Sudden Cardiac Death and Heart Failure

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ABSTRACT

Ischemic heart disease and dilated cardiomyopathy are among the most common cardiovascular disease processes associated with heart failure that can lead to lethal arrhythmias and sudden cardiac death (SCD). With the increasing incidence of heart failure in the United States, many patients are now at risk for SCD. Nurses should understand the pathophysiology, current treatment guidelines, and the rationale for these therapies to effectively manage systolic dysfunction and to mitigate the risk of SCD. Nurses are more involved than ever with this patient population and play a key role as members of the heart failure disease management team. As a result, nurses are uniquely positioned to improve survival and reduce SCD in individuals diagnosed with left ventricular dysfunction. The purpose of this article is to increase the awareness of the risk of sudden death in patients with left ventricular dysfunction. Current evidence-based practice guidelines with rationale are reviewed.

Keywords: cardiomyopathy, heart failure, implantable cardioverter defibrillator, sudden cardiac death

Prevalence and Risk

Sudden cardiac death may happen for a variety of reasons but most notably occurs when the electrical impulses in the heart become...
rapid, usually manifested as ventricular tachycardia or ventricular fibrillation. This results in the abrupt cessation of blood flow to vital organs, with SCD ensuing if not treated promptly. Sudden cardiac death leads to approximately 335,000 deaths in the United States annually and is the cause for nearly 50% of all cardiovascular deaths. This translates to 1000 lives lost daily or 1 life every 2 minutes. Sudden cardiac death claims more deaths than breast cancer, lung cancer, and HIV/AIDS combined. Most SCD events occur outside the hospital setting, with 60% of the cases treated by bystanders or the emergency medical system. Sudden cardiac death is caused by various cardiovascular disease processes, with the highest incidence in those with coronary artery disease, hypertrophic cardiomyopathy, and dilated cardiomyopathy due to nonischemic etiologies. Fatal arrhythmias, including ventricular tachycardia and ventricular fibrillation, account for 80% of sudden death events. For SCD patients, there is a dismal survival rate of 3% to 28%. Patients who experience a myocardial infarction have an incidence of SCD that is 4 to 6 times that of the general population, and those with heart failure have a 6- to 9-fold increased risk compared with the general population. Both genders and all race/ethnic groups are at similar risk. More than 5 million patients are diagnosed with symptomatic heart failure, with an estimated 550,000 new cases diagnosed annually, which accounts for approximately 63% of all deaths due to cardiovascular disease. It is interesting to note that age-adjusted cardiovascular deaths have declined since 1979, but mortality related specifically to SCD has increased. This is likely due to the increasing prevalence of heart failure.

The rate of survival to hospital discharge for out-of-hospital SCD event is only 7.9%. For a witnessed in-hospital event, the survival increases only to 18% for adults. For each minute the patient remains in cardiac arrest, the chance of survival decreases by 10% so that by 10 minutes, the chance of survival is zero. In addition, 71% of those who survive an SCD event reported no preceding symptoms within the hour of the event. Symptoms preceding the event by more than an hour were present only in approximately 29% of patients, and these symptoms included chest pain, shortness of breath, or palpitations. Despite attempts at aggressive risk stratification utilizing diagnostic techniques including Holter monitoring, signal-averaged electrocardiograms, microvolt T-wave alternans, electrophysiology studies, or exercise testing, there has not been an effective algorithm with sufficient sensitivity and specificity to specifically and accurately identify high-risk patients. At this time, the best predictor for SCD is the presence of left ventricular dysfunction.

The literature identifies several risk factors for SCD in adults older than 35 years, which includes the presence of coronary artery disease and post–myocardial infarction, left ventricular dysfunction, dilated cardiomyopathy with impaired left ventricular function, heart failure, a history of ventricular arrhythmias, a prior history of SCD, or a family history of SCD. Even patients with mild to moderate heart failure are at risk of experiencing SCD from cardiac arrhythmias.

**Treatment Options**

**Medication Therapy**

Multiple, placebo-controlled, randomized clinical trials have proven that blocking neurohormonal systems has a significant impact on reducing mortality and sudden death and slowing disease progression in individuals with a reduced left ventricular ejection fraction. The medication regimen for patients with heart failure with reduced ventricular function typically includes angiotensin-converting enzyme (ACE) inhibitors or angiotensin receptor blockers (ARBs), β-blockers, aldosterone receptor antagonists, and a combination of hydralazine and isosorbide dinitrate, which is recommended for African Americans.

