Managing chronic
The body depends on the heart’s ability to circulate blood. When the heart is damaged by illness or injury, the body marshals all of its forces to make up for the loss of function. This article examines what happens when heart failure overcomes the body’s ability to compensate, and offers insights on treatments that can help fix the problem.

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BEA SINGER, age 67, awakens with a start from a sound sleep, gasping. She sits up, trying to catch her breath, and shakes her husband awake. “Harry! Fix my pillows!” Just as he has every night for the past week, he fetches three pillows and arranges them behind her back to prop her up. Then he puts a finger under her chin and looks hard into her frightened eyes. “That’s it, Bea. We’re going to the hospital.”

He’d said it before, and each time she’d pooh-poohed the idea, but tonight it’s different. She can’t catch her breath even sitting up, so she lets him help her into her coat and out to the car. She’s so tired she just wants to sit down.

When she’s examined at the local hospital’s emergency department (ED), Mrs. Singer’s medical history is positive for an anterior wall myocardial infarction (MI) 5 years before. She’s been under treatment for the past 2 years for New York Heart Association (NYHA) Class II heart failure. Her history is negative for smoking, reactive airway disease, cancer, renal insufficiency, and diabetes. Her father died of an MI when he was 65; her mother died of breast cancer 20 years ago at age 66.

So what’s behind Mrs. Singer’s increasing breathlessness?

Winding down
Mrs. Singer presented at the ED with the cardinal signs of heart failure: dyspnea, fatigue, and fluid retention. She’s among the growing number of patients diagnosed with the condition, which is fast becoming one of our most important health care issues. Hospitalizations for heart failure are increasing, in part due to incomplete treatment during hospitalization, poor application of chronic heart failure management guidelines, and patient nonadherence to...
treatment recommendations. And when you consider that millions of baby boomers are now in their 50s and 60s, and more people are surviving longer after a heart attack, it’s easy to see why in the next decade, the number of individuals with heart failure will continue to grow rapidly (see Heart failure by the numbers).

According to the National Institutes of Health, about 20% of patients with heart failure will die within a year of diagnosis; two-thirds will die within 5 years. The American Heart Association (AHA) says that 80% of men and 70% of women under age 65 with heart failure will die within 8 years. Quality of life is adversely affected; patients with heart failure are at increased risk for depression.

In this article, I’ll discuss what’s being done to improve these outcomes. But first let’s see what happens to the body in heart failure.

Get me some air!
In heart failure, the heart can’t supply sufficient quantities of blood and oxygen to meet the metabolic needs of the body’s tissues. It begins with some type of injury to the myocardium that impairs the ability of the ventricles to fill with or to eject blood. This injury may result in dilatation and/or hypertrophy of one or both ventricles, a process called remodeling. Remodeling of the ventricles progresses over time; the patient may remain asymptomatic for months or even years. Inevitably, however, signs and symptoms will appear and worsen without treatment.

There are different types of heart failure. In low output heart failure, the left ventricle can’t eject a normal volume of blood due to weakness of the left ventricular muscle secondary to MI or cardiomyopathy, low flow coming from an impaired right ventricle, or aortic valve stenosis. Causes of impaired right ventricular function include MI or an extrinsic insult like pulmonary hypertension.

High output heart failure occurs when the volume of blood exceeds what the left ventricle can eject. The cardiac output remains high for a while, until the pressure of the high blood volume causes dilatation of the ventricle and a backup of fluid into the pulmonary interstitial spaces. Mitral valve regurgitation, aortic valve insufficiency, hyperthyroidism, anemia, and hypervolemia from an extrinsic cause (such as excess intravenous [I.V.] fluid administration) are associated with high-output heart failure.

Biventricular failure can occur when back-up pressures from the failing left ventricle result in dilatation and failure in the right ventricle.

