

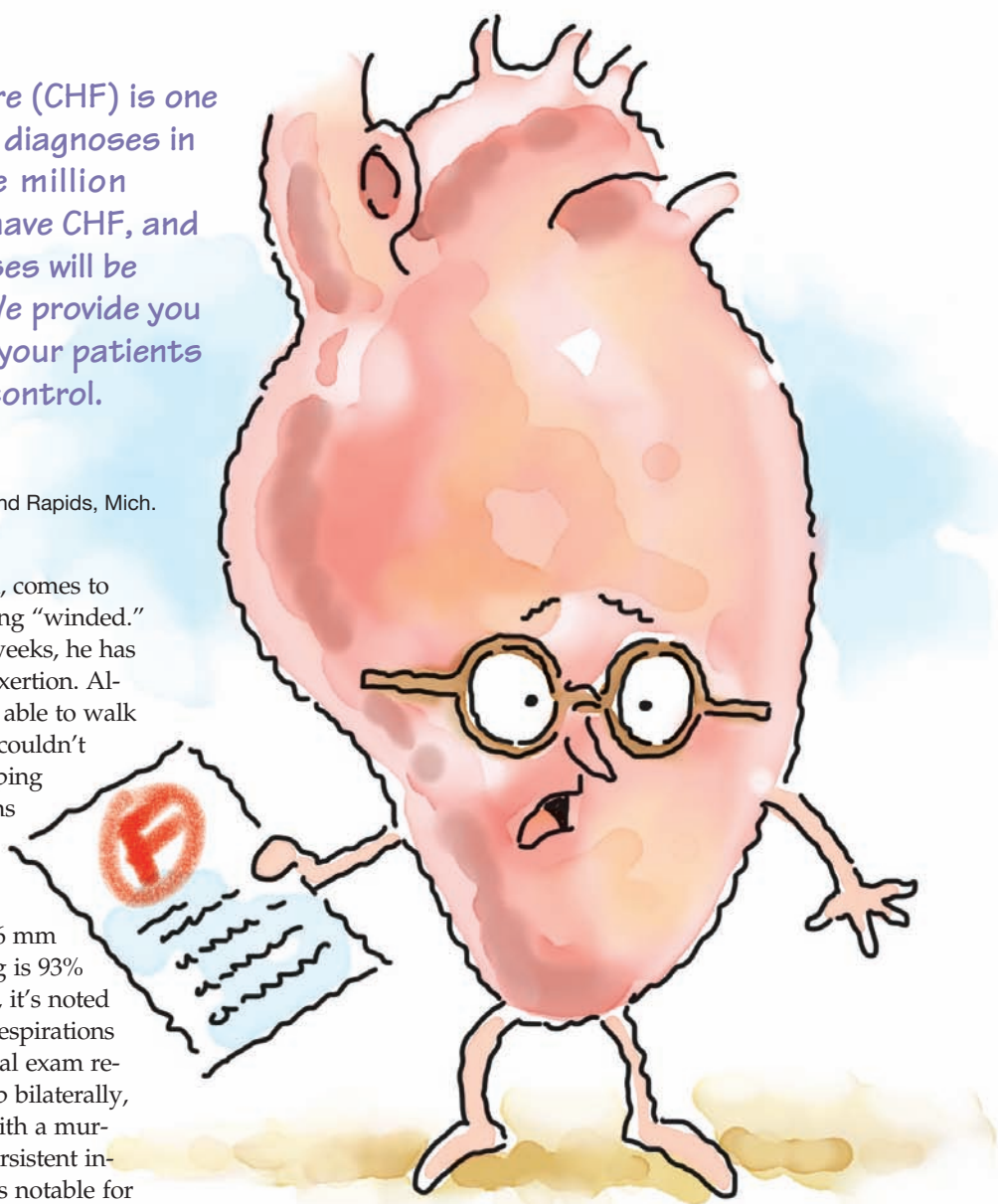
# Congestive heart failure:

## An 'F' isn't an option

*Congestive heart failure (CHF) is one of the fastest growing diagnoses in the world today. Five million individuals worldwide have CHF, and over 400,000 new cases will be diagnosed this year. We provide you with the tools to help your patients keep their CHF under control.*

By Michael Markaity, RN, PCCN  
Staff Nurse  
Spectrum Health Butterworth • Grand Rapids, Mich.

Mr. R, a 55-year-old Black man, comes to the ED with a complaint of being "winded." He states that over the past 2 weeks, he has noted worsening dyspnea on exertion. Although in the past he has been able to walk a 3-mile route easily, today he couldn't walk even 1 mile without stopping for frequent rests. His vital signs show a temperature of 98.6° F (37° C); heart rate, 102 beats/minute; respiratory rate, 20 breaths/minute, and BP, 158/96 mm Hg. His pulse oximetry reading is 93% but after going to the restroom, it's noted to be 86% with an increase in respirations to 26 breaths/minute. A physical exam reveals moist crackles halfway up bilaterally, and an S<sub>3</sub> heart sound along with a murmur. Mr. R has also noted a persistent increasing cough. His lab work is notable for a brain natriuretic peptide (BNP) level of 842 pg/mL.