**Angiotensin-Converting Enzymes**

Angiotensin-converting enzyme inhibitors have been evaluated in more than 30 placebo-controlled studies that have enrolled more than 7000 participants. Angiotensin-converting enzyme inhibitors are considered to be first-line therapy in the management of individuals with depressed left ventricular function. In patients with mild, moderate, and severe heart failure, these medications reduce mortality and hospitalizations independent of heart failure etiology. The current guideline recommendation for the use of ACE inhibitors is class I, which denotes that the therapy should be implemented unless there is a contraindication. The first ACE inhibitor was available in 1975, and current heart failure registries still
show this class of medications to be underutilized. The maximum tolerated dose of an ACE inhibitor should be utilized to decrease hospitalizations, but research has shown that lower doses are still effective in reducing mortality.

The ATLAS (Assessment of Treatment with Lisinopril and Survival) trial enrolled 3162 patients with mild, moderate, and severe heart failure and New York Heart Association (NYHA) functional class II to IV symptoms (Table 1) and treated them with either low- or high-dose lisinopril for a period of 46 months to investigate modes of death in patients with chronic heart failure. The ATLAS trial provided data to help identify deaths caused by sudden death or those due to progressive heart failure. A reduction in the combined end point of all-cause mortality and all-cause hospitalization in the high-dose group was reported compared with the low-dose group. With this strong body of evidence, ACE inhibitors should be administered to all patients with heart failure and a reduced ejection fraction of less than 0.40 unless contraindicated. Clinicians should monitor individuals treated with ACE inhibitors for adverse effects such as hyperkalemia, worsening renal function, hypotension, and angioedema, especially when combined with other therapies that target the renin-angiotensin neurohormonal system.

Angiotensin Receptor Blockers

Angiotensin receptor blockers, as a class of medications, are beneficial in heart failure. For those patients who are intolerant to ACE inhibitors, because of adverse effects, ARBs are an acceptable alternative, and, in this scenario, the use of an ARB in heart failure is regarded as a class I recommendation. Can- desartan and valsartan are the 2 recommended ARBs in those individuals with a reduced left ventricular function. The Candesartan in Heart Failure Assessment of Reduction in Mortality and Morbidity (CHARM) alternative and added arms revealed a modest decrease in hospitalization and a reduction in mortality. The alternative arm of the CHARM study utilized candesartan in the place of an ACE inhibitor, and the added arm included candesartan in addition to an ACE inhibitor. A study of valsartan also revealed a modest decrease in hospitalization but demonstrated no effect on mortality. This class of medications has the same adverse effect profile as ACE inhibitors, such as hypotension, worsening renal function, hyperkalemia, and cough, but there is a lower incidence of angioedema. It is not recommended to treat individuals with left ventricular dysfunction with a combination of an ACE inhibitor and an ARB because of the lack of scientific evidence for this combination and the increased risk of adverse effects. Candesartan and valsartan have a potential antiarrhythmic effect through the blockade of angiotensin II receptors as well as hemodynamic and neurohormonal effects on the cardiovascular system. Because of the lack of evidence, “no specific recommendations for ARBs are included either in the ESC (European Society of Cardiology) task force on SCD or in the recently released guidelines on the management of ventricular arrhythmias and prevention of SCD.”

β-Blockers

β-Blockers in combination with an ACE inhibitor or ARB are considered to be cornerstone therapy in this high-risk population with systolic dysfunction and carry a class I American College of Cardiology/American Heart Association (ACC/AHA) guideline recommendation. β-Blockers decrease ventricular arrhythmias and the incidence of SCD in a variety of cardiovascular disorders. The combination of the sympathetic nervous system and renin-angiotensin system blockade produces a synergistic effect. β-Blockers have been studied in more than 20 placebo-controlled studies with more than 20 000 participants with left ventricular ejection fractions of less than 0.35 to 0.45. β-Blockers have been studied across the continuum from post–myocardial
infarction to severe heart failure. The current randomized clinical trials do not show a class effect for β-blockers related to heart failure, and some β-blockers have been shown to cause untoward adverse effects on cardiac function in those individuals with reduced ejection fraction. Two β-blockers currently approved for use in patients with heart failure and depressed left ventricular function are carvedilol, both immediate release and extended release, and the extended release metoprolol succinate. Bisoprolol is known to be effective in heart failure but is not approved for use in the United States. Bisoprolol and metoprolol succinate are cardioselective, and carvedilol is noncardioselective. All of these evidence-based β-blockers decrease the activation of the sympathetic nervous system and lower the risk of ventricular arrhythmias by affecting the automaticity of cardiac cells and triggered activity in the heart beat.6 The Metoprolol CR/XL Randomized Intervventional Trial in Congestive Heart Failure (MERIT-HF) and the Carvedilol Prospective Randomized Cumulative Survival (COPERNICUS) trial showed a reduction in SCD in patients with heart failure.11 Therefore, β-blockers should be prescribed to all patients with stable heart failure with a reduced left ventricular function unless contraindicated.

