{109}\)

Coronary angiography is obtained with the use of a parenteral form of radiopaque contrast media to aid in visualizing the coronary vessels. The procedure is invasive in nature and is generally performed with the help of specialized intravascular catheters. This procedure is carried out as one of the components of cardiac catheterization, which also includes angiography of other different vascular structures including the aorta and left ventricle.\(^{110}\)

Invasive coronary angiography has been cited to be the gold standard for the analysis and evaluation of the coronary arteries, and for the diagnosis of luminal stenosis. It is often recommended for patients suffering with heart failure to evaluate the presence or absence of coronary artery disease.\(^{111}\)
Rationale for the use of angiography

Coronary angiography is required to outline the coronary anatomy and the quantification of the angle of the luminal obstruction of the coronary arteries. The use of coronary angiography helps to find the presence and the extent of obstructive coronary artery disease. It is also used to evaluate and analyze the ability and the accuracy of different forms of the therapy, including revascularization by percutaneous or surgical interventions. When the assessment and analysis of heart failure or coronary disease becomes difficult through the use of non-invasive techniques, angiography is carried out. An angiogram is always performed after non-invasive diagnostic tests or a stress test to diagnose heart failure have been performed. This is because of the invasive nature of the test and some amount of risk associated with angiography.

Coronary angiography has been found to be useful to help in the diagnosis of heart conditions, devise a treatment strategy and medical procedures. It is mostly recommended in the case of a heart attack, angina and when there is the need to plan interventional or surgical procedures. Coronary angiography is preferred in cases of patients with heart failure who have a prior history of exertional angina or suspected ischemic left ventricular dysfunction after an event of cardiac arrest or those with a strong risk factor profile for coronary heart disease. It is also considered in severe cases of heart failure with shock or acute pulmonary edema or for those not showing an appropriate response to CHF treatment.

Coronary angiography and left ventricular ventriculography is suggested to be carried out in patients with an unknown etiology of heart disease or in patients exhibiting signs of severe mitral regurgitation or aortic valve
disease that may be corrected by surgery.\textsuperscript{27} It is strongly recommended for patients that present with systolic left ventricular dysfunction, and are suspected of hibernating myocardium after the non-invasive diagnostic tests have been done. Angiography may also be needed in those patients who otherwise have normal systolic function but an undefined acute case of pulmonary edema. In this case angiography is highly recommended to exclude any ischemic systolic and/or diastolic dysfunction\textsuperscript{110} and coronary heart disease, and to detect the blockade or the build-up of the fatty substance in the coronary arteries.\textsuperscript{109}

Coronary angiography is also strongly recommended in the following main conditions in patients diagnosed with congestive heart failure where the use of angiography has proven helpful to diagnose and treat.\textsuperscript{110}

1. Congestive heart failure because of the systolic dysfunction with angina or with abnormalities in the motion of the regional wall or in the presence of evidence for myocardial ischemia;
2. Before cardiac transplants;
3. When congestive heart failure occurs secondary to other conditions such as post infarction aneurysm or other mechanical complications of myocardial infarction;
4. When the cause of systolic dysfunction cannot be identified with the use of various non-invasive techniques;
5. Where the episodes of heart failure increase even though the systolic function is normal;
6. Congestive heart failure with previous coronary angiograms depicting normal coronary arteries and lack of any evidence to show the presence of ischemic heart disease.
The procedure of angiography

The catheter has to be inserted during the angiography into the blood vessel in the arms (at the inside of the elbow or near wrist) or the groin of the patient. X-rays are used as a guide to direct the tip of the catheter up to the heart and the coronary arteries to obtain images. Radio contrast dye is used as medium to visualize the images or the angiograms. The patient is given local anesthetic when the procedure is being done. The entire procedure usually takes about 30 to 60 minutes. The X-ray image obtained after the procedure is a detailed depiction of the state of the heart and the coronary arteries.

The catheter is moved through the aorta to the coronary arteries in the heart and is monitored through X-rays on a monitor. There is typically no pain or other sensations during the procedure. With the catheter inserted in place, a small amount of radio contrast dye is injected into the catheter, which travels through the coronary arteries. The use of different catheters for different arteries is needed during the procedure. One is removed before inserting the other catheter in the same place of insertion. The dye is needed in a larger quantity for the examination of the heart muscle.

Risks with angiography

Angiography calls for cardiac catheterization, which is avoided if unnecessary during routine diagnostic tests in the management of patients with heart failure. The use of such an invasive procedure is only made when elucidation of etiology, understanding of prognostic information or revascularization is needed.
Invasive coronary angiography is a costly procedure and has been found to be associated with some inherent risks such as death (0.12%), myocardial infarction (<0.05%), stroke (0.1%) and bleeding (1.6%) which has restricted its routine use in all patients. The use of angiography requires the patient to be in a prone position during the exam. The angiography requires the injection of a notable amount of contrast media that enhances the loading conditions and also has the ability of impairing the patient’s renal function. There is a slight risk with angiography because of the involvement of cardiac catheterization. The risks involved are very rare in nature and can be enumerated as follows:

- Cardiac tamponade
- Irregular heart beats
- Injury to an artery in the heart
- Reduction in the blood pressure
- Allergic reaction to the contrast dye used in the procedure
- Stroke
- Heart attack

There are some procedural risks also related with angiography such as those listed below:

- A risk of bleeding, infection or pain at the IV site.
- A small risk of damage caused to the blood vessels by the use of small plastic catheters
- Formation of blood clots on the catheter which may be left at some other place in the body
- The contrast dye used in the procedure may sometimes damage the kidneys. This effect is more pronounced in patients with diabetes.
**Benefits of angiography**

It’s important for providers to educate patients about the benefits as well as the risks of angiography. It has been observed that patients hospitalized for acute heart failure syndromes also carry a diagnosis of coronary artery disease, and the use of coronary angiography in these cases greatly influence the therapeutic decision-making and early post discharge outcomes in patients suffering with acute heart failure syndromes. Research studies have demonstrated that the angiography carried out in the hospital on patients with acute heart failure syndrome and coronary artery disease led to an increase in the use of aspirin, statins, β-blockers, angiotensin-converting enzymes (ACE) inhibitors and myocardial revascularization. The use of angiography in patients with heart failure has also led to a substantial reduction in death, re-hospitalization and death or re-hospitalization within 60 to 90 days post discharge.116

In clinical practice, the need and the use for angiography is determined by the indicators depicting the need for revascularization as well as a risk profile. High-risk patients such as older patients or those with cardiogenic shock gain more benefit from revascularization. Patients in whom left ventricular dysfunction is present are known to benefit more from revascularization when compared with those exhibiting normal function. Once the angiography is done on patients, it is the condition of the coronary artery that determines whether revascularization is performed and the choice of procedure for treatment.117 Research studies have clearly mentioned angiography to be the definitive procedure for the identification of the coronary anatomy. For this reason, it is considered to be essential form of investigation in the case of heart failure.118
Coronary angiography performed as a routine measure in patients receiving heart transplants is known to detect focal stenosis and diffuse tapering in half of the recipients within 5 years of the transplantation. This is beneficial in enabling early detection of transplant vasculopathy, which assists physicians in taking life saving measures comprised of early surgical, percutaneous and medical therapies. Some of these therapies include bypass surgery, re-transplantation, balloon angioplasty and coronary stenting. Thus, coronary angiography presents clinicians with both diagnostic and therapeutic opportunities. Physicians rely on previous angiograms to assess the risk status of the patients with angina or equivalents of angina.