Ms. T is a 74-year-old White woman who's next door to Mr. R. She was sent to the ED by her physician after an office visit showed a weight gain of 20 lb over the last month. She has a history of smoking and pulmonary hypertension. She complains that it's harder for her to bend over and tie her shoes, and she has also noticed that her shoes are tighter on her feet. Her vital signs and lab work are normal, with the exception of a BNP level of 1,290 pg/mL. Her physical exam reveals bilateral jugular venous distension and some abdominal distension. There's also 2+ pitting edema in her lower extremities, as well as in her feet and ankles. Her lungs, however, are clear.

## How it happens

## To the left!

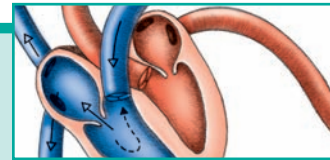
Both Mr. R and Ms. T have CHF, one of the fastest growing diagnoses in the world today. Over 5 million individuals worldwide have a diagnosis of CHF. As this diagnosis becomes more prevalent, over 400,000 new cases will be seen every year. Twenty percent of these patients will die in the first year, and 50% will die within a 5-year period. CHF accounts for 5% to 10% of total hospital admissions, and over \$38 billion is spent annually to treat this disease, according to the Family Practice Network.

Although it affects individuals of all races, Blacks are more prone to the disease, followed by Hispanics, then Whites. Comorbidities play a huge role in both the onset and treatment of CHF. These include, but are not limited to, pulmonary hypertension, coronary artery disease (CAD),

# 5

The right ventricle may now become stressed because it's pumping against greater pulmonary vascular resistance and left ventricular pressure.

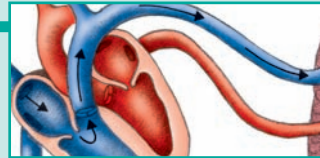
- You may note worsening symptoms.



# 4

Because the left ventricle can't handle the increased venous return, fluid pools in the pulmonary circulation, worsening pulmonary edema.

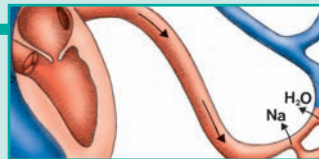
- You may note decreased breath sounds, dullness on percussion, crackles, and orthopnea.



# 3

Rising capillary pressure pushes sodium (Na) and water (H<sub>2</sub>O) into the interstitial space, causing pulmonary edema.

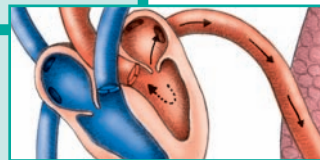
- You may note coughing, subclavian retractions, crackles, tachypnea, elevated pulmonary artery pressure, diminished pulmonary compliance, and increased partial pressure of carbon dioxide.



# 2

Blood pools in the ventricle and atrium and eventually backs up into the pulmonary veins and capillaries.

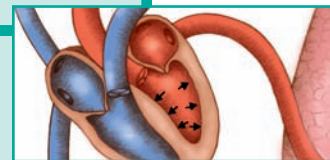
- You may note dyspnea on exertion, confusion, dizziness, orthostatic hypotension, decreased peripheral pulses and pulse pressure, cyanosis, and an S<sub>3</sub> gallop.



# 1

Increased workload and end-diastolic volume enlarge the left ventricle.

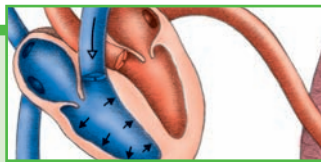
- You may note increased heart rate, pale and cool skin, tingling in the extremities, decreased cardiac output, and arrhythmias.



6

The stressed right ventricle enlarges with the formation of stretched tissue.

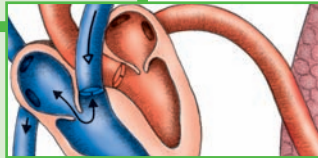
- You may note increased heart rate, cool skin, cyanosis, decreased cardiac output, dyspnea, and palpitations.



7

Blood pools in the right ventricle and right atrium. The backed up blood causes pressure and congestion in the vena cava and systemic circulation.

- You may note increased central venous pressure, jugular vein distention, and hepatojugular reflux.

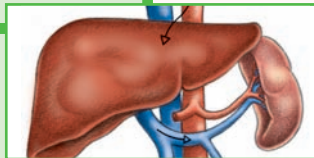


To the right!

8

Backed up blood distends the visceral veins, especially the hepatic vein. As the liver and spleen become engorged, their function is impaired.

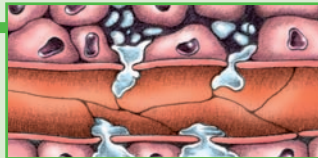
- You may note anorexia, nausea, abdominal pain, palpable liver and spleen, weakness, and dyspnea secondary to abdominal distention.



9

Rising capillary pressure forces excess fluid from the capillaries into the interstitial space.

- You may note edema, weight gain, and nocturia.



and diabetes. Access to, and the availability of, quality medical care can't be underestimated.

The following article will define CHF and provide an overview of its two types and their subclassifications, discuss signs and symptoms, and review current treatment options and modalities.

### A weak pump

To the public, CHF may often be thought of in terms of a "heart attack" or cardiac arrest.

