Lesson 279: PreAnesthetic Assessment of the Patient With Cardiomyopathy

Authored by: Prashan H. Thiagarajah, MD
Research associate, Department of Cardiology, Beth Israel Medical Center, New York, New York

Reviewed by: Somasundaram Thiagarajah, MD, FRCA
Clinical professor of anesthesiology, Albert Einstein College of Medicine, Bronx, New York; anesthesiologist, Beth Israel Medical Center, New York, New York

DATE REVIEWED: DECEMBER 2008

A COURSE OF STUDY FOR AMA/PRA CATEGORY 1 CREDIT
Read this article, reflect on the information presented, then go online and complete the lesson post-test and course evaluation before April 30th, 2010. (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
ACCREDITATION STATEMENT
The Mount Sinai School of Medicine is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to provide continuing medical education for physicians.

RELEASE DATE: April, 2009
TERMINATION DATE: April 30, 2010
TARGET AUDIENCE: Anesthesiologists

CREDIT DESIGNATION STATEMENT
The Mount Sinai School of Medicine designates this educational activity for a maximum of 2 AMA PRA Category 1 Credits.™ Physicians should only claim credit commensurate with the extent of their participation in the activity.

It is the policy of Mount Sinai School of Medicine to ensure objectivity, balance, independence, and scientific rigor in all CME-sponsored educational activities. All faculty participating in the planning or implementation of a sponsored activity are expected to disclose to the audience any relevant financial relationships and to assist in resolving any conflict of interest that may arise from the relationship. Presenters must also make a meaningful disclosure to the audience of their discussions of unlabeled or unapproved drugs or devices.

The author, reviewer, and editor have no relationships with pharmaceutical companies or manufacturers of products to disclose. This educational activity may contain discussion of published and/or investigational uses of agents for the treatment of disease. Some uses of these agents have not been approved by the FDA. Please refer to the official prescribing information for each product for approved indications, contraindications, and warnings.

Needs statement
Cardiomyopathic disorders are being diagnosed increasingly as the result of both improvements in the means of detection and aging of the population. Consequently, more patients with cardiomyopathy as an underlying condition undergo anesthesia. It is essential that anesthesiologists understand the fundamental pathologies of these cases to improve their management.

Learning Objectives

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

1. Identify the types of cardiomyopathy.
2. List the symptoms of cardiomyopathy.
3. Differentiate between ischemic and nonischemic cardiomyopathy, by cause.
4. Discuss the basic medical and surgical management of the patient with cardiomyopathy.
5. Outline the application of an automatic implantable cardioverter defibrillator (ICD).
6. Describe biventricular pacing.
7. Outline the appropriate preparation for surgery of patients with cardiomyopathy.
8. Manage patients requiring anesthesia who have an automatic ICD.
9. Manage patients with a low ejection fraction due to cardiomyopathy.

Case History

To improve the cardiac function of a 78-year-old man with an ejection fraction (EF) of 20%, an operation was scheduled in which his single-chamber, ventricular-inhibited pacemaker would be upgraded to a biventricular pacemaker. The patient’s current medications included furosemide, warfarin (which was discontinued a week before surgery), and losartan (an angiotensin II–receptor antagonist). Electrocardiography (ECG) revealed left bundle branch block. Chest x-ray revealed cardiomegaly and lung congestion. The patient’s vital signs were as follows: blood pressure (BP), 90/65 mm Hg; heart rate, 92 beats/min; respiratory rate, 22 breaths/min; hemoglobin, 14 g/100 mL. His potassium, magnesium, and creatinine levels were all within normal range.

The anesthetic management of patients with cardiomyopathy and reduced systolic function is challenging and may be associated with high mortality rates. Tabib and colleagues retrospectively analyzed autopsies conducted in 1,500 cases of unexpected death and identified 43 that were possibly related to anesthesia and surgery. Pathologic examination revealed cardiac lesions in 40 cases, 20% of which were due to cardiomyopathy (Table 1). Of note, arrhythmogenic right ventricular cardiomyopathy (an inherited disease in which infiltration of the right ventricle with fatty fibrotic tissue causes ventricular arrhythmias and sudden death) was identified in 35% of cases in the subgroup series. T-wave inversion in the anterior precordial leads was observed with ECG.