Angiography cannot be considered perfect due to the fact that the normal or non-diseased coronary segments on an angiogram may show abnormalities on intra-vascular ultrasound. Sometimes, the left main coronary artery appears to be normal in an angiogram even when the disease is actually present. The documentation of coronary heart disease supports more aggressive medical therapies for the prevention of secondary symptoms.

**Post-angiography**

The patient can leave the hospital the same day as the coronary angiography was performed following a period of rest and observation. There may be some tiredness following the angiography. The site of insertion may remain tender for a day or so. Some bruising may also be observed for some time.

Patients are recommended not to indulge in certain activities such as bathing, driving or lifting heavy objects for one or two days after the
angiography procedure. In cases of excessive bleeding or circulation problems in the limbs, the patient should be advised to return to the hospital.\textsuperscript{109}

\textbf{Implanted devices}

Patients with chronic heart failure also suffer from cardiac dyssynchrony. In these cases the interventricular or intraventricular electrical stimulation is delayed and leads to a significant amount of abnormalities in the overall and segmental right and left ventricular activation as well as impaired mechanical performance. The new methods of enhancing cardiac stimulation, which can synchronize and normalize the ventricular function, improve the overall performance of the heart and also increase exercise capacity consist of implanted devices, such as the pacemaker and implantable cardioverter defibrillator.\textsuperscript{121}

\textit{Implantable cardioverter-defibrillator}

Implantable cardioverter-defibrillator is used in the treatment of patients of heart failure. Implantable cardioverter-defibrillator devices are devices that can be implanted in a patient through a procedure that lasts for less than an hour. It is a small device that serves many functions including atrial and ventricular defibrillation, antitachycardia pacing (ATP), backup bradycardia pacing, electrogram storage, and biventricular pacing.\textsuperscript{122}

The implantable cardioverter-defibrillator (ICD) is composed of different components that include batteries, capacitors, a microprocessor and a header containing a site to connect leads. The most significant functions of an ICD are to detect tachyarrhythmia, distinction of tachycardia as a form of treatable arrhythmia, delivering therapy, monitoring of heart
rhythm following treatment and the storage of diagnostic results. Therapy is started following the detection of arrhythmia.\textsuperscript{122}

Implantable cardioverter-defibrillator therapy is known to be effective for the treatment of atrial fibrillation. Currently used dual chamber ICDs are used for atrial ATP and defibrillation capabilities. ICD are effective in stopping arrhythmias as soon as they start and hence its progression to atrial fibrillation is prevented.\textsuperscript{44} ICDs are mostly beneficial in people who are known to be at high risk of having a ventricular tachycardia or ventricular fibrillation. ICD shocks are painful in nature, which also results in limitation in the usage of the ICD.\textsuperscript{122}

\textit{Ventricular assist devices}

A mechanical device, ventricular assist device is used to support the failing heart. The primary function of the ventricular assist device is to aid the failing heart and optimize the blood flow to the vital organs in the body. The U.S. Food and Drug Administration has approved the ventricular assist device in providing permanent or a lifetime support to the patients witnessing end-stage heart failure.\textsuperscript{123}

There are two types of ventricular assist devices, permanent and temporary. The temporary ventricular assist devices are placed to offer short-term support. While the temporary ventricular assist device is implanted, other existing medical problems such as infections or kidney failure can be treated and a better outcome for the patient can be achieved.\textsuperscript{123}
Ventricular assist devices are placed surgically by incising the chest after the patient has been placed on a heart-lung bypass machine. The ventricular assist devices are composed of three different parts:

1. The inflow cannula:
   A large tube that allows for the flow of blood from the heart into the pump;
2. The outflow cannula:
   A large tube returns blood to aorta (when a left ventricular device is used) or pulmonary artery (when a right ventricular assist device is used);
3. The pump:
   It is connected to the heart and aorta through an inflow and outflow cannula.

In some patients there is a requirement for the biventricular assist device or a total artificial heart to be a bridge for coronary transplantation.

The ventricular assist devices that are currently used today are implanted just below the diaphragm in the abdomen or just outside the body on top of the abdomen. If the pump used is smaller it can be implanted adjacent to the heart in the chest cavity.

Ventricular assist devices are used for the following reasons:

1. To act as a pathway to recovery;
2. To be used before the transplantation;
3. To make way for the targeted therapy and outcome.
The ventricular assist devices are used to provide temporary support to the patients while the heart recovers from acute heart failure and then the ventricular assist device is removed. It is also used in patients who are eligible for heart transplant but are waiting for the donor organ. Patients over the age of 70 years who cannot get heart transplant or have other chronic medical problems are given a permanent ventricular assist device to treat end stage heart failure. Ventricular assist devices are also given to the patients who need a supportive period for assessing and evaluating the functions of the vital organs, nutrition and strength so that the heart transplant is done when the patient has improved.

During the hospitalization for heart failure, the patient is made to undergo an extensive medical and psychosocial evaluation to make a decision about the final treatment. Evaluations such as cardiovascular testing, an echocardiogram, stress test, heart catheterization and tests of the functions of other vital organs, are done before the ventricular assist device can be implanted. It is important to educate the patients and their families regarding the use of the ventricular assist device. It should also be understood that the use of the ventricular assist device is aimed at improving the quality of life of patients with congestive heart failure.

Ventricular assist devices are aimed at providing lifesaving therapy and improving the quality of life. There has been positive outcomes with use of the ventricular assist devices where patients have undergone successful heart transplant and been weaned off the ventricular assist device. Observational studies have reported that with the use of the ventricular assist device, patients have shown a significant improvement in their quality of life. Patients have been noted to be able to engage in regular activities such as exercise, travel and sex. Studies have recommended that use of ventricular assist devices be considered for long
term use and to prevent or reduce further clinical worsening of the patient’s condition before a transplant is done as well as reduce the mortality in patients suffering from severe acute myocarditis.\textsuperscript{27}

The patients with a ventricular assist device have to take care of the equipment. This includes cleaning and checking the device, changing the driveline with the help of a sterile technique, monitoring for the presence of any infection, measuring of vital signs, and ensuring the proper functioning of batteries.\textsuperscript{123} Some drawbacks of ventricular assist devices consist of bleeding, infection, stroke or a malfunction of the device,\textsuperscript{123} and patients should be educated as well to observe for and report these issues if they occur.

\textit{Cardiac Pacemaker}

Cardiac pacing has been used as an adjunctive therapy in the treatment of patients suffering with mild to severe congestive heart failure.\textsuperscript{124} The need for a pacemaker arises when the rate of the heartbeat is too slow, and the pacemaker functions to monitor the heartbeat continuously. It is a small device that is implanted in the chest or the abdomen to help to maintain an optimal heart rhythm. The pacemaker is used to generate electrical pulses that stimulate the heart to beat at a regular rate.\textsuperscript{125}

Atrial sequential pacing is done either in the left or in both of the chambers to correct electrical abnormalities and provide symptomatic relief to the patient. Some research studies have suggested electrical synchronization of both the chambers; the septum and atria have been found to be beneficial in the treatment of congestive heart failure.\textsuperscript{126}
Traditionally, pacemakers have been indicated in patients with heart failure that may have normal left ventricular function. The use of dual-chamber (DDD) pacemaker has been studied to be significant in patients with heart failure and sinus rhythm to maintain an optimal chronotropic response and coordination of atrial and ventricular contraction.\textsuperscript{27} Permanent pacing, whether it is a first implant or upgrading of a conventional pacemaker in patients of heart failure with NYHA class II-IV symptoms, low amount of left ventricular ejection fraction (at or below 35%), or left ventricular dilatation, should be done with cardiac resynchronization therapy pacemaker (CRT-P) function.