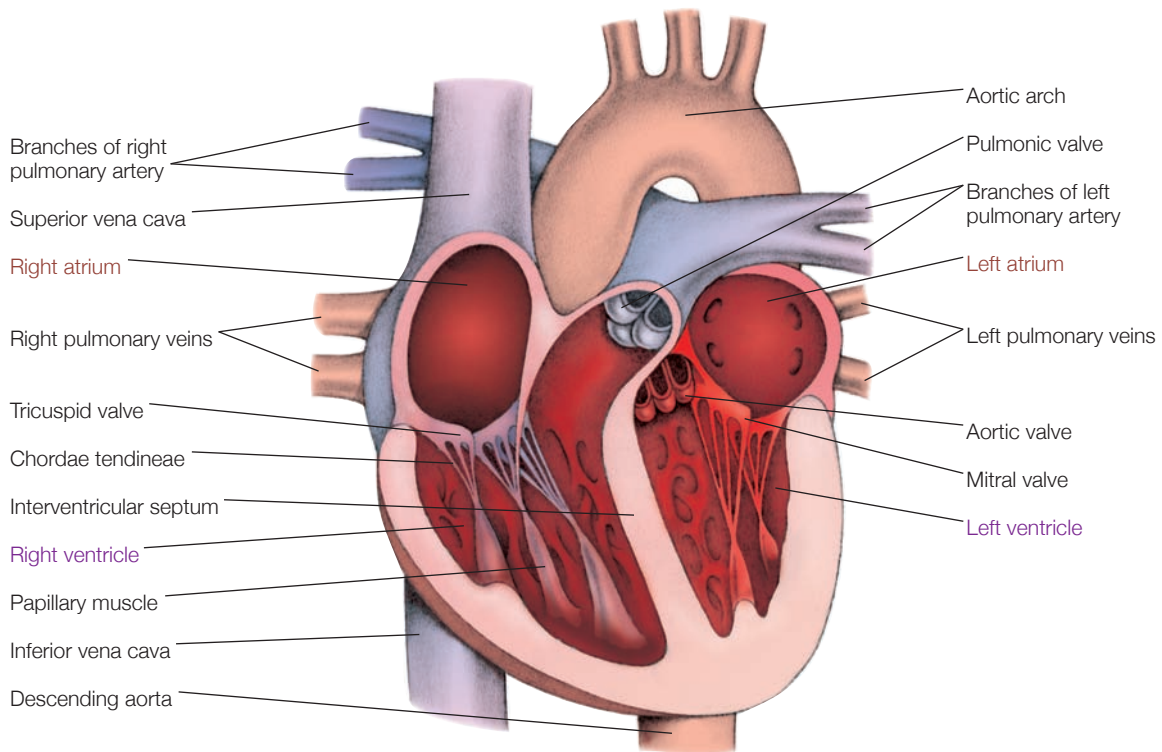
In fact, CHF is the inability of the heart to adequately pump blood throughout the body (see *How it happens*).

Carbon dioxide (CO<sub>2</sub>)-laden blood returns to the heart from both the superior vena cava and the inferior vena cava. It empties into the right atrium and is pumped through the tricuspid valve into the right ventricle. Via the pulmonary artery, the blood is sent to the lungs, where CO<sub>2</sub> and oxygen (O<sub>2</sub>) are exchanged. The reoxygenated blood is then brought to the left atrium by the pulmonary veins and deposited into the left atrium. It's then pumped through the mitral valve into the left ventricle, from which it moves through the aortic valve into the aorta and to the body (see *Inside a normal heart*). In CHF, this process is faulty.

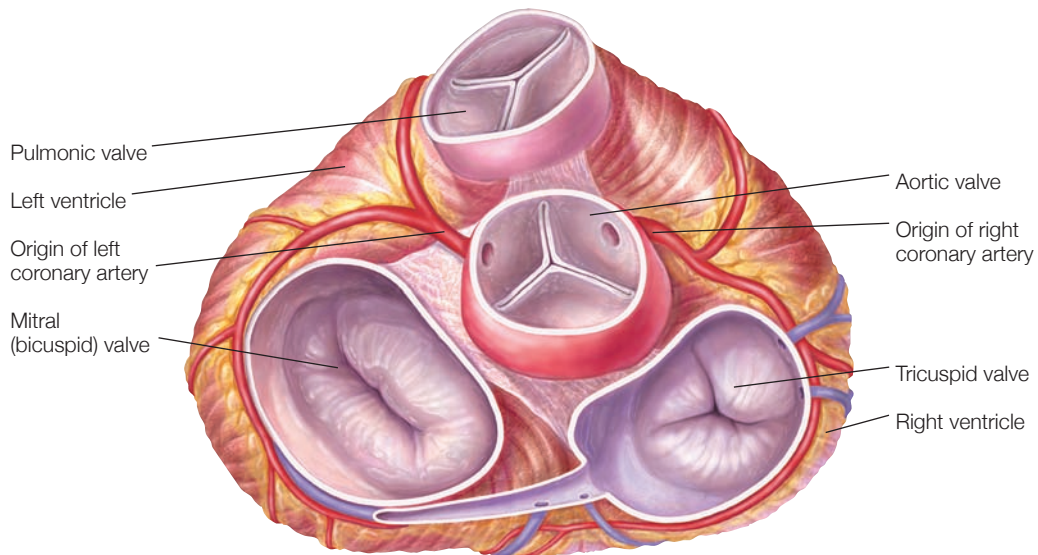
### Left-sided CHF

In left-sided CHF, blood and fluid back up into the lungs. The root cause of this is the left ventricle's inability to propel the blood forward. There are several factors that can lead to this condition; CAD is the most common. A lack of O<sub>2</sub>-carrying blood to the myocardial muscle leads to ischemia. If untreated, the ischemic area dies. The end result is weakened or dead heart muscle, which limits the ventricle's contracting ability.