Another important distinction in the classification of heart failure involves systolic dysfunction versus diastolic dysfunction. Left ventricular systolic dysfunction occurs when the heart muscle is too weak to contract fully. As a result, a reduced volume of blood leaves the ventricles during systolic ejection. In normal heart function, 60% to 80% of the blood contained in the left ventricle is ejected with each beat; this is called the ejection fraction.

Left ventricular diastolic dysfunction occurs when the left ventricle has increased diastolic stiffness (reduced compliance) and can’t fill adequately at normal diastolic pressures. The elevated pressures needed for filling cause signs and symptoms of pulmonary congestion. A person with left ventricular diastolic dysfunction typically has a normal

Heart failure by the numbers
In the United States, there are about 5 million people with heart failure, and more than 550,000 new cases are diagnosed each year, according to the American College of Cardiology/American Heart Association. Heart failure prompts 12 to 15 million office visits and 6.5 million hospital days each year. During the last 10 years, the annual number of hospitalizations for heart failure as a primary diagnosis has increased to over 1 million, at a cost to Medicare and third-party payers of about $24.3 billion. Around 55,000 Americans are predicted to die of heart failure in 2006.
or high ejection fraction. Because the contractility of the muscle isn’t impaired, the normal amount of blood is ejected from the ventricle with each beat. Long-standing untreated systemic hypertension causes cardiac muscle hypertrophy, which can lead to this type of diastolic dysfunction.

Coronary artery disease is responsible for about two-thirds of all cases of heart failure. The remainder of cases are characterized by nonischemic cardiomyopathy. Among the many causes of nonischemic cardiomyopathy are systemic hypertension, thyroid dysfunction, valvular heart disease, and exposure to cardiotoxic substances like alcohol, cocaine, or chemotherapeutic agents. Frequently, the cause is unknown; this is referred to as idiopathic dilated cardiomyopathy.

**On stage**

Heart failure is a progressive disease. A widely used staging system that emphasizes the development and progression of the disease was jointly developed by the American College of Cardiology (ACC) and the AHA. It classifies patients as follows:

- **Stage A** identifies patients at risk of developing heart failure who don’t have any structural heart disease or symptoms of heart failure. Some types of patients whom you may see with Stage A heart failure include those with diabetes, coronary artery disease, or hypertension.
- **Stage B** describes patients with documented structural changes who don’t yet have signs or symptoms of heart failure. Some types of patients whom you may see with Stage B heart failure are those with a history of MI, those with valve regurgitation on echocardiogram, or those with left ventricular hypertrophy on a 12-lead electrocardiogram (ECG). These patients have a documented structural change to their heart, but they don’t yet demonstrate symptoms of heart failure.
- **Stage C** refers to patients who have structural changes and who have or have had symptoms.
- **Stage D** describes patients with refractory heart failure who may need mechanical or pharmaceutical support, a heart transplant, or end-of-life care.

Patients classified as Stage A, although asymptomatic, are treated to reduce their risk for developing heart failure; the goal is to help them achieve longer survival with better quality.

Next, I’ll explain the NYHA functional classification system.

**New York, New York**

The ACC/AHA guideline staging system was developed to complement the NYHA functional classification system, which primarily gauges the severity of symptoms.

- **Class I:** Ordinary physical activity doesn’t cause undue fatigue, dyspnea, or anginal pain.
- **Class II:** The patient has slight limitation of physical activity but is asymptomatic at rest. Ordinary physical activity causes fatigue, palpitations, dyspnea, or anginal pain.
- **Class III:** The patient has marked limitation of physical activity but is typically asymptomatic at rest. Less than ordinary physical activity causes fatigue, palpitations, dyspnea, or anginal pain.
- **Class IV:** The patient can’t perform any level of physical activity without discomfort; symptoms may be present at rest. Discomfort increases with physical activity. This patient will be considered for mechanical support, continuous pharmaceutical support, transplant, or end-of-life care.

Now, let’s see how the heart tries to make up for its inability to pump up to par in left ventricular systolic dysfunction.