Patients with NYHA III-IV class requiring a permanent pacemaker cannot go for right ventricular pacing as it may be detrimental for them and may even cause or increase the existing dyssynchrony.\textsuperscript{127} CRT-P has been recommended with the goal to reduce morbidity and mortality in those patients with NYHA III-IV class that continue to have symptoms after use of optimal medical therapy and that show a reduced left ventricular ejection fraction (LVEF less than or equal to 35%) and QRS prolongation.\textsuperscript{27} Additionally, cardiac resynchronization therapy with defibrillator function (CRT-D) has been suggested for patients with NYHA III-IV class who show symptoms even after the use of medical therapy. It is also recommended for patients with a reduced left ventricular ejection fraction (LVEF≤35%) and QRS prolongation (QRS width ≥ 120 ms).\textsuperscript{27}

The CRT-D device is preferred in patients that are expected to have a survival rate with effective functioning of the heart for more than 5 years.\textsuperscript{85} The use of CRT is not based on the echocardiogram or tissue doppler studies.\textsuperscript{127} Rather, the European Society of Cardiology guidelines have recommended CRT to be used in patients specifically known to have heart failure with electrical dyssynchrony.
The pacemaker is comprised of two parts: a generator containing the battery and the program to control the heartbeat and the leads that connect the heart to the generator and transport the electrical messages to the heart. It is implanted under the skin by making an incision on the left side of the chest below the collarbone. X-ray images are used to guide the surgeon to direct the leads through the incision into a vein and eventually to the heart. These leads are then connected to the pacemaker generator.\textsuperscript{128} Biventricular pacing is implanted by entering the cardiac veins through the coronary sinus by achieving a reasonable threshold in one of the cardiac veins.\textsuperscript{129} Cardiac resynchronization therapy is implanted at three different levels, outlined below:\textsuperscript{129}

1. Atrioventricular level
2. Intraventricular level
3. Interventricular level

The CRT is done by pacing the right atrium, right ventricle, close to the interventricular septum and pacing the left ventricle via the coronary venous branches. This is also known as biventricular pacing.\textsuperscript{129} Some possible risks associated with the use of the pacemaker are production of abnormal heart rhythms, bleeding, infection at the site of insertion or puncture of the heart, which can cause bleeding around the heart, or a punctured lung. A punctured lung or heart is very rare risk associated with pacemaker implantation.\textsuperscript{130}

Various research studies have shown that pacemakers are associated with a wide range of patient benefits. It has been found to improve the functional status of the patient by improving their stamina for exercise duration, quality of life, and reduction in the overall mortality and hospitalization of patients for all cardiovascular events.\textsuperscript{131} The use of CRT
is also associated with a marked reduction in the levels of N-terminal pro-brain natriuretic peptide (NT-pro BNP), a diagnostic screening tool to identify left ventricular systolic dysfunction and that is linked to better patient outcomes.\textsuperscript{132}

**Heart transplantation**

Heart transplantation is the treatment that is intended for end-stage heart failure. It has been generally accepted that transplantation in properly selected patients is beneficial in increasing patient chances of survival, enhance their capacity to exercise, help them to return to their normal lives and improve quality of life as compared to the conventional forms of treatment.\textsuperscript{27} A heart transplant is recommended in patients having symptoms of heart failure, a poor prognosis and with no alternative treatment available for them. Added to this, patients who are being selected for heart transplant have to be well informed, motivated, emotionally stable and capable of good compliance with the intensive and extensive medical treatment that is involved.\textsuperscript{27}

Heart transplantation is needed for life survival in patients with end stage heart failure which may be due to different causes, such as, coronary heart disease, hereditary traits, viral infections of the heart, damage to the valves and muscle of the heart.\textsuperscript{133} Heart transplantation is also done in cases of heart failure without any known cause, use of alcohol and illicit drugs and congenital heart disease.\textsuperscript{134}

**Procedure of heart transplant**

Heart transplant is done through an *open-heart* surgery. A heart-lung bypass machine is attached to the arteries and veins of the heart and it
pumps blood through the patient’s lungs and body during the surgery. The heart of the patient is removed and the donor heart is then attached to replace it and connected to the incoming and outgoing blood vessels. The aorta and the pulmonary arteries of the patient are not replaced during the surgery.\textsuperscript{133,134}

There are many investigations which have to be done before the transplant is carried out and these comprise blood tests, chest X-ray, ECG, coronary angiogram, echocardiogram, cardiopulmonary test, ultrasound of the abdomen and blood vessels, breathing tests and pulmonary function tests, detection of stool samples for blood and measurements of pressure in the heart chambers.\textsuperscript{134} Following the heart transplant surgery, the patient is kept in the cardiovascular intensive care unit and on ventilator support to facilitate proper breathing. Pain medications are given to the patient intravenously during the recovery period.\textsuperscript{134}

Frequent blood tests and measurement of kidney functions, are also carried out to monitor the condition of the patient after the surgery. The patient is continuously observed for the presence of any post-transplant complications or infections. Heart biopsies are also carried out to monitor the patient to assess for any signs of tissue rejection. Also, counseling from a dietician, physiotherapist and a pharmacist is needed to prepare the patient for managing self-care efficiently.\textsuperscript{134}

\textit{Pharmaceutical care post transplant}

Following the transplant surgery many medications are required by the patient, which includes immunosuppressant, statins, antibiotics and some drugs to alleviate high blood pressure or other complications associated
with the surgery. The immunosuppressant drug is intended to lower the risk of rejection while statins are intended to treat cholesterol and maintain it within optimum range.\textsuperscript{134}

\textit{Limitations of the heart transplant}

There is a shortage of donor hearts, which is a primary challenge in heart transplantation. The other limitation related to heart transplantation involves the prevention of rejection of the allograft, which may otherwise lead to death in the first post-operative year. The outcome of the heart transplantation is also restricted by the long-term immunosuppressant therapy, such as, infection, hypertension, renal failure, malignancy and coronary artery disease.