## Inside a normal heart



Top view



### Causes

Uncontrolled hypertension is a leading cause of CHF. Whether primary (no known cause) or secondary (underlying causes such as too much salt and/or fluid in the body, chronic kidney disease, diabetes, and heart valve issues, to name a few), hypertension leads to CHF due to an overstretching of the myocardial fibers in the ventricle. Cardiomyopathy, in which the ventricular muscle becomes enlarged, thickened, or rigid, can contribute to CHF via the same mechanism. As with hypertension, the cause of cardiomyopathy can be either idiopathic (unknown) or a result of alcohol/drug misuse, heart/valve disease, hypertension, or a viral disease affecting the heart.

Heart dysrhythmias may also be the culprit. Atrial fibrillation and atrial flutter cause the atrium to contract suboptimally. This results in the loss of atrial kick, in which up to 30% of the atrium's volume doesn't get to the ventricles and isn't pumped out to the lungs and/or the aorta. Bradycardic rhythms, whether electrical/physiologic (sinus bradycardia, junctional, and AV blocks) or pharmacologic (digoxin, beta-blockers, and calcium-channel blockers in particular) in nature, are also suspects.

### Subclassifications

Left-sided CHF can further be broken down into two subdivisions: systolic and diastolic. Recall that in systole, the ventricles are contracting, which forces the blood forward into the system. In systolic CHF, the problem begins when the left ventricle undergoes an insult and both the cardiac output (CO) and BP decrease. Then neurohormonal activation occurs, including stimulation of the sympathetic nervous system, the renin-angiotensin-aldosterone-system, and the arginine vasopressin system. These mechanisms are all designed to increase both CO and BP. They do so, however, at the cost of increasing the amount of fluid in the bloodstream and the heart rate.

The Frank-Starling law shows that stroke volume increases with an increase of blood filling the heart, known as the end-diastolic filling volume. Over time, this leads to an "overstretched rubber band" syndrome in which the rubber band loses its "snap." The ventricle is unable to eject blood efficiently out to the aorta as it becomes overstretched. This leads to apoptosis, or programmed cell death, within the ventricle, as well as an actual remodeling of the ventricular shape itself. The end result is a decrease in blood ejected from the ventricle.

Diastole is the resting or relaxation phase of the cardiac cycle. In systolic CHF, the heart is unable to adequately pump blood out into the body. Diastolic CHF is the inability of the heart to properly refill after systole. This can be due to a "stiffer" ventricular wall, which then results in a lowered stroke volume and a decrease in CO. Less CO means less O<sub>2</sub> and nutrients to the body. Diastolic CHF has the same causes as systolic CHF.

### Symptoms

The symptoms of left-sided CHF are primarily pulmonary in nature. The backup of blood into the lungs results in increased hydrostatic pressures in the alveoli, which forces fluid into the alveolar sacs and prevents O<sub>2</sub>/CO<sub>2</sub> exchange. In extreme circumstances, this can lead to pink, frothy foam when the patient coughs. Other symptoms include orthopnea (shortness of breath when the patient is lying down) and paroxysmal nocturnal dyspnea (PND). Orthopnea is relieved by having the patient stand or sit up. The patient may complain of having to sleep with two or more pillows or sitting up in a chair. PND is the sensation of shortness of breath that suddenly awakens the patient in the night, often after 1 to 2 hours of sleep. Wheezing may also be noted. In some cases of mild diastolic CHF, the lung sounds may be normal.

Left-sided CHF symptoms are primarily pulmonary.



### Evaluation

The stethoscope is an important tool in evaluating left-sided CHF.  $S_1$  and  $S_2$  are the normal heart sounds that give the classic “lub-dub.” Two unique heart sounds may also be auscultated. Heard after the second heart sound, an  $S_3$  (sometimes called an  $S_3$  gallop) is best heard over the apex of the heart using the bell of the stethoscope. Also known as the third heart sound, it may be likened to the word “Kentucky” with the “y” being the  $S_3$ . Although not uncommon in younger individuals, an  $S_3$  can be indicative of a “floppy” ventricle seen in left-sided CHF. The  $S_3$  is the result of vibrations in the ventricular walls, resulting from the first rapid filling. (See “Heart Sounds: Hear the Story” on page 51.)