**Release the vasoconstrictors!**

Compensated heart failure occurs when the heart is injured (such as by an MI) or
becomes diseased (such as with viral myocarditis). The body launches several compensatory mechanisms to maximize the functioning of the heart, which has begun to pump less efficiently. One of these mechanisms stimulates the sympathetic nervous system to release norepinephrine and epinephrine, increasing the heart rate and thus improving cardiac output. Usually, anytime the heart rate is increased, cardiac output will also increase, according to the equation

$$\text{cardiac output} = \text{stroke volume} \times \text{heart rate}.$$ 

Another compensatory mechanism is the activation of the renin-angiotensin-aldosterone system that occurs when kidney perfusion decreases. Renin converts angiotensinogen to angiotensin I, which is converted into angiotensin II in the lungs by angiotensin-converting enzyme (ACE). Angiotensin II is a powerful vasoconstrictor. This mechanism increases fluid volume and maintains blood pressure. The rise in blood pressure causes increased resistance (or increased afterload) against ventricular ejection, eventually causing ventricular hypertrophy. Aldosterone increases the reabsorption of sodium and water in the kidneys, which in turn raises blood pressure.

The increase in angiotensin II also stimulates the release of vasopressin, or antidiuretic hormone, from the posterior pituitary gland. Vasopressin is another powerful vasoconstrictor, and it promotes renal release of renin. It also prevents diuresis by altering receptors in the collecting ducts of the kidneys.

Endothelin 1, produced in endothelial vascular smooth muscle cells in neurons and in endometrial cells, acts as a modulator of vasomotor tone, cell proliferation, and hormone production. Its release is stimulated by shear stress and the presence of angiotensin II, vasopressin, and epinephrine. And—you guessed it—it’s yet another potent vasoconstrictor; it stimulates growth of myocytes (beating heart cells) in heart failure.

Angiotensin II stimulates the adrenal gland to secrete aldosterone, which acts to retain both sodium and water. This results in compensatory changes in the structure of the heart. Both the ventricles and the atria begin to dilate in response to the excess fluid volume. According to the Frank-Starling law of the heart, the increased volume (increased preload) results in greater contractility and greater cardiac output.

A fourth mechanism to boost low cardiac output is to increase the stroke volume by increasing the amount of water in the bloodstream. Stroke volume, simply put, is the amount of blood pumped by the left ventricle in a single contraction.

The final compensatory mechanism is the release of two amino acid peptides: human atrial natriuretic peptide (hANP) and human brain natriuretic peptide (hBNP). hANP is released by stretch receptors in the atria, and hBNP is released by stretch receptors in the ventricles, both in response to excess blood volume. Their purpose is to cause a loss of sodium and water via the kidneys; hBNP also provides balanced vasodilatation of veins, arteries, and coronary arteries, which reduces the blood pressure and improves blood flow through the coronary arteries. Human BNP reduces aldosterone levels, which helps to promote sodium and water excretion.

When a patient is in compensated heart failure up to a point, the body can compensate for the heart's failings.

Helping hands on the Web

The American Heart Association recommends the following organizations to help caregivers and individuals cope with chronic heart failure:

- The Mended Hearts, Inc.:
  http://www.mendedhearts.org
- Heartmates:
  http://www.heartmates.com
- National Family Caregivers Association:
  http://www.nfcacares.org
- The Well Spouse Association:
failure, all of the aforementioned mechanisms work together in a balanced manner to maximize cardiac output. They act to prevent signs and symptoms of decompensation.

Let’s examine next what happens when heart failure overpowers the compensatory mechanisms.

**No safety net**
Decompensated heart failure is a loss of balance between the mechanisms of heart failure and the body’s attempts to overcome the failure process. As a result, the patient begins to show the signs and symptoms of heart failure. Heart failure can be left-sided, right-sided, or both.