The selection of the transplant patient is to be made carefully on the basis of the severity of their heart failure symptoms, end stage heart failure, absence of any other serious co-morbidities and the lack of availability of any other alternative treatment options.\textsuperscript{72} The patients who have gone for heart transplant are to stay on a lifelong medical care plan that consist of multiple medication and frequent follow up medical check-ups.\textsuperscript{133} One of the most important barriers in heart transplantation is that once a patient has been selected for the heart transplant they are kept on a waiting list for the availability of the donor heart. The waiting period can vary widely from days to months. The conditions determining the period of waiting for a particular patient involves the availability of the organ, blood group, severity of the condition, or the presence of serious medical events.\textsuperscript{134}
Contraindications of heart transplant

A heart transplant is contraindicated in patients that have continued alcohol or drug abuse, lack of proper cooperation, and the presence of any serious mental illness that is not properly controlled. Patients that have undergone cancer treatment, with remission and less than 5 years of follow up, are also contraindicated to receive a heart transplant. Other conditions which also act as a contraindication for heart transplantation can be enumerated as the presence of systemic disease in which multiple organs are involved, active infection, significant renal failure with creatinine clearance (below 50mL/min), irreversibly high pulmonary vascular resistance, recent thromboembolic complications, peptic ulcer which has not been healed, presence of substantial liver impairment, or presence of any other serious co-morbidity with a poor prognosis.27

Risks of heart transplant

There are several risks found to be associated with heart transplantation that include deep venous thrombosis, damage to the kidneys, liver, or other vital organs due to the use of immunosuppressant, development of cancer, heart attack, stroke, irregularities in the rhythm of the heart, increase in cholesterol, diabetes, depletion of bones, increased risk for infections, severe coronary artery disease and infection at the site of insertion. As mentioned above, there is a risk of rejection of the new heart.135

Chronic Management

The pharmacological therapy of heart failure is targeted to reduce the mortality and the morbidity in the heart failure patients. These are briefly outlined in the following section.
Angiotensin Converting Enzyme Inhibitors

Angiotensin converting Enzyme inhibitors (ACEIs) are the drugs of choice in all patients showing symptoms of heart failure. The use of ACEI is known to improve the ventricular functioning and the wellbeing of the patient; it reduces the chances of hospitalization and prevents heart failure from worsening. In patients who are hospitalized for heart failure, the ACEI should be started before the patients are released from the hospital.27

Benefits of ACEIs

The use of angiotensin converting enzyme inhibitors in patients with heart failure has been known to reduce the total mortality and hospitalization due to heart failure across all ages, improve the functional capacities, improve the degree of left ventricular dysfunction and alleviate the causes of heart failure. ACEIs serve as the background therapy in all heart failure trials and helps to improve the hemodynamic as well as the neurohormonal profile of the patient.136

The use of ACEIs reduces the incidence of congestive heart failure by almost 9.0%. It reduces the chances of revascularization, complications of diabetes such as nephropathy, retinopathy, dialysis and new development of diabetes. It has also been observed that the use of ACEIs in high-risk patients prevents further progression of angina.137 The use of ACEIs has been instrumental in reducing total mortality and hospitalization in a broad range of patients.138 Research studies have shown that in older patients who have been hospitalized for heart failure and show preserved ejection fraction there has been modest improvement in the composite end point of total mortality or heart failure hospitalization.139
Mechanism of action and dosing of ACEIs

Angiotensin converting enzyme inhibitors act by inducing vasodilation in patients with heart failure by removing angiotensin II, which has vasoconstrictor action.\textsuperscript{140} It also increases the endogenous levels of kinins in circulating blood as well as the tissues along with an increase in the synthesis of prostaglandins.\textsuperscript{141} ACEIs are also associated with the reduction in the action of hormones, such as, catecholamine and vasopressin, which have a vasoconstrictor action. Its effect is brought about by down regulating the central and peripheral sympathetic nervous systems activity.\textsuperscript{140} Before the ACEI is initiated in patients with heart failure, the patient’s renal function and electrolyte balance is evaluated. The initial dose of different ACEIs range between 0.5 mg to 6.25 mg per day and the target dose is from 4 mg to 100 mg per day.\textsuperscript{27} A detailed description of the starting and the target doses of different ACEIs is shown in the table below.\textsuperscript{27}

<table>
<thead>
<tr>
<th>ACEI</th>
<th>Starting dose (mg)</th>
<th>Frequency</th>
<th>Target dose (mg)</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Captopril</td>
<td>6.25</td>
<td>t.i.d.</td>
<td>50-100</td>
<td>t.i.d.</td>
</tr>
<tr>
<td>Enalapril</td>
<td>2.5</td>
<td>b.i.d.</td>
<td>10-20</td>
<td>b.i.d.</td>
</tr>
<tr>
<td>Lisinopril</td>
<td>2.5-5.0</td>
<td>o.d.</td>
<td>20-35</td>
<td>o.d.</td>
</tr>
<tr>
<td>Ramipril</td>
<td>2.5</td>
<td>o.d.</td>
<td>5</td>
<td>b.i.d.</td>
</tr>
<tr>
<td>Trandolapril</td>
<td>0.5</td>
<td>o.d.</td>
<td>4</td>
<td>o.d.</td>
</tr>
</tbody>
</table>

Contraindications and side effects of ACEIs

There are some cases in which the use of ACEIs is contraindicated and this includes a history of angioedema, bilateral renal artery stenosis, and
potassium serum levels over 5.0mmol/L, serum creatinine levels over 220 µmol/L and the presence of severe aortic stenosis.\textsuperscript{27}

The adverse effect profile of ACEIs is excellent and it is well tolerated by most of patients.\textsuperscript{140} The most common side effects associated with the use of ACEIs in patients of heart failure are further discussed below.

\textit{Reduced renal function}

The use of ACEIs may lead to a rise in the blood urea and creatinine when the therapy is initiated. An increase of creatinine up to 265 µmol/L is acceptable but when it rises above 310 µmol/L then the dose of ACEI needs to be reduced to half and the serum creatinine should be regularly monitored. If the concentration of creatinine rises beyond 310 µmol/L, the ACEI should be immediately discontinued.\textsuperscript{27}

\textit{Hyperkalemia}

Angiotensin converting Enzyme inhibitors use may lead to an increase in the blood potassium levels and when the concentration raises over 6.0 mmol/L the ACEI is stopped immediately.\textsuperscript{27}

\textit{Symptomatic hypotension}

Dizziness due to a reduction in the blood pressure may occur with the use of ACEIs.\textsuperscript{27} This requires reporting and possible dose adjustment by the patient’s healthcare provider.
Cough and angioneurotic edema

Cough and angioneurotic edema has been reported in some cases. Cough has been reported in about 15% of the patients being treated with ACEIs. When this occurs the patient is encouraged to notify their healthcare provider for possible dose adjustment.

Drug interactions

When ACEIs are used with non-steroidal anti-inflammatory drugs, which are nephrotoxic in nature, there is an increase in the creatinine levels and the dosage has to be adjusted. The use of potassium supplements or potassium sparing diuretics should be avoided with ACEIs as there may be an abnormal rise in the serum levels of potassium in the blood. Diuretics and other hypotensive agents except angiotensin receptor blockers (ARBs) or beta-blocker or aldosterone antagonist should be used with caution.

Drugs with an effect on the kidneys, such as furosemide or hydrochlorothiazide, should be used with caution in patients that are on ACEIs. When the ACEI is initiated in patients taking insulin, a drop in the blood sugar level to very low levels may occur. Hence, the ACEI should be given with caution in patients taking insulin. Certain drugs available as over the counter medications, for example, ibuprofen or acetaminophen, may reduce the efficacy of ACEIs and should be avoided.

Iron in congestive heart failure

The two major risk factors of heart failure are anemia and concurrent renal impairment, which consist of the cardio renal impairment and cardiorenal anemia syndrome. Cardiorenal anemia syndrome is defined as
a pathological triangle in which the primary failing organ is the heart and a
dysfunction of the one organ (the heart) leads to the dysfunction of the
other (the kidney). The anemia present in heart failure is a complicated
phenomenon and is due to multiple factors.

The commonly occurring causative factors for anemia in heart failure can
be enumerated as hemodilution, deficiency of iron (absolute or
functional), stimulation of the steps causing inflammation and faults in
the production of erythropoietin. Presence of myelodysplastic syndrome
and chemotherapy may have a further worsening effect on the outcome.
Anemia or renal dysfunction in patients of heart failure results in an
increase in morbidity and mortality.