In the continuum of CHF, the ventricle can become stiffer. This is an end result of the enlarged ventricle and the aforementioned remodeling. As the ventricle becomes stiffer, the left atrium is forced to work harder to move blood into the left ventricle. The fourth heart sound,  $S_4$ , is heard during the second phase of ventricular filling just

before  $S_1$ . This results from the vibrations of the valves and ventricular walls as blood is forced into the ventricle. The sound is like that of the word “Tennessee,” with the “Ten” being the fourth sound. It, too, is best heard over the apex of the heart using the bell of the stethoscope.

Murmurs are indicators of faulty valves. Whether systolic or diastolic, their presence can give further credence to a diagnosis of CHF. Leaky valves can be the cause of retrograde blood flow, either by themselves or in conjunction with ventricular incompetence. Stenotic valve, which is a narrowing within the valve itself, can cause hypertrophy of the ventricle.

Auscultation of the lungs can detect adventitious breath sounds associated with CHF. Crackles are indicative of fluid build-up in the small airways, resulting from increased pressure in the alveoli. They can be heard on both inspiration and expiration. Crackles are described as being fine, medium, or coarse. The location of the crackles should always be included in discussions with the healthcare provider. Wheezes can be heard in patients with CHF. Also known as “cardiac asthma,” this is the result of fluid buildup in the smaller airways of the lungs. Wheezes can be heard on inspiration and expiration. The presence of wheezes from a cardiac origin is an indication of worsening CHF because the wheezing results from increasing fluid in the lungs.

### Right-sided CHF

Conversely, right-sided CHF exhibits little or no pulmonary involvement. Think of it this way: left CHF = lungs; right CHF = rest of the body. The definition—the inability of the heart to adequately pump—is the same. In this case, however, the blood backs up into the body. Right-sided CHF is a result of increased pressure in the lungs. Because the lung pressures are lower than the aorta, the right ventricle doesn’t need to pump as hard as the left. Conditions such as pulmonary hypertension, chronic obstructive

### $S_3$ : Classic sign of heart failure

$S_3$ , also known as *ventricular gallop*, is commonly heard in children and may be normal in patients during the last trimester of pregnancy; however, it may be a cardinal sign of heart failure in other adults. Because it follows  $S_2$ , it’s commonly compared to the “y” sound in “Ken-tuck-y.”

$S_3$  is low-pitched; you’ll hear it best at the apex when the patient is lying on his left side.

- In early ventricular diastole, the pulmonic and aortic valves snap closed, producing  $S_2$ .
- A large amount of blood rushes into the ventricles, possibly as a result of pulmonary edema, an atrial septal defect, or an acute MI.
- The rapid ventricular filling causes vibrations, producing  $S_3$ .



pulmonary disease, pulmonic valve stenosis, and chronic blood clots can lead to an enlargement of the right ventricle. The backup of fluid systemically leads to engorgement of the jugular veins and swelling in the abdomen/sacrum, lower legs, and feet.

Jugular venous distension is reflective of the retrograde flow seen in elevated right atrial pressures. The blood backs up through the superior vena cava into the jugular vein. With the patient lying at a less than 45-degree angle, the distended vein can be seen best over the sternocleidomastoid muscle.

Hepatomegaly, or liver enlargement, isn't in itself a disease, but rather a sign of one. In CHF, this is due to the backup of blood from the inferior vena cava to the portal blood system. An enlarged liver won't be seen, but may be felt by palpating the right upper abdominal quadrant just under the ribs as the patient takes in and lets out a deep breath. Ascites is excess fluid found in the peritoneal cavity. This, too, is a result of backflow through the inferior vena cava. It may or may not be associated with hepatomegaly. In bedridden patients, this excess fluid may also be seen pooling in the sacral area.

Edema is swelling, which can be observed from fluid accumulation in body tissue. It's most commonly seen in the feet, ankles, and legs. Edema can be divided into two categories: pitting and nonpitting. Nonpitting edema usually affects the legs and arms. Pressing on the affected area won't leave any trace behind. In contrast, pitting edema will leave a pit or dent in the skin. A grading scale of +1 to +4 is used. In general, +1 is mild, +2 is moderate, +3 is deep, and +4 is very deep pitting.

### A touch of class

There are two organizations that have classifications for CHF. The American College of Cardiology (ACC)/American Heart Association (AHA) classifies CHF as:

- **High risk of developing heart failure:** This includes hypertension, diabetes, CAD, and family history of cardiomyopathy.

- **Asymptomatic heart failure:** Candidates for this level are those with a previous history of myocardial infarction (MI), left ventricular dysfunction, and valvular heart disease.

- **Symptomatic heart failure:** These are patients with structural heart failure, dyspnea and fatigue, and impaired exercise tolerance.

- **Refractory end-stage heart failure:** These individuals have marked symptoms at rest despite maximal medical therapy.

Similarly, the New York Heart Association heart failure classification also has four classes:

- **Class I (mild):** No limitation of physical activity; ordinary physical activity doesn't cause tiredness, heart palpitations, or shortness of breath.

- **Class II (mild):** Slight limitation of physical activity; the patient is comfortable at rest, but ordinary activity causes tiredness, heart palpitations, and/or shortness of breath.

- **Class III (moderate):** Marked limitations of physical activity; the patient is comfortable at rest, but less than ordinary physical activity causes tiredness, heart palpitations, or shortness of breath.