Left-sided heart failure can be recognized by crackles in the posterior lung fields due to excessive preload. The patient will be tachycardic, dyspneic, and tachypneic. She may have a low SpO₂ via pulse oximetry and complain of having to lie propped up to sleep. She may give a history of waking with paroxysmal nocturnal dyspnea. Auscultation may reveal an S₃/S₄ gallop.

Also, low blood flow through the mesenteric arteries may cause symptoms of reduced blood flow to the gastrointestinal tract, including anorexia, nausea, bloating, and constipation. Low blood flow through the renal artery causes reduced urinary output. Vasodilatation from angiotensin II and vasopressin may cause cool or cold, pale, and possibly cyanotic extremities.

Right-sided heart failure is expressed as signs and symptoms caused by excessive preload on the right side of the heart: jugular venous distention, liver engorgement, ascites, and peripheral edema. Both left-sided and right-sided heart failure are associated with an increase in weight.

These signs and symptoms are suggestive of heart failure. Now let’s see how the diagnosis is made.

**Hear that?**
The most commonly used and useful diagnostic test for heart failure is the transthoracic echocardiogram. It provides the most information about the structure and function of the heart. Coupled with Doppler flow studies, it can show atrial and/or ventricular hypertrophy, valve problems, whether the problem is inside the heart or in the pericardium, and when the problem occurs (during systole or diastole). The echocardiogram can compute ejection fraction and determine whether any of the heart walls are failing to contract normally.

Other helpful tests include 12-lead ECG, chest X-ray, radionuclide ventriculography, and magnetic resonance imaging or computed tomography. The 12-lead ECG and chest X-ray alone don’t provide adequate information to make a reliable diagnosis.

A left heart catheterization with a ventriculogram can also provide important information about the patency of the coronary arteries, as well as structure and function of the left ventricle. The procedure is invasive, however, requiring injection of a contrast medium into the heart. The contrast medium is excreted through the kidneys, which can cause problems in certain patients with impaired renal function. Some patients may have an adverse reaction to the contrast medium.

A right heart catheterization is useful for measuring the pressures in the right side of the heart. This test is also invasive. It’s used

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**Signs and symptoms checklist**

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<thead>
<tr>
<th>Left-sided heart failure</th>
<th>Right-sided heart failure</th>
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<tr>
<td>Crackles (posterior lung fields)</td>
<td>Jugular venous distention</td>
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<tr>
<td>Tachycardia</td>
<td>Liver engorgement</td>
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<td>Dyspnea</td>
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<td>Tachypnea</td>
<td>Peripheral edema</td>
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<tr>
<td>Low SpO₂</td>
<td>Weight gain</td>
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mainly to evaluate the degree of heart failure and to guide therapies when the presence of moderate to severe heart failure is known or suspected.

Once heart failure is diagnosed, treatment begins. Let’s look at what’s available.

Battling back

Treatment is designed to restore the balance between compensatory and decompensatory mechanisms. Here’s the equation again: cardiac output = stroke volume \times heart rate. Let’s look at the treatment options for the three components of stroke volume, one at a time.

• **Preload**—Recall that preload is the amount of fluid in the ventricles at the end of diastole. The first line of treatment for heart failure, therefore, is to reduce the preload, typically with a diuretic. That usually means I.V. furosemide (Lasix), which provides a relatively quick diuresis. It’s especially useful if the patient is in respiratory distress from left-sided heart failure.

ACE inhibitors are also used to reduce preload. They act as vasodilators, and they promote excretion of sodium and water through the kidneys.

Nesiritide (Natrecor), a synthetic BNP, mimics the actions of endogenous natriuretic peptides. It promotes diuresis by causing the kidneys to excrete large amounts of sodium and water. It works with furosemide to cause rapid diuresis to decrease preload.

• **Afterload**—As the body tries to compensate for heart failure by raising the blood pressure through the renin-angiotensin-aldosterone system, afterload may become too high. Because ACE inhibitors are vasodilators, they’re used to reduce both afterload and preload. Beta-blockers, nitrates, and hydralazine may also be used to promote vasodilation.

