Lesson 295: PreAnesthetic Assessment of the Patient With Systolic or Diastolic Heart Failure

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Read this article, reflect on the information presented, then go online and complete the lesson post-test and course evaluation before the termination date below. (CME credit is not valid past this date.) You must achieve a score of 80% or better to earn CME credit.

TIME TO COMPLETE ACTIVITY: 2 hours
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Needs statement

Diagnosing congestive heart failure (CHF) may be difficult in the elderly or obese patient. Complications during anesthesia can lead to increased morbidity and mortality. Recognizing this common condition has been identified by committee as required knowledge for anesthesiologists.

Learning Objectives

At the end of this activity, the participant should be able to:

1. List the diagnostic criteria for CHF with preserved ejection fraction (EF).
2. Differentiate the diagnostic criteria between CHF with, and without, preserved EF.
3. Define the American College of Cardiology/American Heart Association and New York Heart Association classifications for CHF.
4. Identify differences in ventricular structural abnormalities in CHF with and without preserved EF.
5. Quote the prevalence and demographics of CHF.
6. Outline the pathophysiology leading to CHF.
7. Discuss the treatment options for patients with CHF.
8. Evaluate the patient with acute decompensated heart failure.
9. Explain the anesthetic implications for CHF.
10. Establish hemodynamic goals for patients with CHF.
Case History

A 13-year-old girl was brought to the emergency department by her parents after developing progressive swelling of the face, lips, and tongue. Her parents explained that over the course of the afternoon, the child’s voice had become increasingly hoarse, and she began to complain of difficulty in swallowing. Earlier that day, she had been to the dentist for routine dental cleaning. She had been well at the time, and the visit itself was uneventful.

On physical examination, there was marked edema of the perioral region. Her tongue was severely enlarged such that she had difficulty keeping her mouth closed and controlling her saliva. She resisted lying down, preferring to sit in a forward-leaning position. No other constitutional symptoms, including rash or urticaria, were apparent. Vital signs included blood pressure, 100/60 mm Hg; heart rate, 88 beats per minute; oxygen saturation, 96%; and temperature, 97.2°F.

Heart failure (HF) is a complex syndrome that can be defined hemodynamically as an inability to provide adequate cardiac output to support the metabolic demands of the body, or to do so only at elevated filling pressures. Clinically, it is characterized by signs and symptoms of dyspnea on exertion, fatigue, orthopnea, edema, and rales. Congestive heart failure (CHF) typically has been subdivided into systolic heart failure (SHF) and diastolic heart failure (DHF). The ejection fraction (EF) is normal or near normal in almost 50% of patients diagnosed with HF.¹

Numerous underlying alterations in cardiovascular physiology lead to HF, including myocardial ischemia, congenital anomalies, valvular disease, and pericardial disease; all result in a constellation of similar clinical signs and symptoms. HF continues to be a major health problem in the United States with nearly 5 million people affected.² As the management of HF improves, the number of individuals living with the disease increases. As a result, more patients admitted for surgery have HF.

Prevalence and Demographics

The prevalence of HF has been increasing both within the United States and worldwide. In the United States, about 500,000 new cases are diagnosed annually. Approximately 1 in every 5 people aged 40 in the United States will develop HF in his or her lifetime, with the prevalence increasing with increasing age. Approximately 6% to 10% of people older than 65 years have HF.
The prevalence of DHF is estimated to be 15%, 33%, and 50% at ages less than 50 years, 50 to 70 years, and more than 70 years, respectively. Patients with DHF are more likely to be older, women, and hypertensive, and less likely to have had previous myocardial infarction or coronary artery disease (CAD) than patients with SHF.

**Clinical Manifestations**

The symptoms are similar between SHF and DHF. Typically, patients have poor exercise tolerance, dyspnea on exertion, fatigue, orthopnea, paroxysmal nocturnal dyspnea, or chest pain. Accompanying signs indicating fluid overload include peripheral edema, an S3 or S4 heart sound on auscultation, pulmonary crackles, and jugular venous distention. Some patients predominantly experience dyspnea and intolerance to activity, whereas others may have signs of fluid overload exclusively, without dyspnea.