**Aldosterone Receptor Antagonists**

Aldosterone has been shown to have deleterious effects on the cardiovascular system, including, but not limited to, the potentiation of ventricular arrhythmias, potassium and magnesium loss, endothelial dysfunction, vascular inflammation and injury, an increase in catecholamines, and a prothrombotic state.12 Both ACE inhibitors and ARBs reduce the level of aldosterone. The use of an aldosterone receptor antagonist in addition to an ACE inhibitor or ARB and a β-blocker provides additional benefit in reducing all-cause mortality. Two trials have been performed to evaluate the efficacy of aldosterone receptor antagonists in decreasing the risk of death and heart failure hospitalization.4 The first aldosterone receptor antagonist trial in patients with depressed left ventricular function was the Randomized Aldactone Study (RALES), which evaluated the use of spironolactone in patients with NYHA functional class III and IV symptoms with a mean left ventricular ejection fraction of 0.25.11 The participants continued to receive standard medical therapy for heart failure and were initiated on low-dose spironolactone versus placebo. Mortality decreased by 30%, with a 35% decrease in heart failure hospitalization and an improvement in NYHA functional class symptoms over a 2-year period.13

The second trial randomized ischemic patients who were 3 to 14 days post-myocardial infarction with a left ventricular ejection fraction of 0.40 or less to 25 or 50 mg of eplerenone daily.6,14 The participants were followed for 36 months and the results revealed a 15% decrease in all-cause mortality and, remarkably, a 33% decrease in SCD, which is approximately the benefit realized from implantable cardioverter defibrillators (ICDs). As with ACE inhibitors and ARBs, there is an increased risk of hyperkalemia and worsening renal function with aldosterone receptor antagonists; therefore, the practitioner should closely monitor electrolytes and renal function in this patient population. These agents should be avoided as stated in the guidelines if the serum creatinine level is 2.0 mEq/dL or more (females) and more than 2.5 mEq/dL (males), or the serum potassium level is 5.0 mEq/dL or more.6 Aldosterone receptor antagonists are beneficial in patients following myocardial infarction and advanced heart failure in reducing sudden death and mortality. These agents are used in addition to ACE inhibitors and β-blockers in advanced heart failure.

**Isosorbide Dinitrate and Hydralazine**

Two studies were performed that evaluated the role of vasodilators in heart failure.15,16 The Effect of Vasodilator Therapy on Mortality in Chronic Heart Failure (V-HeFT I) trial enrolled patients to evaluate the effect of a combination of isosorbide dinitrate and hydralazine or prazosin on mortality when compared with placebo in patients with heart failure receiving digoxin and diuretic therapy.15 The Comparison of Enalapril with Hydralazine-Isosorbide Dinitrate in the Treatment of Chronic Heart Failure (V-HeFT II) trial compared the efficacy of hydralazine and isosorbide dinitrate with enalapril in patients with heart failure who were also receiving digoxin and diuretic therapy.16 Together, V-HeFT I and V-HeFT II failed to demonstrate efficacy of hydralazine and isosorbide dinitrate in the total patient population, but in a retrospective view of the data, it appeared that the combination of these
agents may be uniquely beneficial in decreasing all-cause mortality in the African American population. Therefore, the prospective African American in Heart Failure Trial (A-HeFT) was conducted and subsequently randomized more than 1000 participants self-identified as African Americans with NYHA functional class III and IV symptoms to a combination of isosorbide dinitrate and hydralazine versus placebo with an 18-month follow-up. The results were striking with a statistically significant composite score of improvement in functional capacity, decrease in mortality, and decrease in first hospitalization. When added to standard heart failure therapies, there was a 43% decrease in mortality and a 39% decrease in first hospitalization. The ACC/AHA clinical practice guidelines now recommend this therapy, class I, in African American patients with heart failure already on optimal medical therapy. Although this combination has been proven to be especially beneficial in heart failure, post hoc analyses demonstrate that the mortality benefit is a reduction in death due to progressive heart failure and not due to a reduction in SCD events.