Nesiritide causes balanced arterial and venous dilatation, thus lowering systemic blood pressure. The left ventricle is better able to eject the blood when the blood pressure comes down, which increases cardiac output.

Nesiritide has also been shown to reduce serum levels of norepinephrine and aldosterone, indicating that it can blunt activation of the renin-angiotensin-aldosterone system in heart failure. Metoprolol (Lopressor), a beta-blocker, and carvedilol, a combination of alpha- and beta-blockers, also slow activation of the renin-angiotensin-aldosterone system.

Spironolactone (Aldactone) acts as a potassium-sparing diuretic and an aldosterone antagonist; it reverses sodium and water retention. Patients given spironolactone and an ACE inhibitor have been shown to have better ventricular function and exercise tolerance than those given an ACE inhibitor alone.

Eplerenone (Inspra), another aldosterone antagonist, is used for patients who develop heart failure following an MI.

• **Contractility**—If the heart’s contractility is strengthened, cardiac output improves. Digoxin (Lanoxin), a digitalis glycoside, increases the strength of contractions and slows the heart rate. Although once the cornerstone of heart failure treatment, digoxin is now used less often and at lower doses.

Currently, research is under way to test an implantable device to improve cardiac contractility. The device delivers a small amount of electricity, similar to a pacemaker, to open calcium channels in the heart muscle cells. The theory behind the device is that by making extra calcium available, the myofibrils will improve the strength of their contractions.
Levosimendan, a calcium sensitizer, is in a Phase III clinical trial designed to study its effects on patients with class III or IV heart failure. Besides providing vasodilation, it improves contractility of the heart during systole and allows more normal relaxation during diastole.

Now, let’s see what your role is in caring for patients with heart failure.

**Catch your breath**

Remember, your first considerations in caring for a patient in heart failure are related to affecting preload adjustments. You need to begin by assessing the patient’s clinical signs and symptoms. Mrs. Singer, you’ll recall, came into the ED with dyspnea, tachycardia, and tachypnea. Auscultation revealed crackles in the posterior lung fields bilaterally and an S3 gallop. These are signs of increased left ventricular preload. Mrs. Singer also displayed neck vein distention, liver engorgement, and pedal edema, which reflect an increase in right ventricular preload.

Once you take vital signs on a patient like Mrs. Singer and give her supplemental oxygen, you should weigh her. This baseline weight is vitally important in evaluating the effectiveness of therapies.

**The ins and outs of the heart...**

To better understand stroke volume, think of the heart as a balloon that’s constantly being inflated and deflated.

**Preload: Inflation**

*Preload* is the stretching of muscle fibers in the cardiac ventricles. It reflects the end-diastolic volume, which is influenced by diastolic pressure and the composition of the myocardial wall. The Frank-Starling principle states that the degree of preload within a physiologic range is proportional to the systolic performance of the ensuing ventricular contraction. Because ventricular function is abnormal in heart failure, the response is inadequate.

**Contractility: It’s a stretch**

*Contractility* refers to the inherent ability of the myocardium to contract normally. It’s influenced by preload: the greater the preload, the more forceful the contraction of the heart muscle. Contractility is characterized by the force and velocity of contraction; it’s often expressed as the ejection fraction (left ventricular stroke volume/endo-diastolic volume).

**Afterload: Deflation**

*Afterload* refers to the pressure that the ventricular muscles need to exert to overcome the higher pressure in the aorta to move blood out of the heart. *Resistance* is the force the heart muscle must counteract. Afterload is determined by ventricular pressure, blood volume in the chamber, and wall thickness at the time of the aortic valve opening.
Medical therapies that are implemented early on may include an I.V. bolus dose of a diuretic, followed perhaps by a continuous infusion of diuretics, along with administration of an ACE inhibitor and/or a beta-blocker, as prescribed.