Patients with DHF typically have exercise intolerance secondary to an elevation in left atrial and pulmonary venous pressures that occur with worsening left ventricular (LV) relaxation. Under normal circumstances, exercise results in an increased heart rate that reduces diastolic filling time and may result in pulmonary edema and worsened cardiac output in patients with impaired ventricular relaxation.

**Classifications of Heart Failure**

The American College of Cardiology/American Heart Association (ACC/AHA) guidelines for the assessment of chronic HF classifies patients based on 4 stages of symptoms (Table 1). The New York Heart Association (NYHA) categorizes HF based on levels of physical exertion required to illicit symptoms (Table 2). In addition to various methods for classifying chronic HF, it has been divided classically into 2 distinct phenotypes: HF with reduced ejection fraction (HFrEF) or SHF and HF with preserved ejection fraction (HFpEF) or DHF (Table 3).
HFrEF or SHF is a pathologic state in which abnormal cardiac function results in an inability of the heart to pump adequate blood at a rate required to meet the metabolic demands of the body. The principal derangement in SHF is impaired contractile function leading to a decreased EF.

HFrEF or DHF is a pathologic state in which impaired relaxation or resistance to LV filling results in congestion, or in filling of the ventricular chamber only at elevated pressures to maintain sufficient stroke volume. DHF affects approximately 50% of patients with chronic HF.

**Table 2. New York Heart Association Classification System**

<table>
<thead>
<tr>
<th>Stage</th>
<th>Description</th>
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<tbody>
<tr>
<td>I</td>
<td>Asymptomatic with normal physical activity</td>
</tr>
<tr>
<td>II</td>
<td>Symptomatic with moderate exertion</td>
</tr>
<tr>
<td>III</td>
<td>Symptomatic with minimal exertion</td>
</tr>
<tr>
<td>IV</td>
<td>Symptomatic at rest</td>
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**Table 3. Comparison of Systolic and Diastolic Heart Failure**

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<thead>
<tr>
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<th>SHF</th>
<th>DHF</th>
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<tr>
<td>Demographics</td>
<td>Younger, male, CAD/history of MI</td>
<td>Older, female, hypertensive</td>
</tr>
<tr>
<td>Pathophysiology</td>
<td>Impaired contractile function, decreased EF</td>
<td>Impaired myocardial relaxation, normal EF</td>
</tr>
<tr>
<td>Chamber/cellular remodeling</td>
<td>Ventricular chamber dilation, elongated/narrow myocytes, reduced myofibrillar density, degradation of fibrillar collagen</td>
<td>Normal or decreased ventricular chamber size, increased ventricular wall thickness, increased ratio of mass-to-chamber volume, increased myocyte diameter, increased collagen and collagen crosslinks</td>
</tr>
<tr>
<td>Treatment</td>
<td>β-blockers, ACEIs/ARBs, diuretics, CRT</td>
<td>β-blockers, ACEIs/ARBs, aldosterone antagonists (lack of significant evidence for survival benefit), dihydropyridine CCB Note: sensitive to preload reduction; caution with nitrates, diuretics, ACEIs</td>
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ACEIs: angiotensin-converting enzyme inhibitors; ARBs: angiotensin receptor blockers; CAD: coronary artery disease; CCB: calcium channel blocker; CRT: cardiac resynchronization therapy; DHF: diastolic heart failure; EF: ejection fraction; MI: myocardial infarction; SHF: systolic heart failure