Other Medication Therapies
Diuretics and digoxin are routinely used in the treatment of heart failure because they improve symptoms; however, they do not have a mortality benefit. Loop diuretics have been shown to increase the excretion of urinary sodium, thereby improving the patient’s symptoms of excess fluid volume. However, there have been no long-term studies to evaluate the effect of diuretics on mortality and morbidity. Blood pressure and laboratory levels, particularly levels of potassium and creatinine, must be monitored closely because of the possibility of overdiuresis and dehydration.

Digitalis has been studied in several short-term, placebo-controlled studies and a long-term trial evaluating the effect of digoxin on survival over 3 years. The long-term study of 6800 patients with NYHA functional class II or III symptoms showed a 28% decrease in heart failure hospitalizations and improvement in symptoms but did not show a benefit or an adverse effect on mortality. The risk of digoxin toxicity is significant and remains a point of caution in the use of digoxin. Therefore, it is suggested, as a class IIa recommendation, by the ACC/AHA heart failure guidelines that patients who remain symptomatic after being treated with diuretics and an ACE inhibitor or ARB and a β-blocker may be initiated on digoxin. It is recommended that plasma levels of digoxin be maintained at 0.5 to 1.0 ng/mL, with close monitoring for adverse effects.

Antiarrhythmic Agents
Sudden cardiac death is commonly the result of ventricular tachyarrhythmias. It has been estimated that 50% to 70% of patients with a depressed ejection fraction and symptomatic heart failure have episodes of nonsustained ventricular arrhythmias. Antiarrhythmic drug therapy has not been shown to improve survival in any of the currently published randomized, prospective, controlled trials in this high-risk population. Multiple trials have evaluated the efficacy of antiarrhythmic agents in the prevention of SCD in patients with depressed ejection fractions. Some trials of antiarrhythmic agents have outcomes related to either negative inotropic effects or increased incidence of arrhythmias. Class Ia antiarrhythmic agents (eg, quinidine and procainamide), class Ic antiarrhythmic agents (eg, flecainide and propafenone), and certain class III agents (eg, D-sotalol) have been shown to worsen outcomes and increase mortality. Amiodarone is a class III agent that has been studied in several trials and found to have at least a neutral effect on mortality when prescribed for patients with depressed left ventricular function. Amiodarone therapy is not considered to be standard care in this patient population but may be used in patients who require antiarrhythmic suppression for recurrent atrial fibrillation or symptomatic ventricular tachycardia. Sotalol, like amiodarone, has also been used in patients with ventricular arrhythmias and heart failure but has not been shown to improve survival from SCD.

Exercise Training
The lack of exercise in patients with heart failure and reduced left ventricular function often leads to deconditioning and progressive physical symptoms. There have been multiple randomized controlled trials to evaluate the effect of exercise on quality of life, exercise capacity, symptoms, and mortality. Only 1 study has evaluated the long-term effects of exercise on mortality and hospital admissions. The results demonstrated a reduction in mortality and hospital readmissions for heart failure.
more recent studies have not shown benefits of exercise in patients with heart failure. Currently, cardiac rehabilitation is not reimbursed for a diagnosis of heart failure or cardiomyopathy but the ACC/AHA guidelines give exercise training a class I recommendation.

Evidence-Based ICD Recommendations
This section provides a review of some of the significant randomized clinical trials that have evaluated the effectiveness of ICDs: (1) the Multicenter Automatic Defibrillator Implantation Trial (MADIT), (2) the Multicenter Automatic Defibrillator Implantation Trial-II (MADIT-II), (3) the Multicenter Automatic Defibrillator Implantation Trial-II at Eight Years, and (4) the Sudden Cardiac Death in Heart Failure Trial (SCD-HeFT).