### Table 1. Cardiac Causes of Death Reported by Tabib and Colleagues

<table>
<thead>
<tr>
<th>Cause</th>
<th>Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arrhythmogenic right ventricular cardiomyopathy</td>
<td>14</td>
</tr>
<tr>
<td>Coronary artery disease</td>
<td>9</td>
</tr>
<tr>
<td>Cardiomyopathy</td>
<td>8</td>
</tr>
<tr>
<td>Structural abnormalities of the bundle of His</td>
<td>7</td>
</tr>
<tr>
<td>Mitral valve prolapse</td>
<td>1</td>
</tr>
<tr>
<td>Acute myocarditis</td>
<td>1</td>
</tr>
</tbody>
</table>

Classification of Cardiomyopathy

Cases of cardiomyopathy, broadly defined as heart muscle disease that decreases cardiac function, can be classified into 4 types: dilated, hypertrophic, restrictive, and Takotsubo (Table 2).

Dilated cardiomyopathy is characterized by a large heart cavity with impaired systolic function in one or both ventricles. It occurs more frequently in males (3:1) and African Americans (2.5:1) than in Caucasians; the overall prevalence is 920 per 100,000. Dilated cardiomyopathy can be ischemic or nonischemic. The ischemic type is related to atherosclerosis and ischemic heart disease. The nonischemic type can be secondary to viral infection (eg, HIV, coxsackievirus, cytomegalovirus), toxoplasmosis, Chagas’ disease, trichinosis, leptospirosis, Lyme disease, chemotherapy (eg, doxorubicin), or drug abuse (eg, alcohol, cocaine, methamphetamine, heroin), or it may develop during the peripartum period.
The clinical presentation of dilated cardiomyopathy includes symptoms such as dyspnea, orthopnea, weakness, fatigue, and leg edema. Physical findings are similar to those seen in congestive heart failure. Patients may have jugular venous distention, rales and pulmonary edema, resting tachycardia, S3 and S4 heart sounds, and cardiomegaly.

Hypertrophic cardiomyopathy may develop in conjunction with an increased hemodynamic workload (called hypertensive hypertrophic cardiomyopathy) or without provocation (called hypertrophic obstructive cardiomyopathy or idiopathic hypertrophic subaortic stenosis). The idiopathic type is inherited in an autosomal dominant pattern with variable penetrance. Echocardiography reveals disease in about a quarter of first-degree relatives.

Restrictive cardiomyopathy, the least common type in Western countries, is most frequently caused by sarcoid disease.

The fourth type, Takotsubo cardiomyopathy, has been described as transient ventricular dysfunction causing severe hypotension. It is rare, usually occurring in postmenopausal women, and is associated with stress and chest pain. ECG may show ST-segment elevation; echocardiography and ventriculography show apical and mid left ventricular ballooning with hypokinesia. The basal segment of the left ventricle may be hyperkinetic.

**Patient Management**

Two goals are key in the management of patients with cardiomyopathy; one is to improve systolic function, and the other is to prevent sudden death due to ventricular arrhythmias.

Several types of treatment for dilated cardiomyopathy aim at improving systolic function. Patients initially should be managed medically. Biventricular pacing, cardioplasty, or cardiac transplantation also may be necessary to improve cardiac function. Arrhythmias are treated with the administration of amiodarone and/or placement of an automatic implantable cardioverter defibrillator (ICD). Amiodarone prevents life-threatening arrhythmia, and an ICD promptly treats an episode of arrhythmia with an electrical shock impulse.

Medical management to improve systolic function includes the administration of diuretics, β-blockers, angiotensin-converting enzyme (ACE) inhibitors, or angiotensin-receptor blockers.

In the renin–angiotensin system, angiotensin II causes vasoconstriction and the release of aldosterone, vasopressin, and antidiuretic hormone. The effect of these hormones is to increase the blood volume and thus BP. ACE inhibitors prevent the conversion of angiotensin I to angiotensin II and the subsequent hypertensive effects. The net effect is a reduced afterload. In patients with cardiac disease, ACE inhibitors reduce the risk for death by slowing cardiac remodeling, preventing the heart from becoming less efficient over time, and improving ventricular function.
Biventricular pacing devices (also known as cardiac resynchronization therapy defibrillators) often are used to improve systolic function in patients with cardiomyopathy (Figure 1). Biventricular pacing is beneficial for patients who have moderate to severe congestive heart failure with an EF below 30%, and with ventricular asynchrony. Interventricular conduction defect manifested by wide Q wave–R wave–S wave (QRS) complex is indicative of asynchrony of the 2 ventricles. Biventricular leads are programmed to synchronize the contraction of the right and left ventricles, thereby improving the EF. Biventricular pacing improves left ventricular systolic function, decreases left ventricular size, decreases mitral regurgitation, and shortens the prolonged QRS interval. The patient’s quality of life is markedly improved.5