Whether SHF and DHF are 2 distinct forms of HF, or instead are extremes in a spectrum of overlapping phenotypes remains controversial. Those who support the spectrum hypothesis claim that HF is a complex multifactorial disease, and similar to other complex diseases, it has a broad linear distribution with varying phenotypes at opposite ends of a bell-shaped spectrum. The distribution of EFs in HF falls along a bell-shaped curve, and under this hypothesis, DHF and SHF fall at opposite ends of the spectrum. It is for this reason, proponents argue, that the 2 forms of HF have different characteristics and clinical responses to therapy. They note that systolic dysfunction is not unique to HFrEF, as aspects of systolic dysfunction can be found in HFrEF.
Proponents of the theory that SHF and DHF are 2 separate phenotypes within a spectrum of HF argue that the distribution of EFs seen in HF actually is bimodal, not unimodal. They also point out that the ventricular and cellular remodeling patterns seen in SHF and DHF are distinct from one another, that SHF and DHF tend to develop in different patient populations, and that the progression and pathogenesis of the disease are distinct.²

Pathophysiology

The syndrome of HF can progress from a number of chronic cardiovascular conditions, including hypertension, ischemic myocardial disease, myocarditis, valvular disease, disorders of the pericardium, and other cardiomyopathies ultimately leading to the expression of similar signs and symptoms in the spectrum of HF. The underlying pathogenesis of HF is widespread. Hypertension and advancing age are the greatest risk factors for the development of DHF. By contrast, CAD causes about two-thirds of SHF cases; other nonischemic causes include myocarditis, thyroid disease, alcohol use, and other behaviors inflicted on the myocardium.²,⁴

Adequate peripheral circulation in HF is maintained by compensatory mechanisms that include hypertrophy, stimulation of the renin–angiotensin–aldosterone system, and retention of salt and water. Myocardial remodeling and conversion from compensated hypertrophy to HF involve complex molecular and cellular changes, including myocyte growth and death, as well as changes in the extracellular matrix. These alterations result in changes to myocardial structure and function that impair systolic function, diastolic function, or both. Stimuli for these changes include mechanical strain on myocytes, neurohormones (eg, norepinephrine and angiotensin), inflammatory cytokines, and other growth factors.⁹

Increased LV mass is seen in most forms of HF; however, the patterns of ventricular remodeling differ between SHF and DHF. The ventricular chamber often is dilated in HFrEF, but less so in HFrEF.² In HFpEF, the ventricular cavity size often remains the same or decreases, and end-diastolic and end-systolic volumes remain normal or decrease. In HFpEF, there is an increase in wall thickness, mass, and the ratio of mass-to-chamber volume.⁵

In HFrEF, the cardiomyocyte is narrow and elongated with reduced myofibrillar density. By contrast, in HFpEF the myocyte diameter and resting tension are both increased. There also are differences in the balance between matrix metalloproteinases (enzymes that play a role in cell proliferation and apoptosis) and their inhibitors in HFrEF and HFpEF.² In animal models of HFrEF, fibrillar collagen is degraded. This differs from the pressure-overloaded hypertrophy seen in HFpEF, a result of increased collagen and collagen crosslinks.⁵

The resulting hemodynamic profile may be similar between SHF and DHF. In SHF, a reduced EF leads to decreased cardiac output, increased LV end-diastolic volumes, and a passive increase in left atrial and pulmonary venous pressures. In DHF, there also is an increase in left atrial and pulmonary venous pressures resulting from the increase in LV diastolic pressure. Thus, both forms may lead to pulmonary congestion, and chronically can lead to increased pulmonary vascular resistance.⁵

Normal diastolic function involves a process of active myocardial relaxation, which occurs early in diastole. It results in a rapid decline in pressure causing a suction effect that helps LV filling, as well as passive distention of the LV that occurs in late diastole. During this later phase of diastole, atrial
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contraction contributes approximately 20% to 30% of the total LV filling volume at low pressures. In diastolic dysfunction, loss of normal LV relaxation and distensibility from either structural or functional abnormalities results in impaired ventricular filling and elevated filling pressures. LV filling shifts from predominantly occurring in early diastole to more later-phase diastole due to an inability of the ventricle to actively relax. Atrial contraction in turn contributes more to diastolic filling under these conditions.10

Diagnosis

The signs and symptoms of HF often are nonspecific and include dyspnea, exercise intolerance, weakness, and fatigue; these also can be present in other disease states—including non-cardiac-mediated diseases such as thyroid abnormalities, pulmonary disease, obesity, and anemia. There is no officially established set of diagnostic criteria for HF; however, there are a number of commonly used systems including the Framingham criteria, Boston criteria, and Duke criteria.