MADIT was the first randomized primary prevention clinical trial to evaluate the effectiveness of an ICD compared with standard medical therapy in a population of patients with post–myocardial infarction with impaired left ventricular function. MADIT enrolled 196 patients with a Q-wave or enzyme-positive myocardial infarction, nonsustained ventricular tachycardia of less than 30 beats, inducible ventricular tachycardia that could not be suppressed, and a left ventricular ejection fraction of 0.35 or less. Patients were excluded if they had a percutaneous coronary intervention within the last 2 months or coronary artery bypass graft within the last 3 months. The outcomes of MADIT after a mean follow-up period of 27 months showed a 54% reduction in all-cause mortality and a 75% reduction in mortality caused by arrhythmias in the ICD group compared with standard medical therapy.

MADIT-II was a randomized clinical trial to evaluate the effectiveness of an ICD compared with medical therapy in patients with post–myocardial infarction with impaired left ventricular function. MADIT-II enrolled 196 patients with a Q-wave or enzyme-positive myocardial infarction, nonsustained ventricular tachycardia of less than 30 beats, inducible ventricular tachycardia that could not be suppressed, and a left ventricular ejection fraction of 0.35 or less. Patients were excluded if they had a percutaneous coronary intervention within the last 2 months or coronary artery bypass graft within the last 3 months. The outcomes of MADIT after a mean follow-up period of 27 months showed a 54% reduction in all-cause mortality and a 75% reduction in mortality caused by arrhythmias in the ICD group compared with standard medical therapy.

MADIT-II was a randomized clinical trial to evaluate the effectiveness of an ICD compared with medical therapy in patients with post–myocardial infarction with impaired left ventricular function. This study enrolled 1232 ischemic patients who had NYHA class I to III symptoms with an ejection fraction of 0.35 or less, and a left ventricular ejection fraction of 0.35 or less. Patients were excluded if they had a percutaneous coronary intervention within the last 2 months or coronary artery bypass graft within the last 3 months. The outcomes of MADIT after a mean follow-up period of 27 months showed a 54% reduction in all-cause mortality and a 75% reduction in mortality caused by arrhythmias in the ICD group compared with standard medical therapy.

MADIT-II was a randomized clinical trial to evaluate the effectiveness of an ICD compared with medical therapy in patients with post–myocardial infarction with impaired left ventricular function. This study enrolled 1232 ischemic patients who had NYHA class I to III symptoms with an ejection fraction of 0.35 or less. The mean follow-up period was 20 months. MADIT-II identified a QRS duration of 120 milliseconds as an independent risk factor for mortality. The outcomes of MADIT-II revealed a reduction in overall mortality by 31% and a reduction in arrhythmic deaths by 61%. This study paved the way for the recommendation to implant ICDs in patients with post–myocardial infarction with a reduced left ventricular function to prevent SCD.

Recently, there was an 8-year follow-up to MADIT-II that revealed an increase in life year saved from 0.2 to 1.2. In MADIT-II, the cost for ICD therapy for 3.5 years of extra life was estimated to be $235,000, and with the mortality curves continuing to separate at 8 years, the cost per life year saved decreases to $50,000. During the 8-year follow-up of MADIT-II, the number of patients needed to treat with an ICD in order to save a life decreased from 17 to 6. It is important to note that for this benefit to be realized, a patient must be free of other life-threatening conditions because the benefit of the ICD is not short term and accumulates over time.

SCD-HeFT is the largest primary prevention ICD trial. It was a multicenter, randomized, controlled trial that enrolled 2521 patients with ischemic or nonischemic dilated cardiomyopathy and NYHA class II or III symptoms. Patients enrolled in this trial were not required to undergo risk stratification to demonstrate ventricular dysrhythmias. Patients were required to have a left ventricular ejection fraction of 0.35 or less, on heart failure therapy for at least 3 months, and be on optimal medical therapy for heart failure including ACE inhibitors and β-blockers. Participants were randomized to 1 of 3 arms: standard medical therapy, standard medical therapy with a single-lead ICD that provided shock-only therapy, and standard medical therapy with amiodarone. Participants were followed for 5 years. The study revealed similar outcomes for the placebo group and the amiodarone group, but the defibrillator group had a significant 23% reduction in the risk of SCD.

These studies have led to an increase in the number of ICDs over the last couple of decades, moving from secondary prevention (survivors of an SCD event) to higher use in primary prevention (patients who have not experienced an SCD event). Patients who are at high risk have either ischemic or nonischemic left ventricular dysfunction with a left ventricular ejection fraction of less than 0.35.