Right ventricular pacing is achieved by placing a lead in the apex of the right ventricle. This lead, in addition to its function as a pacer, can detect arrhythmia and function as a defibrillator. Two shock coils are incorporated in the right ventricular lead for defibrillation. Left ventricular pacing is obtained by placing a lead in the obtuse marginal branch of the coronary sinus (Figure 1). The pacing of the 2 ventricles can then be synchronized.

Cardioplasty to improve systolic function has evolved in several aspects. In the most common type, the latissimus dorsi muscle is placed around the heart as a free flap, and its contractions are synchronized to augment ventricular systolic function. Mitral valve repair also improves cardiac function.

Cardiac transplantation may be recommended for patients with end-stage dilated cardiomyopathy in whom other therapies have not caused significant improvement. Left ventricular assist devices may be used as a “bridge” while the patient awaits a donor heart for transplantation.

Antiarrhythmic Drugs and Implantable Cardioverter Defibrillators

In addition to a low EF, ventricular arrhythmias tend to develop in patients who have cardiomyopathy, with death occurring suddenly. Therefore, oral administration of the antiarrhythmic medication amiodarone is prescribed, or a cardioverter defibrillator is implanted to treat ventricular tachycardia (VT).6

An ICD, which is more effective than amiodarone in reducing mortality in high-risk patients with previous myocardial infarction, is usually the primary treatment. Amiodarone may be used as an adjunct to reduce the frequency of ICD shocks.7

A single-chamber ICD consists of a generator that contains a battery and a small computer. A ventricular lead with 2 shock coils is attached to the generator (Figure 2). The battery life usually
ranges from 4 to 6 years. At the tip of the lead are a sensor and a pacer. These devices are able to distinguish between ventricular fibrillation and VT. In case of ventricular fibrillation, a 25-J shock is delivered from the right ventricular lead shock coil of the ICD. In case of VT, the device may pace the heart faster than the rate of VT, to override and break the VT. This is referred to as antitachycardia pacing. In case of bradycardia, the pacing function is initiated.

ICD placement also is indicated in patients in whom ventricular arrhythmia is likely to develop, such as those with idiopathic hypertrophic subaortic stenosis, prolonged QT syndrome, or Brugada syndrome. The latter is an inherited disease in which the risk for sudden cardiac death is increased because of ventricular fibrillation. ECG findings may show a pattern of right bundle branch block.

Preoperative Preparation

The preoperative preparation of these patients must be meticulous because they have minimal or no cardiac reserve (Table 3). Any decrease in myocardial contractility or the heart rate or increase in vasodilation can cause profound hypotension. Preoperatively, patients tend to be dehydrated because most have undergone diuresis—a further cause of hypotension in patients receiving anesthesia. Dehydration is generally beneficial for these patients and improves their limited cardiac function; however, they can easily become hypotensive with anesthetics, which cause vasodilation.

Preoperatively, hydration may not be desirable because it can lead to congestive heart failure. Fluid management is critical. Erring on the side of hypovolemia is prudent. A rational approach is to administer a vasopressor to counteract the vasodilator effect of the anesthetic.

Because ventricular arrhythmia may develop in the perioperative period, antiarrhythmic medications should be continued. Some patients may have already received an ICD. During anesthesia, a drug interaction resulting in hypotension requiring vasopressor therapy has been reported in patients taking ACE inhibitors combined with diuretics.

Arrhythmias develop when potassium or magnesium blood levels decrease. These electrolytes should be measured preoperatively and levels corrected as necessary.