In the commonly used Framingham criteria, the diagnosis of HF requires the simultaneous presence of 2 major criteria, or 1 major and 2 minor criteria (Figure 1).11

It is difficult to distinguish between SHF and DHF based on clinical signs and symptoms. An elevated level of brain natriuretic peptide is a reliable means of diagnosing HF, but does not help distinguish between SHF and DHF. A brain natriuretic peptide level greater than 100 pg/mL has been shown to be 95% sensitive for the diagnosis of HF, but only 14% specific for detecting systolic failure.12 Nonspecific tests—such as chest x-ray that reveals cardiomegaly and electrocardiography (ECG) that reveals signs of LV hypertrophy or ischemia—can be used as diagnostic aids. Overall, the diagnosis of SHF involves assessing various clinical signs and symptoms from the patient’s medical history, in addition to echocardiographic evidence of decreased EF.

The diagnosis of DHF is more controversial and can include a number of diagnostic methods. In the European Society of Cardiology guidelines, 3 conditions must be met concurrently for a diagnosis of DHF (Figure 2).13 There are a number of methods for evaluating impaired relaxation, the third parameter. Cardiac catheterization is the gold standard because it directly measures ventricular diastolic pressure, particularly the rate of decline in LV

<table>
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<tr>
<th>Major Criteria</th>
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<tr>
<td>Paroxysmal nocturnal dyspnea</td>
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<tr>
<td>Neck vein distension</td>
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<tr>
<td>Rales</td>
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<tr>
<td>Radiographic cardiomegaly</td>
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<tr>
<td>Acute pulmonary edema</td>
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<tr>
<td>S3 gallop</td>
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<tr>
<td>Increased central venous pressure (&gt;16 cm H2O at right atrium)</td>
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<tr>
<td>Hepatojugular reflux</td>
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<tr>
<td>Weight loss &gt;4.5 kg in 5 days in response to treatment</td>
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<table>
<thead>
<tr>
<th>Minor Criteria</th>
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<tbody>
<tr>
<td>Ankle edema, bilaterally</td>
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<tr>
<td>Nocturnal cough</td>
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<tr>
<td>Dyspnea on ordinary exertion</td>
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<tr>
<td>Hepatomegaly</td>
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<tr>
<td>Pleural effusion</td>
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<tr>
<td>Decrease in vital capacity by one-third from maximum recorded</td>
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<tr>
<td>Tachycardia (heart rate &gt;120 bpm)</td>
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**Figure 1. In the commonly used Framingham criteria, the diagnosis of heart failure requires the simultaneous presence of 2 major criteria, or 1 major and 2 minor criteria.**
Blood inflow velocity in the transmitral valve is evaluated by Doppler ECG. Peak velocities of blood flow during early diastolic filling (E-wave) and atrial contraction (A-wave) are measured and the ratio calculated. Under normal conditions, E-wave velocity is greater than A-wave velocity because atrial contraction contributes little to diastolic filling. The E-to-A ratio is approximately 1.5.

In early diastolic dysfunction due to a slower relaxation time secondary to ventricular stiffness, atrial contraction contributes more to filling; the relationship reverses and the E-to-A wave ratio is less than 1—which is typical of grade I diastolic dysfunction. In grade II diastolic dysfunction due to increasing left atrial pressures, early diastolic velocities are elevated. This causes an increase in the E-wave, and the E-to-A ratio increases again to “normal” or pseudonormal levels.

With further worsening of diastolic dysfunction, LV diastolic pressure rises considerably. Atrial contraction contributes less to ventricular filling because of high end-diastolic pressures; thus, the E-to-A wave ratio rises significantly due to the drop in A-wave pressure. The ratio often goes above 2, which is seen in grades III and IV diastolic dysfunction.