Researchers have also shown that anemia occurs commonly in patients
with congestive heart failure and is also considered to be an independent
prognostic marker for mortality in community based patients with
congestive heart failure. The therapeutic approach to correct this
deficiency depends on the pathophysiology of cardiac anemia and includes
the improvement of hemoglobin levels and oxygenation of the tissue.
Hence, it is important that correction of anemia is done to reduce the
severity of heart failure.

The improvement of hemoglobin levels is directly related with an
improvement in the energy production in cardiomyocytes. Anemia is
known to increase the severity of the symptoms of heart failure. When
there is volume overload there is further depletion of hemoglobin and
worsening the anemia in the patient. The symptoms of anemia are
shortness of breath, tachycardia, dizziness, faintness and fatigue, all of
which are linked to the severity of the symptoms of congestive heart
failure.\textsuperscript{151} Patients with congestive heart failure exhibit endothelial dysfunction, which also contributes to anemia in CHF.\textsuperscript{151}

\textit{Deficiency of iron}

Hematinic abnormalities, especially iron deficiency and hemodilution, are a major cause of anemia in patients with congestive heart failure. Patients with CHF and chronic kidney disease have shown a more pronounced case of anemia.\textsuperscript{153} More than the severity of heart failure is the iron deficiency as a problem leading to worse outcomes.\textsuperscript{154} In most cases, iron stores are decreased and iron deposits in the bone marrow are also reduced. In others, the iron deficiency is characterized as functional or relative (defined as an increase in hemoglobin (Hb) or a decrease in erythropoiesis-stimulating agent (ESA) requirement. The reason for this absolute deficiency of iron has been cited to be:

1. Low intake of iron, which is due to low protein diet, anorexia.
2. Loss of blood through gastrointestinal system, which may be due to a dysfunction of the platelets, abnormalities in blood coagulation.
3. Poor absorption of iron.

The functional iron deficiency present in congestive heart failure is related to the disuse of iron.\textsuperscript{27} The release of iron from ferropoetin is also blocked by hepcidin and hence there is inadequate delivery of iron to the bone marrow erythroblasts, which further leads to iron deficiency.\textsuperscript{156} In congestive heart failure, iron metabolism is impaired and either absolute or functional deficiency of iron results in impairment of the oxidative metabolism, cell energetic and immune mechanisms of the cell.
Iron has an anti-inflammatory action and iron-deficiency anemia further leads to an enhancement of the red cell oxidative stress. A study has reported that disordered iron homeostasis to be an independent risk factor for death. Iron deficiency in congestive heart failure also causes thrombocytosis, which eventually increases thrombosis and the mortality rate.

_Treatment of iron deficiency_

The recommendations for the treatment of anemia are to correct the causes of anemia such as iron, folate or vitamin B12 deficiencies. Iron deficiency may contribute to an increase in mortality in cases of congestive heart failure whereas correction of the iron deficiency is related to an improvement in the symptoms and status of the syndrome. Intravenous preparations of iron are reported to improve certain signs and symptoms of congestive heart failure. Correction of iron deficiency is also related with an improvement in exercise tolerance and duration along with an improved quality of life for patients with congestive heart failure.

Studies have also reported that the oral form of iron is not beneficial in congestive heart failure in terms of improving any parameters of CHF. Oral or intravenous iron without erythropoiesis stimulating agents (ESA) has been used to improve hemoglobin levels and has shown a positive effect in congestive heart failure related to the need for hospitalization, NYHA functional class, cardiac and renal function, reduced b-type natriuretic peptide, stamina to exercise and an improved quality of life. Other studies have also reported the need for ESA and intravenous iron in congestive heart failure.
The treatment with iron may prove beneficial in alleviating the symptoms of heart failure in patients. Clinical trials have been conducted to demonstrate that intravenous iron can improve symptoms and physical functions in patients with congestive heart failure and anemia. In animal models, iron therapy has shown to improve work performance and improve exercise capacity without any change in the hemoglobin levels. Studies thus conclude that iron is a cofactor needed for exercise. Iron deficiency is also associated with an impairment of myocardial mitochondrial electron transport in the heart of rats. Hence, it has been clearly deduced that intravenous iron treatment may lead to subjective and objective outcomes in patients with heart failure. Experimental evidence has also suggested iron to be a cofactor for muscle function.¹⁶²

**Palliative Care**

Palliative care or supportive care for the elderly to manage the condition of heart failure is targeted to take care of the symptoms, psychosocial or existing distress and ways to manage their condition and to help them cope with heart failure. Palliative care is needed for the elderly along with the clinical therapy that is ongoing for them. The various aspects of palliative care consist of providing supportive therapy, educating the patient and the family about the care needed, and facilitating communication and decision making between physicians and patients.¹⁶³

Heart failure is marked by exacerbations of the symptoms of heart failure and frequent hospitalizations. Palliative care includes a discussion of prognosis and advanced strategies, management of the symptoms, comorbidities and hospice care. The use of ACEIs, beta-blockers, spironolactone, diuretics and digoxin indicated for heart failure patients,
and proper self-care education, can help in alleviating the symptoms of the patient and also improve his/her quality of life.\textsuperscript{164}

The main aim of palliative care is to alleviate the symptoms related to heart failure; such as, fatigue, dyspnea, and exertion. Dietary interventions, which are aimed at increasing fluid intake and reducing the sodium intake, help in reducing fatigue and edema in patients. Oral opioids function to improve dyspnea and the ventilatory response to exercise.\textsuperscript{165} Optimal medical management is the cornerstone in alleviating the symptoms of heart failure and improving the patient’s quality of life. The aim of palliative treatment is to provide comfort to the patient along with medical management.\textsuperscript{164}

**Pain management**

Studies have claimed that patients dying of advanced heart failure suffer from the most distressing symptom of pain and almost 41\% of the patients hospitalized with heart failure experienced moderate to severe pain in the last three days of their lives. The cause of the pain may be cardiac causes inclusive of angina, edema, and presence of comorbidities, such as, osteoarthritis, diabetic neuropathy or shingles or even medical interventions such as chest tubes. Irrespective of the cause of the pain, the patients must be given opioids or other medications for the management of the pain.\textsuperscript{164}

Pain occurs commonly in elderly patients with heart failure. The pain is predominant in the chest, legs and joints. Pain is also present in other sites in the body.\textsuperscript{165} The use of non-steroidal anti-inflammatory drugs is contraindicated in patients of heart failure because its use may lead to
sodium and fluid retention, affecting the kidney function and hence further worsening the condition of heart failure.\textsuperscript{167}

The patients suffering with osteoarthritis or chronic musculoskeletal pain are given opioids for the alleviation of pain. A combination of muscle-strengthening exercises, use of assistive devices, heat, cold ultrasound modalities and intra-articular joint injection are also recommended for these patients.\textsuperscript{163} Intracoronary stenting has been found to be appropriate to be used in selected patients when angina pain is recalcitrant to pharmacotherapy. Opioids should be used as the first line drugs for the alleviation of moderate to severe pain.\textsuperscript{27} Elderly patients can be safely administered opioids during cardiac anesthesia. Opioids have a wide range of effects on the cardiovascular system as well as the nervous and endocrine systems. Its use can also cause bradycardia, hypotension and a suppression of the respiratory function hence opioids are given under supervision via the parenteral route.\textsuperscript{163}

Some recommendations for the use of opioids in pain management are listed below.\textsuperscript{163}

- Pain relief is provided by initiating therapy with short acting opioids and then titrating the dose to the amount of pain relief.
- If the pain is intermittent, intermittent opioids are given for chronic or for persisting pain long–acting opioids are given through the day.
- Laxatives are given along with all opioid prescriptions.