The most recent ACC/AHA treatment guidelines for heart failure released in March 2009 recommend
ICD therapy for primary prevention of sudden cardiac death to reduce total mortality in patients with nonischemic dilated cardiomyopathy or ischemic heart disease at least 40 days post–myocardial infarction, with an LVEF less than or equal to 35%, NYHA functional class II or III symptoms while receiving chronic optimal medical therapy, and who have reasonable expectation of survival with a good functional status for more than 1 year as a Class I recommendation.4,20

The ICD is considered to be standard therapy in the prevention of SCD. The ICD has been proven through multiple randomized trials to be the only effective therapy in the treatment of SCD due to lethal arrhythmias.6,30

Evidence-Based Cardiac Resynchronization Recommendations

Despite the advances in medical therapy that target the neurohormonal axis, many patients with heart failure remain symptomatic with poor quality of life and high mortality rates. Several large long-term cardiac resynchronization therapy (CRT) trials with pacing capability (with or without a defibrillator) have shown a mortality and morbidity benefit. The Cardiac Resynchronization Therapy with or without an Implantable Defibrillator in Advanced Chronic Heart Failure trial (COMPANION) enrolled 1520 patients with NYHA functional class III or IV symptoms in 1 of 3 arms: optimal medical therapy alone, optimal medical therapy with CRT with pacing only (CRT-P), and optimal medical therapy with cardiac resynchronization and a defibrillator (CRT-D).31 The patients enrolled had ischemic or nonischemic etiology. The primary end point was combined all-cause mortality and all-cause hospitalization. A 19% risk reduction in the primary end point with CRT-P was reported compared with optimal medical therapy and a 20% risk reduction with CRT-D. The risk of the combined end point of death or hospitalization due to heart failure was reduced by 34% in the pacemaker group and by 40% in the pacemaker-defibrillator group.19

The effect of CRT on morbidity and mortality in heart failure trial (CARE-HF) enrolled 813 participants to either standard medical therapy or CRT. The mean follow-up was 29 months.32 The inclusion criteria were NYHA functional class II through IV symptoms, left ventricular ejection fraction of 0.35 or less, a QRS duration of 120 milliseconds or longer with evidence of left ventricular dyssynchrony, and receiving optimal medical therapy. The primary end point of combined all-cause mortality or cardiovascular hospitalization was significantly reduced by 37% compared with the optimal medically treated group. The secondary end point of all-cause mortality decreased by 36% in the CRT group as compared with the optimal medical treatment group.32 Echocardiographic evidence was present for left ventricular reverse remodeling, with a decrease in the amount of mitral regurgitation and decreases in left ventricular end-systolic volume index and interventricular mechanical delay.27 Individuals have also reported an improvement in functional capacity, in NYHA functional class symptoms, and quality of life.

The current ACC/AHA guidelines for chronic heart failure recommend CRT as a class I indication and recommend it for individuals receiving optimal medical therapy, a left ventricular ejection fraction of 0.35 or less, a QRS duration of 120 milliseconds or longer, and NYHA functional class III or ambulatory class IV symptoms.30 Individuals treated with devices need to be aware that the device is an additional therapy option and does not take the place of their current heart failure medical regimen.

Patient Education Related to Devices

Nurses can play an important role in the prevention of SCD through community education, knowledge of current evidence-based guidelines, and identifying those patients at high risk for SCD. Nurses should stay abreast of the new recommendations for cardiopulmonary resuscitation and advanced cardiac life support to care for patients and educate family members. We should encourage family members of those individuals at high risk to enroll in cardiopulmonary resuscitation classes.

Devices are not without complications, such as device failure, lead failure, and inappropriate device discharges, and nurses will need to recognize potential complications. Many psychological stressors occur with device implants when a patient anticipates or receives a shock. Issues such as anxiety, panic attacks, and depression may occur at the time of implant or any time thereafter.
A goal for nursing is to educate and provide resources for patients, family members, and the public on preventing sudden cardiac arrest. Providing education prior to implant will help alleviate many of patients’ and family members’ concerns and anxieties. The anxieties are often related to fear of the unknown. Clinicians should fully explore any patient concerns prior to implant and focus on the ICD not just being a “shock box” but also a tool with diagnostic features that can assist the clinician in managing patients with heart failure. Key education points for the clinician to consider are as follows: emotions such as fear and anxiety, what patients may experience when receiving a shock, daily activities, physical activity, intimacy, driving, travel, precautions with medical procedures such as magnetic resonance imaging (MRI), travel through airports, and patient preparation in case of an emergency.