E-to-A wave ratios are affected by mitral valve anatomy, blood volumes, and abnormal cardiac rhythms including atrial fibrillation. Therefore, in these settings the ratios are of limited use for diagnosing diastolic abnormalities. In clinical practice, a diagnosis of DHF is usually one of exclusion in symptomatic patients who have documented preserved EF.

### Evaluating Acute Decompensated Heart Failure

In patients with a history of HF, acute decompensation can commonly present as respiratory failure. It is important for the anesthesiologist to recognize the signs and symptoms of this hemodynamic state because it presents a significant risk to the patient for complications. Elective surgery should be avoided until the condition is stabilized.

Acute decompensated HF typically results from SHF or DHF with a number of different stimulating factors. The clinical presentation commonly includes significant dyspnea because of a rapid accumulation of fluid in interstitial lung spaces that typically results from acutely elevated cardiac filling pressures. Patients characteristically present with cough, dyspnea, fatigue, and occasionally chest pain. Tachypnea, tachycardia, and either hypertension or hypotension may develop. Classical physical findings include crackles or wheezing on auscultation (indicating significant pulmonary edema), S3 or S4 heart sound on cardiac examination, and elevated jugular venous pressures (indicating elevated pressure in early diastole, and end-diastolic pressure. The procedure involves the use of fluid-filled catheters or micromanometer catheters. Doppler ECG is less invasive, however, and is now the primary way to noninvasively assess diastolic function.\(^{12}\)

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right-sided filling pressures).13

The patient should be evaluated for precipitating factors. Common causes include myocardial infarction or ischemia, severe hypertension, atrial fibrillation or other arrhythmias, and acute valvular abnormalities such as mitral or aortic regurgitation. The diagnosis of acute decompensated HF is based on a typical clinical presentation. Supporting tests to confirm the diagnosis include ECG for signs of ischemia, chest radiography for signs of pulmonary edema, brain natriuretic peptide levels that can be significantly elevated, and echocardiography to assess valvular abnormalities and overall cardiac function.

Treatment

Based on a number of randomized controlled trials, drug therapy has been the primary treatment for SHF. Surgical therapy makes use of several devices as well as transplantation. Diuretics are a key component of drug therapy for symptomatic relief of water retention in patients with SHF. The dose is titrated by monitoring weight and electrolyte status with the goal of maintaining euvolemia. Diuretics, however, have not been shown to prolong survival.

By inhibiting the renin–angiotensin–aldosterone system (which often is abnormally activated in the progression of HF), angiotensin-converting enzyme inhibitors (ACEIs) have significantly benefited patients, including fewer symptoms of HF and hospital admissions, and prolonged survival.

Angiotensin receptor blockers (ARBs) are an alternative therapy in patients with ACEI intolerance. Multiple randomized trials of various ARBs found beneficial effects, including decreased hospitalizations. Spironolactone, an aldosterone receptor antagonist, has been found to increase survival in patients with HF, in addition to ACEIs. A key component of drug therapy for HF is the use of β-blockers to decrease chronic sympathetic activation.3

Cardiac transplantation remains the definitive treatment for HF, although there is an overall lack of available transplant organs in the United States. A number of alternative surgical treatment options exist, however, including coronary revascularization for ischemic causes of HF, valve replacements, and LV reconstruction for pathologic remodeling.

Cardiac resynchronization therapy, which involves the use of biventricular pacing in a synchronized mode, has been used in patients in whom EF is less than 35% and QRS duration is greater than 0.12 seconds (ie, patients with abnormal conduction and possible ventricular contraction dyssynchrony). Cardiac resynchronization therapy in combination with optimal medical management has been shown to improve functional class, exercise capacity, and EF, and reduce mortality in patients with SHF.