Other drugs that are given to the elderly for pain relief are morphine and codeine. Morphine and codeine are only given in intermittent doses because they are actively cleared renal metabolites and may lead to delirium or myoclonus in heart failure patients.\textsuperscript{163} Fentanyl and
methadone can be given to heart failure patients for pain relief in the elderly. Methadone can cause torsades de pointes.\textsuperscript{169}


\textit{Hospice care}

Hospice care can be referred to as formal palliative care where the palliative care is initiated and the heart failure care is ceased. Research studies have shown a slow movement of patients with decompensated heart failure from the hospital to hospice.\textsuperscript{170}

Hospice care includes the provision of oral medications for heart failure patients, and opioids for the treatment of pain-like symptoms in the heart failure patient. Some larger hospices also provide more complicated and expensive treatments such as intravenous pharmaceuticals or inotropes.\textsuperscript{171} A continuing assessment using prognostic models helps in directing the need for hospice care. If an increase in the hospitalization frequency has been noticed in the last 6 months, it is an indicator for hospice care.

Currently, an integrative model is being used which states that palliation occurs when life-prolonging therapies are administered. With the progression of the illness, palliative therapies are generally expanded. The decision for hospice care has to be made in consensus with the patient, family members and physicians. Generally when the harm of therapies outweighs their benefits, hospice care is chosen.\textsuperscript{168} For enrolment in hospice care the physician must refer the patient. Certain guidelines have been established by the National Hospice and Palliative Care organization for enrolment of the patient in hospice care to ensure efficient management of their disease.\textsuperscript{173}
The patients suffering with heart disease should meet the following criteria:

- The condition of the patient should be life limiting and the patient and his family should be aware of this.
- The treatment selected is directed towards providing the symptomatic relief from the condition.
- Patients should have either a worsening of the primary disease, hospitalization in the last 6 months, multiple emergency visits to the doctor, and has received home health services or are witnessing a recent decline in their health condition.
- The patients show impairment of the nutritional status such as progressive weight loss within 6 months or serum albumin less than 2.5 g/dL.
- Presence of intractable or frequently recurrent symptoms of heart failure or angina pectoris with heart failure.
- Patients have been initiated on pharmaceutical therapy and tolerated it.
- Symptoms of arrhythmias, prior history of cardiac arrest, resuscitation or syncope cardiogenic brain embolism or concomitant HIV disease point towards poor prognosis of the disease.

In the United States hospice care can be defined as the at-home service for people with various life threatening conditions. Hospice care is beneficial in providing relief from physical, emotional, psychological, spiritual and existential suffering for the patient and his/her family. Another important aspect of hospice care is that hospice also offers support to the caregiver, which is an additional benefit and respite to the patient and his family.\textsuperscript{164}
Clinicians and nurses with heart failure expertise are needed to work at hospice to manage patient care and to improve the skills and the knowledge of the health staff providing care for hospice patients. A patient cannot stay indefinitely at hospice but for specified lengths of time at the end of which either the patient should be discharged or recertified that he/she is likely to die within six months.

Careful management may lead to an improvement of the condition of the patient and prognostic tools become helpful in re-evaluation of the patient. The patient can also revoke the benefits of hospice care if he/she can get better care elsewhere. Hence, clinicians should show an ability to adopt a palliative care approach, which has an emphasis on improving the quality of life, opens communication with the patient and shows respect for patient autonomy.

**Nursing and homecare guidelines**

Nurse led outpatient clinics and patient education and self-care are the focus of this section. As one of the major reasons for the morbidity and mortality in most developed countries, patients with congestive heart failure are looking for more cost–conscious and effective care strategies to manage their illness. Nurse practitioners play an important role in this context and studies have emphasized the role of multi-disciplinary outpatient congestive heart failure clinics.

According to different clinical trials and research studies nurse led follow up at outpatient heart failure clinics improved survival and self-care behavior in patients with heart failure; it reduced the number of events and the need for hospital care. This type of nurse–led follow up is also considered to be cost effective.
There are challenges in the care and management of heart failure. Different issues are prevalent with respect to the management of heart failure in patients that are challenge the provision of effective care and to improve outcomes. One of these reasons is the lack of optimal treatment given to the patient, including ineffective patient education on heart failure and self-care. Non-compliance with medications, diet or monitoring and not recognizing the symptoms of CHF leads to readmissions to the hospital.

The interventions and benefits of nurse led outpatient clinics are important to note and disseminate within communities. Researchers have emphasized the importance of nurse led clinics as an effective measure of clinical intervention. The interventions offered by the nurse led clinics include assessment, monitoring and consultation on the risk factors of heart failure. Patients were thoroughly educated about lifestyle modifications, prevention of disease, and compliance with medication intake in these nurse led clinics. Nurse led clinics may be of two types:

1. Nurse led clinic in general practice settings;
2. Nurse led clinic based in hospital with home follow-up.

A study conducted to assess the difference in the outcome of usual care and nurse led outpatient care reported that even though there was no effect on the overall mortality, the benefits of nurse led heart failure interventions included a reduction in hospital usage and a reduction in the number of readmissions as well. The nurse led clinics provided post discharge heart failure education, medication counseling, review and telephone follow up. Discharge planning in the nurse led clinics has also been effective in the care of the heart failure patients. Clinical trials conducted to evaluate the importance of nurse led interventions for CHF
patients has shown that nurse led intervention and clinical care led to an improvement in event-free survival just after a single home-visit by the nurse following hospitalization.

The nurse led care has brought about a significant reduction in patient admission to the hospital (by almost 43%) while the stay in the hospital has also been shown a reduction in almost 41% of patients. The nurse led care is also efficient in improving the self-care behavior in patients. Clinical studies have reported that with the help of nurse led care, compliance with daily weighing, alerting health care at weight gain and restricting the fluid intake improved considerably. Thus, it can be concluded that nurse led care is not only beneficial in improving self-care but it also helps the patient in recognizing symptoms and monitoring them regularly.

The role of nurse led clinics

Different health care teams have incorporated nurse-led or nurse-coordinated disease management programs for patients with heart failure whose integral services consist of early follow up following hospitalization, detailed and intensive patient education. The management is led in such a way so as to prolong the survival without exacerbations of any symptoms, cut down the number of hospital admissions, improve care and drug compliance, improve self-care behavior and the quality of life. It is important that nurses working in these clinics should be adequately equipped with special education and training to be able to work independently and to provide high quality medical intervention to the patient. The changes made in the treatment plan include ongoing management of medication dosing and any required changes, such as the ACEI or the beta-blocker.
After discharge from the hospital, the first visit at a nursing led clinic is scheduled within 2 to 3 weeks. The check-up includes anamnesis (preliminary case history), auscultation of the heart and the lungs and inspection of edema. In an hour-long visit, the cardiac nurse performs an assessment and evaluation of the patient and optimizes the treatment given to the patient. If the nurse feels that there is a need to optimize the treatment, then in consultation with the cardiologist and according to the current clinical guidelines, the treatment may be changed. During the visit, the nurse gives education to the patient and support to the family members and caregivers. During the visit the nurse decides if there is a requirement for another visit to the clinic.