![Image](image_url)

**Figure 2.** Chest x-ray showing ICD leads. *The right ventricular lead has 2 shock coils (translucent segments).*

**Table 3. Preoperative Assessment**

<table>
<thead>
<tr>
<th>Volume status</th>
</tr>
</thead>
<tbody>
<tr>
<td>Continuation of antiarrhythmic drugs</td>
</tr>
<tr>
<td>Drug interactions with ACE inhibitors</td>
</tr>
<tr>
<td>Electrolyte corrections: potassium, magnesium</td>
</tr>
<tr>
<td>Optimization of hemoglobin</td>
</tr>
<tr>
<td>Deactivation of ICD</td>
</tr>
<tr>
<td>Administration of inotropes, if necessary (resistance to usual dose may be observed)</td>
</tr>
<tr>
<td>Intra-aortic balloon pump, if necessary</td>
</tr>
</tbody>
</table>

ACE, angiotensin-converting enzyme; ICD, implantable cardioverter defibrillator
The patient’s oxygen-carrying capacity should be adequate; the main determinants are the cardiac output and the hemoglobin level. Therefore, hemoglobin should be maintained at a higher level; 13 to 14 g/100 mL has been recommended. To improve cardiac output, inotropes, biventricular synchronized pacing, or an intra-aortic balloon pump may be required.

**Management of the Patient With an ICD Before Surgery**

Many patients with cardiomyopathy may already have an implanted device for defibrillation but may not be aware of the type—whether it is a simple pacemaker or an ICD. The generators of ICDs are larger than those of pacemakers. The right ventricular lead of an ICD, unlike a pacemaker lead, has 2 sets of spiral segments, which can be identified by chest x-ray.

An ICD can cause difficulty during surgery when the ICD sensor mistakes the energy discharged during the use of electrocautery for a tachyarrhythmia. The antiarrhythmic function of the ICD is then activated, and the patient receives an inappropriate shock. This shock, if discharged during the vulnerable phase of the cardiac electrical cycle, may induce ventricular arrhythmia. Also, multiple shocks with repeated uses of the cautery can damage the heart, decrease cardiac function, damage the generator, or deplete the ICD battery. Therefore, all ICDs should be deactivated before surgery if electrocautery is to be used.

Placing a magnet on the generator of some ICDs may deactivate the shock therapy function, but not the pacing or sensing capability. With other ICDs, the placement of a magnet will not have any effect on function. Ideally, in the management of these patients, a cardiologist or the manufacturer’s representative should be consulted preoperatively.

After the ICD has been deactivated, defibrillator pads must be placed on the patient’s chest and connected to an external defibrillator as a standby in case ventricular arrhythmia develops.

If a critically ill patient with cardiomyopathy requires surgery that is complex and absolutely necessary, an intra-aortic balloon pump may be placed preoperatively.

**Anesthetic Management**

The anesthetic management of patients with severe cardiomyopathy is associated with a high rate of morbidity and mortality and therefore requires careful planning, preparation, and monitoring (Table 4). Many patients are undergoing surgery for placement of an ICD or biventricular pacemaker. However, they may require an emergency procedure or another type of surgery.

Preoperatively, the diagnosis of cardiomyopathy may be lacking or not easily forthcoming. A patient who has a history of coronary artery disease with a low EF is usually treated with furosemide and ACE inhibitors and receives an ICD. In the preoperative evaluation, 4 signs (quadratic sign), if present, should alert the anesthesiologist to a low EF: treatment with furosemide, treatment with ACE inhibitors, history of CHF/CAD, and cardiomegaly indicated on chest x-ray.

**Table 4. Clinical Presentation of Patients With Cardiomyopathy**

<table>
<thead>
<tr>
<th>History of CHF/CAD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Meds: furosemide, ACE inhibitors, digoxin, β-blocker</td>
</tr>
<tr>
<td>Cardiomegaly indicated on chest x-ray</td>
</tr>
<tr>
<td>ICD/biventricular pacemaker with ICD (EF &lt;30%)</td>
</tr>
<tr>
<td>ECG may show conduction defects</td>
</tr>
<tr>
<td>Blood pressure may be low, heart rate elevated</td>
</tr>
</tbody>
</table>

ACE, angiotensin-converting enzyme; CAD, coronary artery disease; CHF, congestive heart failure; ECG, electrocardiogram; EF, ejection fraction; ICD, implantable cardioverter-defibrillator
inhibitors, presence of an ICD, and cardiomegaly in the chest x-ray. If the preoperative diagnosis of cardiomyopathy is missed, routine anesthetic management carries considerable risk to the patient, who will likely become profoundly hypotensive.\textsuperscript{17,18}

Prompt recognition of hemodynamic instability and immediate intervention with appropriate vasoactive or inotropic medications is required to prevent deterioration of the patient’s condition. The anesthesiologist should administer phenylephrine in incremental doses of 100 mcg every 30 to 40 seconds; in addition, norepinephrine should be infused at a rate of 4 to 8 mcg per minute, or dopamine infused at 5 mcg/kg per minute. Doses should be adjusted to titrate the systolic BP to above 90 to 100 mm Hg.