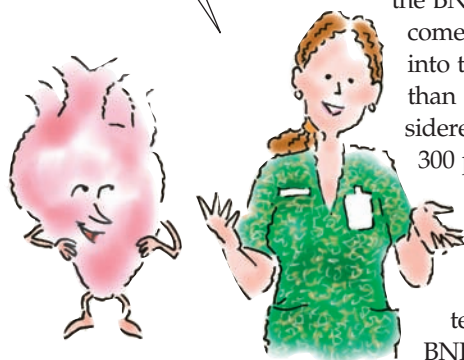
### S<sub>4</sub>: An MI aftereffect

Also called an *atrial gallop*, S<sub>4</sub> is an adventitious heart sound that you'll hear best over the tricuspid or mitral area when the patient lies on his left side. Patients who are elderly and those with hypertension, aortic stenosis, or a history of MI may have an S<sub>4</sub>. It's commonly described as sounding like "Ten-nes-see" because it occurs just before S<sub>1</sub>, after atrial contraction.

- In atrial diastole, the atria contract to eject blood into the ventricles.
- If the ventricles don't move or expand as much as they should, the atria must work harder to eject the blood. This causes the atria to vibrate, producing a sound known as S<sub>4</sub>.
- As the ventricles fill and pressure rises, the mitral and tricuspid valves snap close, producing S<sub>1</sub>.



The higher the BNP level, the more severe the heart failure.



- **Class IV (severe):** Severe limitation of physical activity; the patient is unable to carry out any physical activity without discomfort. Symptoms are present at rest, and any physical activity increases that discomfort.

### Diagnostic methods

A diagnosis of CHF is obtained by utilizing several methods. Blood is drawn to pinpoint the BNP level. As the ventricles become overstretched, BNP is released into the bloodstream. Levels of less than 100 png/mL are generally considered negative for CHF. At 100 to 300 png/mL, CHF is considered to be present. As the levels increase above 300 png/mL, the degree of CHF worsens. A similar lab test is for N-terminal-pro-BNP (NT-pro BNP). NT-proBNP levels rise in proportion to the severity of CHF.

Unlike BNP, it has been shown to be influenced by both the age and sex of the patient. Be aware of the type and parameters of BNP being used by the reporting lab to ascertain the meaning of the results.

Chest X-rays will be ordered to verify if there's any fluid in the lungs. They can also be used to determine if the heart is enlarged. An echocardiogram will also be ordered to visualize the walls of the heart as they contract, as well as the competence of the heart valves. An echocardiogram also measured the ventricle's ejection fraction (EF)—the percentage of blood ejected by the ventricle during systole. A normal EF is between 50% and 70%. A percentage of 40% to 50% may indicate some damage in the heart. An EF below 40% may indicate either CHF or cardiomyopathy.

Cardiac catheterization may also be helpful. Catheterizing the left side of the heart can be used to determine if there are any occlusions that may be causing the myocardium to become ischemic. Catheterizing the right side of the heart can be used to examine the fluid status and pressures within the heart to determine if right-sided CHF is present.

### Managing CHF

Treatment for CHF begins with lifestyle modifications. If the patient smokes, he or she should be encouraged to stop. Exercise should be encouraged if not contraindicated. Dietary modifications and fluid intake restrictions should be utilized.

### Medications and procedures

The proper medications are an important component of CHF treatment. They include inotropic agents, both oral and I.V., to improve the contraction strength of the ventricles, resulting in improved CO. Beta-blockers slow the heart rate and lower BP, lessening the strain on the heart that comes from sympathetic nervous system stimulation. In fact, studies have linked certain beta-blockers to the suppression of apoptosis and stabilization of the mitochondrial walls in the epithelial cells.

Angiotensin-converting enzyme (ACE) inhibitors and angiotensin II receptor blockers (ARBs) are also key medications for CHF. Whether inhibiting the conversion of angiotensin I to angiotensin II (ACE inhibitors) or blocking the angiotensin II receptors in the blood vessels (ARBs), the end result is the same. The blood vessels in the body are dilated, which reduces BP and decreases afterload of the ventricles. ACE inhibitors can sometimes lead to a chronic cough, which is one reason why ARBs may be prescribed instead. Along the same lines, human B-type natriuretic peptides can be used I.V. to dilate the blood vessels. Care should be taken when assessing BNP levels because this medication is actually synthetic BNP, which will falsely elevate the BNP level.

Diuretics play a major role in CHF treatment. Diuretics act within the kidney to promote increased urination. The use of these medications can cause impressive urinary amounts and drastic weight loss in the first few days of therapy. One liter of urine output is equal to 1 kg of weight lost. Examples of diuretics include loop diuretics, thiazides, and carbonic anhydrase inhibitors. These

medications may cause depleted potassium, a critical electrolyte within the body. In such cases, potassium-sparing diuretics may be ordered. These are broken down into two classes: aldosterone antagonists and epithelial sodium channel blockers. Regardless of the type of diuretic used, lab values such as blood urea nitrogen, creatinine, sodium, and potassium should be monitored closely.