**Patient education**

Individual education is provided to the patient in oral form as well as written form by the nurse. The education provided to the patient is based on current clinical guidelines. Both the patient and their families are given education about the disease course of CHF, treatment and management. The informative content includes a definition of heart failure, sign and symptoms of heart failure, etiology of the disease, the rationale for the treatment, and counseling about the drugs prescribed. Additionally, the patient is informed about the significance of non-pharmacological treatment. The nurse informs the patient about the essential dietary changes, such as, restricted fluid, cessation of smoking, limited use of alcohol, restricted fluid, prevention of malnutrition, exercise if the heart failure is stable, the adjustment of calorie intake and energy to prevent being overweight, and prophylaxis of infection with vaccinations.

The education given to the patient at the nursing led clinic is completely individualized based on the previous knowledge given to him/her, the
level of education and cognitive functionality of the patient. The primary aim of educating the patient is to improve their self-care and to improve the disease condition and to monitor symptoms, such as weight gain, increased breathlessness and edema.

Psychosocial support

Nursing intervention at the clinic also includes improving the support and communication between the nurse and the patient and their families. The patients can contact the nurse by telephone. Nurses take initiative to call and give psychosocial support to clinic patients, to estimate the changes of the drugs or other medical interventions implemented due to worsening of the symptoms or presence of side effects.

Heart failure is a complicated disease and poses a challenge to the patients and their families in managing the health of the patient. Because of this there is a need for customized education on self-care, its maintenance and management throughout the natural progression of the disease, suitable recommendations on the self-care and lifestyle modifications from the early stage of the disease through to terminal care.

Recognition of symptoms

It has been recommended that health professionals should make themselves aware of the personal situation of patients and give them customized advice and education to improve their quality of life. Some of the areas where patient education is recommended includes symptom recognition and, as previously mentioned, fluid and sodium management, nutrition and weight management, smoking and alcohol, physical activity,
immunization, improving self-care in heart failure, sexual advice, depression, remote patient monitoring, sleep disordered breathing and travel.

Symptoms of heart failure are indicative of the disease condition and also impact the patient’s lifestyle and mental state, such as anxiety or depression. The patient should be educated well so that he/she can recognize the warning symptoms such as breathlessness, fatigue, dizziness or loss of appetite and report to the health care team so that a timely and appropriate action can be taken. It is up to the clinician to assess and interpret the symptoms described by the patient. Use of standardized scales has been advocated so that the clinician can rate the intensity of the symptoms. Scales which can be used are numeric rating scale, visual analogue scale or a Likert scale.

Adequate patient education during the patient’s visits to the nurse led outpatient clinics or home visits by the nurses not only enhances the self-care behavior in heart failure patients but also helps them to maintain healthy functioning and overall well-being. Self-care attitudes such as adherence to medication, diet and exercise along with monitoring and self-management of symptoms and daily monitoring of weight to assess fluid and sodium retention are improved with proper patient education. Education should be provided according to patient learning needs, for example, through oral instructions, written material and health content on websites. Well-educated patients seek medical help when symptoms occur and are also actively involved in their care thereby reducing the readmissions and improving survival.
The role of the nurses providing care in nurse-led and outpatient clinics includes educating patients to promote self-care behavior. The nurse can identify various psychosocial factors affecting the patient such as depression, anxiety, and impaired cognition, poor understanding of health information, illiteracy and sleep disturbances and can appropriately educate the patient. Depression may affect the learning and cognitive ability of the patient, his/her willingness and ability to perform self-care, and a lack of motivation may affect the outcome of heart failure. Nurse-led patient clinics can provide social and psychological support to the patient and promote self-care to enable a successful treatment.

*Remote monitoring*

As part of the patient education, remote monitoring and support in patient’s homes have been effective in improving outcomes in patients with heart failure. Remote monitoring in the form of telemonitoring or web chats can decrease the use of health care facilities by reducing the number of hospitalizations for chronic heart failure, fewer heart failure related readmissions and more effective and efficient device management. Telemonitoring is quickly gaining approval and is being integrated in nurse led care of heart failure patients. It is especially beneficial in cases where patients reside in remote and rural areas and for homebound elderly patients at risk of clinical worsening of their symptoms of heart failure. Owing to these benefits, the follow-up after hospitalization led by the nurses is an integral part of the guidelines for diagnosis and treatment of chronic heart failure.

*Summary*

Congestive heart failure is one of the leading causes of morbidity affecting on an average 550,000 new people every year in the U.S. alone. The main
concern about the CHF is that it is a chronic, progressive disease and significantly decreases the patient’s quality of life. CHF affects multiple body systems and the individual as a whole; holistic approach is required in the care of CHF patients; and, it is classified into various classes as per the sides of the heart affected, the type of pathophysiology and the nature of the illness. CHF is considered to be a high output failure when the cardiac output is more than the body requirement (> 8 liters) occurring due to impairment in the systemic vascular resistance or excessive administration of fluid or blood. In the low output heart failure the cardiac output is very lower than the body requirement (< 3 liters) occurring due to disturbance in the contractile functions (either systolic dysfunctions and/or diastolic dysfunctions) of the myocardium of the heart.

CHF is termed as left sided heart failure when there is involvement of the left ventricle of the heart that is characterized by respiratory symptoms. That is why it is also known as pulmonary edema heart failure. While the right-sided heart failure occurs when there is right ventricular failure alone or as a consequence of left ventricular failure characterized by systemic symptoms like edema, hepatomegaly, and cyanosis. That is why it is also known as systemic heart failure.

When both the heart ventricles are involved it is known as biventricular heart failure. The congestive heart failure is termed as acute heart failure when the patient presents with acute symptoms of heart failure, which can be of new onset or an acute exacerbation of chronic CHF; whereas, chronic heart failure refers to long-standing heart failure of a progressive nature. There are variety of causes of heart failure associated with the more common forms of heart disease, such as, coronary artery disease, cardiomyopathy, hypertension, diabetes mellitus, valvular heart disease,
alcoholism, infective disease, cardiac arrhythmias, and anemia. It remains extremely impotent to determine the cause of CHF when deciding the treatment and the prognosis of each individual case of CHF.

Signs and symptoms of CHF are purely dependent on the side of the heart involved in heart failure. The cardinal manifestations of left sided heart failure are comprised of shortness of breath, fatigue, cyanosis, pulmonary rales, S3 gallop, and various types of cardiac murmurs. The cardinal manifestations of right-sided heart failure include leg swelling, ascites, hepatomegaly, and peripheral cyanosis. The clinical diagnosis involves a combination of clinical evaluation, physical examination, blood investigation, chest X-ray and echocardiogram. Further disease evaluation is very necessary to determine the cause of the CHF since the cause determines the management of each case of CHF.

Most of the time, the treatment of CHF is multifactorial requiring a holistic approach, which commences with significant lifestyle changes like avoiding bad habits such as the use of alcohol, tobacco, smoking and acquiring good habits like regular exercise, losing weight, and dietary changes. Self-care can easily form the cornerstone of heart failure management and treatment. It has a definite effect on the symptoms, functional ability, wellbeing, morbidity and prognosis of the disease.