Life-threatening ventricular arrhythmias can also develop in patients; anesthesiologists should be prepared to administer lidocaine or amiodarone, or to use defibrillation to correct the arrhythmia. In addition to basic monitoring (BP, pulse oximetry, ECG, end-tidal CO\textsubscript{2}), direct monitoring of the arterial BP is required to identify abrupt hemodynamic changes.\textsuperscript{18} Whenever the surgical procedure is complex or of long duration, monitoring with transesophageal echocardiography (TEE) is also appropriate.\textsuperscript{19} When a patient becomes hypotensive under anesthesia, TEE identifies the cause of hypotension, which can be due to global hypokinesia, regional ischemic ventricular dysfunction, or hypovolemia. These changes can be treated with inotropes, coronary vasodilators, or fluids, as indicated.

The cardinal feature of dilated cardiomyopathy is diminished systolic function or reduced left ventricular EF. Patients with a left ventricular EF above 45% usually do not require any change in anesthetic management.

Conventional anesthetics (eg, propofol, sodium thiopental, isoflurane) in the recommended doses depress not only the central nervous system but also the cardiac function. They tend to depress the myocardium, slow the heart rate, and dilate the blood vessels. Anesthetic management needs to be customized for patients with a left ventricular EF below 45%. The selection of anesthetics and doses that result in minimal vasodilation and myocardial depression is prudent. For example, ketamine, etomidate, and opioids minimally depress cardiac function and are used frequently.\textsuperscript{18-20}

The use of nerve blocks is a rational approach for appropriate surgical cases because they are associated with minimal hemodynamic abrasion.

Anesthetic management of the parturient with cardiomyopathy is challenging. The welfare of the baby must be considered when the critically ill mother is treated. Regional techniques are usually preferred for cesarean delivery in a normal parturient but may exaggerate the hypotension in a parturient with cardiomyopathy. If general anesthetics are indicated in an emergency for a decompensating mother, drugs that have a minimal effect on the baby need to be selected. The combination of etomidate with remifentanil has been successfully used without causing neonatal respiratory depression. Remifentanil, which crosses the placenta, is quickly metabolized by enzyme hydrolysis.\textsuperscript{21}

The administration of vasoactive or inotropic drugs may be required frequently to counteract the negative effects of anesthetics on cardiac function. Inotropic drugs (eg, dopamine, epinephrine, dobutamine, and milrinone) significantly increase the EF. In clinical reports, dopamine is frequently administered during the anesthetic care of these patients.\textsuperscript{19} Dopamine in the appropriate dose range
has positive inotropic, chronotropic, and vasoconstrictive effects, which make it an ideal agent to counteract the adverse cardiovascular effects of anesthetics.\textsuperscript{22}

The $\beta_1$ and $\beta_2$ myocardial receptors control contractility; the $\beta_3$ receptors influence relaxation. In cardiomyopathy, the density or sensitivity of the $\beta$-adrenergic myocardial receptors is decreased.\textsuperscript{23,24} Therefore, conventional doses of $\beta$-adrenergic stimulants may be inadequate and larger doses may be required.

**Anesthetic Management of the Case Presented**

After the placement of basic monitoring devices, a peripheral vein, radial artery, and central vein were cannulated. The arterial cannula was used to monitor the BP closely because sudden, significant decreases in BP during induction may be missed by noninvasive monitoring with a BP cuff. Central access was established to administer inotropes or vasopressors and to assess volume status.

Anesthesia was induced with etomidate, and tracheal intubation was accomplished with use of the muscle relaxant succinylcholine. Anesthesia was maintained with oxygen in nitrous oxide and the titrated dosing of sevoflurane. As the patient’s BP decreased, he received an infusion of norepinephrine, titrated to maintain the systolic BP at about 90 mm Hg.