Precisely because the kidney can become overstressed during diuresis, a procedure called aquapheresis was developed. Using the same principle as dialysis, aquapheresis (or ultrafiltration) uses a catheter to remove excess fluid from the body. The blood is then returned to the body via another catheter. This process can remove up to 500 mL or 1.1 kg of water weight per hour without the use of diuretics. This can be done as an outpatient (for 3 days/week) or as an inpatient (for 24 to 36 hours).

Patients who have either atrial fibrillation or atrial flutter may be candidates for cardioversion or ablation. Cardioversion is the delivery of an electric shock to the heart that attempts to reset the heartbeat to sinus rhythm. Ablation calls for a special device that delivers energy to the area of the heart or conduction system that's responsible for the arrhythmia.

### Devices and surgery

Medical device implantation is another option. Electrically induced bradycardia patients may benefit from a pacemaker. Healthcare providers of patients at risk for lethal arrhythmias as a result of severe left ventricular failure or cardiomyopathy may suggest placing an implantable cardioverter defibrillator (ICD). In addition to functioning as a pacemaker, ICDs are able to perform antitachycardia pacing and internal cardioversion/defibrillation in the presence of lethal ventricular dysrhythmias.

CHF can lead to asynchronous contractions between the right and left ventricles. In these patients, this leads to an even greater decrease in CO. Cardiac resynchronization

## Classifying CHF

cheat

sheet

### The ACC/AHA classification

- **High risk of developing heart failure:** Hypertension, diabetes, CAD, family history of cardiomyopathy
- **Asymptomatic heart failure:** Previous history of MI, left ventricular dysfunction, valvular heart disease
- **Symptomatic heart failure:** Structural heart failure, dyspnea and fatigue, impaired exercise tolerance
- **Refractory end-stage heart failure:** Marked symptoms at rest despite maximal medical therapy

### The New York Heart Association classification

- **Class I (mild):** No limitation of physical activity; ordinary physical activity doesn't cause tiredness, heart palpitations, or shortness of breath
- **Class II (mild):** Slight limitation of physical activity; the patient is comfortable at rest, but ordinary activity causes tiredness, heart palpitations, and/or shortness of breath
- **Class III (moderate):** Marked limitations of physical activity; the patient is comfortable at rest, but less than ordinary physical activity causes tiredness, heart palpitations, or shortness of breath
- **Class IV (severe):** Severe limitation of physical activity; the patient is unable to carry out any physical activity without discomfort; symptoms are present at rest, and any physical activity increases that discomfort

therapy, also known as biventricular pacing, can be beneficial. This leads to a more harmonious relationship between the ventricles, improving both CO and EF.

Another type of medical device is the ventricular assist device (VAD). This can be implanted in either ventricle and helps pump blood out into the body. VADs can be used as a bridge to a heart transplant or as the functioning part of the heart if the patient isn't a transplant candidate.

Surgery may also be an option. Valve replacement/repair may be justified in the presence of incompetent or stenotic valves. Coronary bypass surgery may be an option if it's felt that the myocardium may benefit from revascularization. Heart transplantation can also be considered.

In the very last stages of CHF, palliative care and/or hospice might be utilized. Palliative care allows the patient options to remain comfortable for as long as possible. Hospice is able to help patients and their families as they transition through end-of-life

care and, for the families, the aftermath of losing their loved one.

### Patient teaching pearls

After diagnosis, CHF is often a lifelong process. Patients, as well as their families, must be educated on not only the disease process, but also the modifications and lifestyle changes that they'll experience. This education should be initiated in the healthcare provider's office or in the hospital if the patient is admitted. A brief description of the heart and its function should be given, as well as a simple, yet clear, definition of CHF. The patient/family should be made aware of the causes and symptoms associated with CHF. The medications that have been prescribed should be reviewed, as well as their functions and adverse reactions. Of course, plenty of time should be available to allow patients and their families to ask questions.

The Joint Commission has set up specific discharge guidelines for post-hospitalization CHF patients. Evaluation of left ventricular systolic function must be performed. Adult smoking cessation advice/counseling needs to be addressed. Discharge instructions must be given to each patient, including the prescription of either an ACE inhibitor or ARB medication or the documented reasons for not doing so.

Discharge instructions should be clear and concise. Beta-blockers should be strongly considered in conjunction with the ACE inhibitor/ARB. Patients should be instructed on proper diet and fluid parameters. Patients are placed on a fluid restriction of no more than 2 L of fluid/day. One suggestion to help patients get used to this restriction might be to fill up an empty 2 L soda bottle with water in the morning. Whenever fluid is to be consumed, first pour an equal amount of the water into the drinking container, empty it, and then fill it with the beverage. In this manner, patients and their families will have a clear idea of how much fluid is left for the day.

Patients should also be encouraged to follow a low-sodium diet, typically totaling no more than 2 g of sodium/day. Sample menus can be sent home with the patient, although a good rule of thumb is eliminating table salt. Salt leads to fluid retention, which can lead to increased BP and more stress on the heart. Using herbal seasonings rather than salt substitute may be beneficial. Salt substitute is often potassium chloride, so the potential for hyperkalemia is present. Patients and their families should be encouraged to become label readers to eliminate as much sodium from the diet as possible.