Position the patient for comfort. Generally, the semi-Fowler’s position or seated on the edge of the bed with her arms resting on the over-bed table works well. Advise the patient that her daily fluid intake is limited to 1 to 2 liters. If she has a dry mouth, she can relieve it by brushing her teeth and rinsing her mouth with water or mouthwash. Also, tell her that it’ll be important for her nurses to keep accurate records of intake and output to evaluate the success of her therapy. During her hospital stay, she’ll likely be put on a 2 g/day sodium diet and given recommendations for dietary restrictions after discharge.

Next, we’ll look at interventions related to the effect of increased afterload.

**Take a load off**

To increase cardiac output in heart failure, afterload needs to be lowered to improve stroke volume of the impaired left ventricle. Remember, afterload is the amount of resistance the heart has to overcome to eject blood from the ventricle. It’s influenced by the volume and mass of blood ejected, the size and wall thickness of the ventricle, and the impedance of the vasculature.

Administration of vasodilators (nesiritide, ACE inhibitors, beta-blockers, or hydralazine plus nitrates) is effective, and doses can be adjusted according to the measurements from the right heart catheter. How can you tell if the drug therapy is succeeding? Here’s one indicator: pulse pressure, the difference between systolic and diastolic blood pressures. The narrower the pulse pressure, the greater the afterload. A widening pulse pressure is associated with a decrease in afterload.

Whether you use pulse pressure or not, it’s important to monitor blood pressure closely with these drugs. The best blood pressure is one that’s as low as the patient can tolerate without getting dizzy. Continue giving the vasodilator, even if the systolic pressure is only in the 90s. Withholding the vasodilator will just allow the afterload to increase, causing cardiac output to fall. If the systolic pressure falls below 90 and the patient becomes dizzy, however, therapy may need to be adjusted. In the case of nesiritide, for example, the infusion should be stopped until the systolic pressure rises over 90. Then, the infusion can be resumed at 30% of the previous rate; no bolus is given when the infusion is resumed.

**Mrs. Singer’s progress**

To evaluate the effect of her therapy, Mrs. Singer’s nurse in the cardiac care unit (CCU) takes her vital signs again and does another physical assessment. Mrs. Singer’s heart rate is now below 100 and her respiratory rate is at 22. Within about 3 hours of being treated with I.V. furosemide in the ED, Mrs. Singer states that she’s less short of breath. She also received a bolus and infusion of nesiritide in the ED before being transferred to the CCU. Her blood pressure is now 98/56; she denies feeling dizzy. Her diastolic pressure indicates the wonderful afterload reduction achieved by the combination of therapies.

The crackles in Mrs. Singer’s lung fields have decreased to fine bibasilar crackles. During the next couple of days, her jugular venous distention and edema are reduced. She’s producing more urine than liquid taken by mouth, and her daily weights confirm this: Her 24-hour intake and output level is 2 L negative, and she’s 4.4 pounds lighter.

Now that Mrs. Singer’s feeling better, it’s time to start teaching her about better management of heart failure. Patient teaching should include medications, fluid management, low-sodium diet, daily weights, and the importance of regular exercise. A patient
who smokes should be advised to stop. Mrs. Singer should also be told to avoid using nonsteroidal anti-inflammatory drugs for pain because they may cause sodium retention and myocardial depression. Taking medications as prescribed and following strict fluid-management and sodium-reduction plans after discharge will greatly reduce the number of readmissions for recurrence of symptoms.

Stress the importance of notifying the primary health provider or cardiologist immediately after a gain of 2 pounds overnight or 5 pounds in a week, or after awakening suddenly with shortness of breath.

Smooth sailing
Heart failure is a major and growing health care issue due to an aging population, improved survival after an MI, and improvements in the management of chronic heart failure. Nursing care is vitally important in all health care settings. It’s our job as nurses to evaluate our patients’ responses to all therapies. A good understanding of preload and afterload can help you to make the best clinical decisions when managing your patients with heart failure.