Providing psychological support to the patient is the most challenging part for health care providers and family members. Management of CHF is always done with setting long-term goals in combination with pharmacotherapy like ACEIs and regular monitoring of the patient in the home as well as through follow up visits with their health care providers on a regular bases. Recent advancement in the treatment consists of
various implantable devises like pacemakers and an implantable cardioverter defibrillator that are meant to assist with the heart’s pumping action and improving the circulation.

In end-stage heart failure when all other treatment fails to improve CHF the only available option remaining is heart transplant. Palliative care or supportive care to the elderly to manage the condition of heart failure is targeted to take care of the symptoms, psychosocial or existing distress, and is focused on ways to manage their condition and help them cope with heart failure. Since the disease is chronic in nature, people often fail to sustain treatment and search for more cost–conscious and effective care strategies. This has significantly increased the morbidity and mortality due to CHF.

The nurse has a pivotal role in the care of patients with CHF. The nurse practitioner role, in this context, has been emphasized and promoted through studies focused on the role of multi-disciplinary outpatient congestive heart failure clinics. According to different clinical trials and research studies the nurse led follow up at outpatient heart failure clinic improved survival and self-care behavior in patients with heart failure; it reduced the number of events and the need for hospital care. This type of nurse–led follow up is also considered to be cost effective. This approach is expected to lead to better outcomes in cases of CHF.

Please take time to help NurseCe4Less.com course planners evaluate the nursing knowledge needs met by completing the self-assessment of Knowledge Questions after reading the article, and providing feedback in the online course evaluation.

Completing the study questions is optional and is NOT a course requirement.
1. What is the condition in which the heart is unable to meet the demand for oxygen and nutrients for the cells of the body?

a. Coronary artery disease  
b. Congestive heart failure  
c. Metabolic syndrome  
d. Hypoxemia

2. What is an indicator that a patient is suffering from high output failure?

a. Urine output more than 8 liters per day  
b. Cardiac output more than 8 liters per minute  
c. Urine output less than 8 liters  
d. Cardiac output less than 3 liters

3. The following terms are used in the classification of CHF except

a. Acute and chronic heart failure  
b. High output and low output heart failure  
c. Upper and lower heart failure  
d. Left sided and right sided heart failure

4. Left sided heart failure is also known as

a. Systemic failure  
b. Pulmonary edema heart failure  
c. Respiratory failure  
d. Circulation failure

5. Which of the following is also termed as systemic failure?

a. Acute heart failure  
b. Chronic heart failure  
c. Right sided heart failure  
d. Biventricular failure
6. Which of the following is a key feature of backward failure of the heart?
   a. Decreased after load
   b. Increased pre load
   c. Decreased pre load
   d. Increased after load

7. Low output congestive heart failure carries a relatively ________ prognosis in comparison to high output heart failure.
   a. Poor
   b. Better
   c. Similar
   d. Favorable

8. When right ventricular failure occurs due to chronic pulmonary obstructive disease, the condition is known as
   a. Respiratory failure
   b. Cor pulmonale
   c. COPD
   d. PCOD

9. Following are the most common causes of CHF except
   a. Hypertension
   b. Heart attack
   c. Ischemic heart disease
   d. Appendicitis
10. Deficiency of vitamin B1 leads to which disease?
   a. Paget’s disease
   b. Beriberi
   c. Biventricular failure
   d. Scurvy

11. Paget’s disease has cardinal pathology of
   a. Increased bone resorption and bone formation
   b. Increased heart rates
   c. Increased vasoconstriction
   d. Immunosuppressant

12. Ballooning of the aorta means
   a. Aortic stenosis
   b. Aortic rupture
   c. Aneurysm of aorta
   d. Dilatation of aorta

13. Cocaine toxicity leads to an increased level of the following substance in the blood
   a. Adrenalin
   b. Catecholamines
   c. Renin-angiotensin
   d. Coca

14. The most common presenting symptom of left sided heart failure is
   a. Shortness of breath
   b. Peri-oral cyanosis
   c. Leg swelling
   d. Weight loss
15. When does “paroxysmal nocturnal dyspnea” occur?
   a. At Night when sitting upright
   b. At night when lying down
   c. In the evening on exertion
   d. All of above

16. Which of the following is not a diagnostic feature of right sided heart failure?
   a. Leg swelling
   b. Abdominal bloating
   c. Ascites
   d. S3 gallop

17. The disturbance in the electrical conduction associated with low voltage on the ECG is suggestive of
   a. Thyrotoxicosis
   b. Amyloidosis
   c. Sarcoidosis
   d. Valvular heart disease

18. Which of the following is proved to be the best and cost effective blood test for diagnosing CHF in recent years?
   a. Natriuretic peptide
   b. Troponin
   c. Aldosterone
   d. Creatinine
19. What should be the fluid intake limit for the patients suffering from hyponatremia?

a. Up to 6 liters per day  
b. Up to 1 liters per day  
c. Up to 1.5 to 2 liters per day  
d. Up to 4 to 5.5 liters per day

20. What care should be taken by the patient of CHF related to sex?

a. Avoid sex  
b. Have sex only in the daytime  
c. Take sublingual nitro-glycerine before sex  
d. Take vitamin supplements before sex

CORRECT ANSWERS:

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HELPFUL WEBSITES:

American Heart Association. Heart Failure @ http://www.heart.org/HEARTORG/Conditions/HeartFailure/Heart-Failure_UCM_002019_SubHomePage.jsp.

National Hospice and Palliative Care Organization @ http://www.nhpco.org/.
References Section

The reference section of in-text citations include published works intended as helpful material for further reading. Unpublished works and personal communications are not included in this section, although may appear within the study text.


20. Harrison’s principles of internal medicine, volume 1, 18th edition: heart failure (ch 234), McGraw-Hill Companies, Inc.


23. ROBERT SOUFER, M.D. heart failure, yale university school of medicine heart book, ch – 14

24. Leo G. Kevin, Matthew Barnard, Right ventricular failure, Continuing Education in Anaesthesia, Critical Care & Pain j Volume 7 Number 3 2007.


26. The criteria committee of the newyork heart association: nomenclature and criteria for diagnosis diseases of heart and great vessels, ed 9, little, brown, boston, 1994, p 253.


34. Nicki R. Colledge, Brian R Walker, Stuart H. Ralston; Davidson’s principles and practice of medicine, Churchill Livingstone, Elsevier, 2010


37. Mihai Gheoghiade,MD; Robert O. Bonow, MD chronic heart failure in the united states a manifestation of coronary artery disease (http://circ.ahajournals.org/content/97/3/282.short#; page 282)

38. Gradman AH1, Alfayoumi F; From left ventricular hypertrophy to congestive heart failure: management of hypertensive heart disease


46. Robin P Choudhury and John MacDermot Heart failure in thyrotoxicosis, an approach to management, British journal of clinical
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1873689/)

47. Stern AB1, Klemmer PJ; High-output heart failure secondary to arteriovenous fistula. Hemodial Int. 2011 Jan 12. doi:
10.1111/j.1542-4758.2010.00518.x,

http://www.nhlbi.nih.gov/health/health-topics/topics/chd/types.html


http://www.ncbi.nlm.nih.gov/pmc/articles/PMC152844/


96. Cardiac catheterization and coronary angiography. NHS choices Web Site.


137. Silverberg DS, Wexler D, Blum M, Iana A. The cardiorenal anemia syndrome: correcting anemia in patients with resistant congestive heart failure can improve both cardiac and renal function and reduce hospitalization. Clin Nephrol. 2003;60(1, supplement 1): 593-3102.


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