If hospitalized, the patient should expect to be weighed every morning. This practice should be continued at home. Patients should be alert for any changes in weight and record them in a daily log. A weight gain of 1 to 2 lb/day or 4 to 5 lb/week may be indicative of increasing fluid retention. If this occurs, the patient should alert his or her healthcare provider or outpatient clinic for instructions. The patient may then be instructed to take extra doses of the diuretic, if prescribed, to prevent further retention, as well as dealing with the current overload.

Lastly, patients should follow up with their healthcare provider, whether a primary care provider, cardiologist, or CHF clinic. In doing so, both patients and their healthcare providers can keep tabs on progress made, as well as keeping alert for any potential problems. Hospital readmission for CHF is high: 24.7% in the first 30 days, 27% in the first 6 months, and 35% within the first year. (See the online exclusive "Heart Failure Readmissions: Can Hospital Care Make a Difference?" at <http://www.NursingMadeIncrediblyEasy.com>.)

### Teamwork for success

CHF is a complex disease and a lifelong process requiring diligence and discipline. It isn't unusual for a CHF patient to be seen in the ED after eating a piece of pizza. Patients may need hospitalization if they run out of their medication for a few days.

Follow The Joint Commission discharge guidelines for CHF patients.



Patient involvement, education, and outpatient care are key principles in managing CHF outside the hospital. Prompt diagnosis and timely interventions are core inpatient tactics. The alliance between the patient and healthcare provider outside the hospital, and the patient, nurse, and healthcare provider during admissions, must be strong to facilitate proper care of the CHF patient. ■

### Learn more about it

Cuculich PS, Joseph S. Cardiac resynchronization therapy: What? Who? When? How? *Am J Med.* 2011;124(9):813-815.

Family Practice Notebook. Congestive heart failure. <http://fpnotebook.com/CV/CHF/CngstvHrtFlr.htm>.

Lemond L, Allen LA. Palliative care and hospice in advanced heart failure. *Prog Cardiovasc Dis.* 2011;54(2):168-178.

Liao L, Anstrom KJ, Gottdiener JS, et al. Long-term costs and resource use in elderly participants with congestive heart failure in the Cardiovascular Health Study. *Am Heart J.* 2007;153(2):245-252.

Mayo Clinic. Heart failure. <http://www.mayoclinic.com/health/heart-failure/DS00061>.

MedlinePlus. Heart failure. <http://www.nlm.nih.gov/medlineplus/ency/article/000158.htm>.

Optimal Heart Health. <http://www.optimal-heart-health.com>.

Sabbah HN, Sharov VG, Gupta RC, Todor A, Singh V, Goldstein S. Chronic therapy with metoprolol attenuates cardiomyocyte apoptosis in dogs with heart failure. *J Am Coll Cardiol.* 2000;36(5):1698-1705.

Torpy JM, Lynn C, Glass RM. JAMA patient page. Heart failure. *JAMA.* 2009;301(18):1950.

The author and planners have disclosed that they have no financial relationships related to this article.

DOI-10.1097/01.NME.0000411090.01371.77

Need more CE? You got it!



For more than 40 additional continuing education articles related to cardiovascular topics, go to [Nursingcenter.com/CE](http://Nursingcenter.com/CE).

## CE CONNECTION

Earn CE credit online:

Go to <http://www.nursingcenter.com/CE/nmie> and receive a certificate *within minutes*.

### INSTRUCTIONS

#### Congestive heart failure: An 'F' isn't an option

##### TEST INSTRUCTIONS

- To take the test online, go to our secure Web site at <http://www.nursingcenter.com/CE/nmie>.
- On the print form, record your answers in the test answer section of the CE enrollment form on page 55. Each question has only one correct answer. You may make copies of these forms.
- Complete the registration information and course evaluation. Mail the completed form and registration fee of \$24.95 to: Lippincott Williams & Wilkins, CE Group, 2710 Yorktowne Blvd., Brick, NJ 08723. We will mail your certificate in 4 to 6 weeks. For faster service, include a fax number and we will fax your certificate within 2 business days of receiving your enrollment form.
- You will receive your CE certificate of earned contact hours and an answer key to review your results. There is no minimum passing grade.
- Registration deadline is April 30, 2014.

##### DISCOUNTS and CUSTOMER SERVICE

- Send two or more tests in any nursing journal published by Lippincott Williams & Wilkins together by mail and deduct \$0.95 from the price of each test.
- We also offer CE accounts for hospitals and other health care facilities on nursingcenter.com. Call 1-800-787-8985 for details.

##### PROVIDER ACCREDITATION

Lippincott Williams & Wilkins, publisher of *Nursing made Incredibly Easy!*, will award 2.5 contact hours for this continuing education activity.

Lippincott Williams & Wilkins is accredited as a provider of continuing nursing education by the American Nurses Credentialing Center's Commission on Accreditation.

This activity is also provider approved by the California Board of Registered Nursing, Provider Number CEP 11749 for 2.5 contact hours. Lippincott Williams & Wilkins is also an approved provider of continuing nursing education by the District of Columbia and Florida #FBN2454.

Your certificate is valid in all states.

The ANCC's accreditation status of Lippincott Williams & Wilkins Department of Continuing Education refers only to its continuing nursing educational activities and does not imply Commission on Accreditation approval or endorsement of any commercial product.