Because of the prompt and effective treatment Mrs. Singer received, she and Harry will be able to go on that Caribbean cruise they’ve been looking forward to for so long!

Learn more about it

INSTRUCTIONS
Too pooped to pump: Managing chronic heart failure

TEST INSTRUCTIONS
• To take the test online, go to our secure Web site at www.nursingcenter.com/ce/nmie.
• On the print form, record your answers in the test answer section of the CE enrollment form on page 52. 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 $19.95 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. Deadline is February 29, 2008.
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Too pooped to pump: Managing chronic heart failure

GENERAL PURPOSE: To familiarize the registered professional nurse with the pathophysiology of heart failure and appropriate treatment and nursing care for the patient. LEARNING OBJECTIVES: After reading the article and taking this test, you should be able to: 1. Describe the pathophysiology and signs and symptoms of heart failure. 2. Discuss the diagnosis and treatment of heart failure.

1. Which of the following are considered the cardinal signs of heart failure?
   a. shortness of breath, hypotension, and edema
   b. anxiety, chest pain, and diaphoresis
   c. dyspnea, fatigue, and fluid retention

2. Which of the following best describes what happens to the body in heart failure?
   a. The heart can’t supply sufficient quantities of blood and oxygen to meet the metabolic needs of the body’s tissues.
   b. The lungs can’t sufficiently oxygenate the blood, resulting in a decreased workload to the heart.
   c. The heart muscle relaxes, resulting in an enlarged heart and improved pumping ability.

3. Which of the following is true about remodeling?
   a. It happens quickly.
   b. It results in dilation of the atria only.
   c. It begins with some type of injury to the myocardium.

4. Hyperthyroidism and anemia can cause
   a. low-output heart failure
   b. high-output heart failure
   c. biventricular failure

5. Normal ejection fraction is
   a. 40% to 55%.
   b. 60% to 80%.
   c. 85% to 95%.

6. Long-standing systemic hypertension can lead to
   a. left ventricular diastolic dysfunction.
   b. right ventricular systolic dysfunction.
   c. idiopathic dilated cardiomyopathy.

7. A patient experiences fatigue, palpitations, and dyspnea with normal activity. Which NYHA functional class would be assigned?
   a. Class II
   b. Class III
   c. Class IV

8. Raising the heart rate causes the cardiac output to
   a. also increase.
   b. decrease.
   c. stay the same.

9. Which of the following is a compensatory mechanism to maximize the functioning of the heart?
   a. reduction of the stroke volume by decreasing the amount of water in the bloodstream
   b. stimulation of the parasympathetic nervous system
   c. activation of the renin-angiotensin-aldosterone system

10. A patient with only jugular venous distention and pе-

11. Which of the following is the most commonly used and most useful diagnostic test for heart failure?
   a. 12-lead electrocardiogram
   b. cardiac catheterization with ventriculogram
   c. transthoracic echocardiogram

12. The amount of fluid filling the ventricles at the end of diastole is called
   a. preload.
   b. afterload.
   c. stroke volume.

13. The first-line treatment for heart failure is to reduce
   a. preload.
   b. afterload.
   c. contractility.

14. Which of the following drugs is usually the initial treatment of heart failure?
   a. digoxin (Lanoxin)
   b. furosemide (Lasix)
   c. metoprolol (Lopressor)

15. Obtaining a baseline weight for a patient with heart failure is most important for
   a. assessing intake and output.
   b. indicating when the patient needs to have afterload increased.
   c. evaluating the effectiveness of therapies.

16. Administration of nesiritide results in
   a. fluid retention.
   b. an increase in preload.
   c. a decrease in preload.

17. Advise your patient to notify her primary care provider or cardiologist if she
   a. awakens at night to urinate.
   b. loses 5 pounds in a week.
   c. gains 2 pounds overnight.

Turn to page 52 for the CE Enrollment Form.