Increasingly, LV-assist devices (LVADs) are used because many patients develop refractory HF despite medical management. The REMATCH (Randomized Evaluation of Mechanical Assistance in the Treatment of Congestive Heart Failure) trial, in which patients were randomized to receive optimal medical management, or LVAD plus optimal medical management, found that the LVAD group had better 1- and 2-year survival rates than patients in the medical management alone group.3

The goals in treating patients with DHF include relief of symptoms, elimination of exacerbations, and reduced mortality; however, the mainstay drugs used in treating SHF—including ACEIs, ARBs, β-blockers, and aldosterone antagonists—have not been shown to be as effective against DHF in multiple
studies. According to the ACC/AHA guidelines, the management of DHF should include treatment of hypertension, maintenance of sinus rhythm, prevention of tachycardia and ischemia, and reduction of venous pressure.6

Blood pressure control reduces LV end-diastolic pressure, improves relaxation of the ventricle leading to improved early filling, decreases ischemia, and reduces LV hypertrophy, thus inhibiting further diastolic dysfunction. ACEIs have been shown to increase exercise capacity and possibly reduce the risk for hospitalization of patients with DHF; however, ACEIs have not been shown to improve overall prognosis in a number of studies evaluating these drugs for treatment of hypertension in HfPfEF. Despite a lack of data on overall decrease in mortality, ACEIs, ARBs, and aldosterone antagonists are the recommended drugs for treating hypertension in diastolic failure.

Maintenance of sinus rhythm or avoidance of tachycardia improves relaxation and increases diastolic filling time. If conversion to sinus rhythm is not possible, rate control is important. Calcium channel blockers or β-blockers are recommended. Specifically, dihydropyridine calcium channel blockers have been shown to be beneficial in the treatment of diastolic dysfunction, in contrast to SHF.6 Patients with DHF who have a small, stiff left ventricle are particularly sensitive to decreases in preload; this can result in a significant fall in cardiac output. Therefore, these patients may not tolerate diuretics, venodilators such as nitrates, ACEIs, or calcium channel blockers.

Implications and Goals for Anesthesia

CHF has important implications for surgery and anesthesia. A number of studies have demonstrated significant risks for morbidity and mortality after noncardiac surgery in patients with HF. A study by Hammill et al14 looked at 159,327 patients with HF undergoing noncardiac surgery and found an 8.0% incidence of operative mortality—more than double the incidence among patients with CAD and more than triple the incidence in the control group. After controlling for comorbidities, there was a 63% higher incidence of operative mortality among patients with HF compared with controls; the rate for all causes of 30-day readmission also was significantly higher.

Perioperatively, a number of hemodynamic changes can occur that affect both systolic and diastolic functions. A thorough preoperative evaluation of the patient’s HF symptoms and exercise tolerance should be obtained. Patients with HF are particularly sensitive to changes in volume status, as well as increases in afterload. Given that there are many different underlying causes of HF, anesthetic and hemodynamic management of these patients may vary depending on the primary pathology. Valvular pathology leading to HF should be treated based on the specific valve affected and the appropriate hemodynamic goals associated with those lesions. In patients with ischemic cardiomyopathies, anesthetic management to optimize myocardial oxygen supply and demand is essential.

For the surgical patient with DHF, several hemodynamic changes during the perioperative period exert adverse effects on diastolic filling, including tachycardia, abnormal cardiac rhythm, or myocardial ischemia. Tachycardia decreases the late phase of diastole and reduces cardiac filling, LV end-diastolic volumes, and cardiac output. Atrial fibrillation is not tolerated because optimal cardiac function relies on atrial contraction for LV filling and stroke volume. Acute, severe elevations in systemic blood pressure worsen LV wall stress and can impair myocardial relaxation. Additionally, patients with DHF can be extremely sensitive to decreases in preload, and venodilators should be used with caution. Volume status should be closely monitored, and
hypovolemia or volume overload should be avoided. In addition to the negative effects of ischemia on the myocardium, ischemia itself can cause reversible impairment in myocyte relaxation, which further worsens diastolic function.