A TEE probe was inserted to help the surgeon locate the coronary sinus and to monitor changes in cardiac function and volume status. The left ventricular pacing lead was placed by Seldinger technique in the left subclavian vein and was then directed into the obtuse marginal branch of the coronary sinus. Precise placement of the left ventricular lead into the branch of the coronary sinus is technically challenging. The right ventricular pacing lead was replaced with an ICD lead. The surgical incision was infiltrated with local anesthetics to minimize postoperative discomfort. Minimal fluid was infused intraoperatively.

At the conclusion of surgery, the patient regained adequate respiratory function, and his trachea was extubated. His BP stabilized with dissipation of the anesthetics, and the infusion of norepinephrine was discontinued. He was transported to the postanesthetic care unit with monitors attached and a source of oxygen. A postoperative chest x-ray revealed good positioning of the ICD leads and no evidence of pneumothorax. The patient had an uneventful postoperative course and was discharged home the following morning.

**Acknowledgment**

The author wishes to thank Charles Geller, MD, a cardiac surgeon at Beth Israel Medical Center in New York City, for his helpful suggestions during the preparation of this manuscript.

\textit{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 \textit{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, \textit{Preanesthetic Assessment}, Volumes 1 through 3 (Birkhäuser, Boston) and 4 through 6 (McMahon Publishing, New York City).}
REFERENCES


Visit www.mssm.procampus.net today for instant online processing of your CME post-test and evaluation form. There is a registration fee of $15 for this non-industry-supported activity. For assistance with technical problems, including questions about navigating the Web site, call toll-free customer service at (888) 345-6788 or send an e-mail to Customer.Support@ProCEO.com. For inquiries about course content only, send an e-mail to ram.roth@mssm.edu. Ram Roth, MD, is director of PreAnesthetic Assessment Online and assistant professor of anesthesiology at The Mount Sinai School of Medicine, New York, NY.

Post-test

1. Characteristics of patients with dilated cardiomyopathy include:
   a. reduced systolic function
   b. ejection fraction (EF) above 55%
   c. aortic regurgitation
   d. mitral stenosis

2. An implantable cardioverter defibrillator (ICD) is indicated in all of the following patients, except those with:
   a. Brugada syndrome
   b. cardiomyopathy with EF below 30%
   c. prolonged QT syndrome
   d. complete heart block

3. Angiotensin-converting enzyme inhibitors are indicated for which of the following conditions:
   a. congestive heart failure
   b. hypertension
   c. cardiomyopathy with EF below 30%
   d. all of the above

4. Which of the following is a usual feature/function of ICDs?
   a. The device is unaffected by cautery.
   b. The device is unable to distinguish between ventricular tachycardia and ventricular fibrillation.
   c. The device has the capability to reverse ventricular fibrillation.
   d. The device can improve the EF.

5. Biventricular pacing is indicated in patients with:
   a. left axis deviation
   b. low EF, congestive heart failure, and an intraventricular conduction defect
   c. diastolic dysfunction
   d. hypertension
6. The preoperative preparation of patients who have an ICD includes:
   a. discontinuing all antiarrhythmic therapy
   b. deactivating the defibrillator function of the ICD and placing external defibrillator pads if cautery is to be used
   c. avoiding the administration of depolarizing muscle relaxants
   d. initiating a lidocaine infusion

7. Which anesthetic has a minimal effect on cardiovascular function?
   a. Propofol
   b. Thiopental
   c. Isoflurane
   d. Etomidate

8. What is a potential problem in patients with cardiomyopathy undergoing anesthesia?
   a. Profound hypotension
   b. Atrial flutter
   c. Hyperkalemia
   d. Interaction of anesthetics with amiodarone

9. On chest x-rays, which feature of an ICD can be used to distinguish it from an implanted pacemaker?
   a. The right ventricular lead of the ICD has 2 radiopaque coils.
   b. The right ventricular lead of the ICD is positioned in the coronary sinus.
   c. The generator of the ICD is smaller than that of the pacemaker.
   d. The right atrial lead of the ICD has 2 shock coils.

10. Which modality does not increase ventricular function?
    a. ICD
    b. Intra-aortic balloon pump
    c. Dobutamine
    d. Milrinone