Several techniques to alleviate patients’ fears about the CRT and/or the ICD include focusing on the improved quality of life with the CRT and preventing a sudden death event with an ICD. Discuss concerns with patients and suggest that they make a list of these concerns to review with their health care provider and significant other in order to develop a plan to cope. Provide education to help cope with negative thinking through the use of biofeedback, meditation, becoming engaged in a pleasant activity, or focusing on an upcoming family event such as a wedding, anniversary, or birthday. Nurses may provide patients with several helpful Web sites to help the patients become more knowledgeable or suggest that patients attend a support group meeting or talk with other patients in similar situations.

Discuss the possibility of receiving a shock from the device and encourage the patient to stay calm because the device is likely doing its job. After the ICD has delivered a shock, encourage the patient to record the type of activity at the time of the shock and the presence of any prodromal symptoms. The patient should be aware of the process after receiving a shock, such as notifying the health care provider who manages the device. The patient’s family member will need to call 911 if the patient remains unconsciousness for more than a minute following the device discharge.

Patients have concerns about daily activities and should be advised that using everyday items such as cell phones is not contraindicated, but the phone should be placed at least 6 in from the CRT and/or the ICD. Other items such as wireless-enabled computers and products that contain small magnets or transmitters should also be placed 6 in from the pacing/defibrillator devices. Most work environments are perfectly safe, but environments or equipment that produce electromagnetic fields may affect the functioning of the device, and patients should discuss this with their health care provider.

Most daily activities including hobbies, employment, exercise, intimacy, and travel may be resumed in approximately 2 to 6 weeks postimplant. Patients should avoid rough physical activities that will cause a direct impact on the device or the device site. Patients need to discuss activities such as hunting and scuba diving with their health care provider before resuming activities. Most patients are able to resume driving 2 to 6 weeks postimplant after the device site has healed if the device was placed for primary prevention. Most practitioners recommend not driving for 6 months if the device was placed for secondary prevention due to a recent event. Before resuming driving, patients should talk to their practitioner and look at the individual state department’s public safety guidelines.

Travel is safe postimplant, but if traveling by plane, the airport security systems will detect the device and the patient may require a hand search. Patients should have their device identification card available. Whatever the mode of travel, patients should be prepared for emergencies and should wear a medical emergency bracelet or necklace. Examples of areas that need to be avoided when traveling are areas where there are strong hydroelectric systems, such as touring inside Hoover Dam.

Many medical and dental procedures are safe, including routine dental procedures, diagnostic radiology studies, electrocardiograms, mammograms, laser surgery, positive emission tomography scans, bone densitometry, and the use of sleep apnea machines. Several medical diagnostic procedures that warrant precautions and should be discussed with the patient’s health care provider include external defibrillation, electrocautery, electrolysis, high-energy radiation, and lithotripsy. Magnetic resonance imaging procedures should be avoided in patients with a CRT-D or an ICD. Currently, MRI-compatible devices are being studied but they have not been approved.

Patients should be prepared for emergencies at all times. They should be advised to carry...
their device identification card, a list of their current medications, emergency phone numbers, and the necessary steps to take in the event they receive a shock.

A discussion regarding end-of-life issues should take place prior to the device implant. When death is imminent, patients should be aware that they have a choice in deciding if they want the CRT deactivated to prevent repeated episodes of device shocks. Very few situations exist that would warrant the CRT being deactivated, but 1 instance would be if the patient has irreversible cognitive disorders and the device is not meeting the goals of care.27

**Summary**

Sudden cardiac death remains a major health problem and will continue to increase with an increase in the number of high-risk patients. Current methods to identify this high-risk population remain inadequate, and nurses practicing in any domain are likely to care for patients at risk for SCD. With the continuing growth in heart failure diagnosis, knowledge of the current practice guidelines and the rationale for these interventions will ensure patients receive the best quality care available. Multiple registries have demonstrated that current therapies known to improve outcomes in heart failure are being underutilized.8 It is our responsibility as health care professionals to ensure that patients are receiving optimal medical therapy and being referred for device therapy if they meet the current guideline indications. This is our opportunity as nurses to provide education, support, direct interventions, and referrals to other disciplines as needed. Nursing can also be very effective in educating colleagues in various venues in the current management of advanced heart failure and the risk of sudden death.

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**References**