Myocardial ischemia is one of the main mechanisms of LV diastolic dysfunction postoperatively. Factors including pain, shivering, tachycardia, hypertension, and hypoxia increase myocardial oxygen demand and cause ischemia. Volatile agents as well as opioids and muscle relaxants do not appear to worsen diastolic dysfunction alone.

No studies have demonstrated improved outcomes with certain anesthetic techniques over others, including regional or general anesthesia; however, the anesthesiologist must keep in mind that hemodynamic effects can vary depending on the anesthetic method.

**Management of the Case Presented**

After the patient completed a physical examination and provided her medical history, it was determined that her episodes of HF had been medically optimized. The echocardiogram indicated preserved EF and abnormal relaxation of the left ventricle. Thus, the patient met criteria for diastolic heart dysfunction with episodes of DHF.

Because this was a laparoscopic repair, general endotracheal anesthesia was chosen. Lidocaine, fentanyl, midazolam, propofol, and vecuronium were administered for induction. Large fluctuations in blood pressure and heart rate were avoided on direct laryngoscopy by administration of 75 mcg of fentanyl before intubation. Routine monitors were used and fluid management was directed at maintaining euvolemia. Small doses of labetalol were administered during emergence and extubation to control heart rate and blood pressure. IV morphine in 5-mg doses was given postoperatively for pain management.

Dr. Elizabeth A.M. Frost, who is the editor of this continuing medical education series, is clinical professor of anesthesiology at The Mount Sinai School of Medicine in New York City. She is the author of Clinical Anesthesia in Neurosurgery (Butterworth-Heinemann, Boston) and numerous articles. Dr. Frost is past president of the Anesthesia History Association and former editor of the journal of the New York State Society of Anesthesiologists, Sphere. She is also editor of the book series based on this CME program, Preanesthetic Assessment, Volumes 1 through 3 (Birkhäuser, Boston) and 4 through 6 (McMahon Publishing, New York City).
REFERENCES


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Post-test

1. The American College of Cardiology/American Heart Association guidelines for assessing chronic heart failure:
   a. classify patients according to severity of symptoms
   b. classify patients based on the stage of symptoms
   c. include 5 levels of severity
   d. are the same as the New York Heart Association classification system

2. The principal derangement in diastolic heart failure (DHF) is:
   a. impaired contractility
   b. reduced ejection fraction
   c. impaired ventricular relaxation and compliance
   d. preserved left ventricular (LV) diastolic pressure

3. Which of the following is not a ventricular compensatory mechanism to offset stress on cardiac performance?
   a. Hypertrophy
   b. Salt and water retention
   c. Stimulation of the renin–angiotensin–aldosterone system
   d. Regional wall motion abnormalities

4. Risk factors for the development of DHF include:
   a. hypertension and advancing age
   b. high-fat diet
   c. coronary artery disease
   d. end-stage renal disease

5. Using the Framingham criteria, a diagnosis of heart failure requires:
   a. 4 major criteria
   b. 2 major, or 1 major and 2 minor criteria
   c. echocardiographic evaluation
   d. correlation with another system for an accurate diagnosis
6. The European Society of Cardiology recommends the use of all of the following criteria to diagnose DHF, except:
   a. presence of symptoms of congestive heart failure (CHF)
   b. normal LV systolic function
   c. hypertension
   d. documented impairment of LV compliance

7. The management of patients with DHF should include:
   a. treatment of hypertension
   b. cardiac resynchronization therapy
   c. aggressive diuresis
   d. inotropic agents

8. The incidence of operative mortality for patients with CHF undergoing noncardiac surgery has been reported to be:
   a. 1%
   b. 3%
   c. 8%
   d. 15%

9. Hemodynamic goals during the perioperative period include the following, except:
   a. maintaining normal sinus rhythm
   b. avoiding tachycardia
   c. avoiding hypertension
   d. avoiding volatile anesthetics because of possible worse outcome

10. In DHF, angiotensin-converting enzyme inhibitors have been shown to:
    a. improve exercise capacity
    b. increase the risk for hospitalization
    c. improve overall prognosis
    d. lower overall mortality