Clinical trials have established the superiority of the implantable cardioverter-defibrillator (ICD) over antiarrhythmic drug therapy in survivors of sudden cardiac death and in high-risk patients with coronary artery disease. The ICD has evolved to overcome the limitation of earlier devices that required thoracotomy for implantation and were fraught with inappropriate shock delivery. Current ICDs are implanted in a similar manner to cardiac pacemakers and incorporate sophisticated rhythm-discrimination algorithms to prevent inappropriate therapy. Managing the patient with an ICD requires an understanding of the multiprogrammable features of modern devices. Drug interactions and potential sources of electromagnetic interference may adversely affect ICD function. Driving restrictions may be necessary under certain conditions. The cost-effectiveness of ICD therapy appears favorable, given the marked survival benefit seen in randomized trials relative to antiarrhythmic drug treatment. The growing number of ICD recipients necessitates an understanding of the specialized features of the modern ICD and the role of device therapy in clinical practice.

In the United States alone, sudden cardiac death (SCD) accounts for an estimated 350,000 lives lost annually.1 The majority of these events occur outside the hospital setting and are associated with consequential delays in implementing external defibrillation. The ability of the implantable cardioverter-defibrillator (ICD) to provide therapy within 5 to 15 s of arrhythmia detection allows for defibrillation success rates approaching 100%.

Numerous clinical trials have established the superiority of the ICD over drug therapy in reducing mortality rates for survivors of cardiac arrest or patients at high risk for SCD.2–7 On the basis of these studies, the ICD is now recommended as the treatment of choice in survivors of SCD and in patients with symptomatic, sustained ventricular arrhythmias.8 Current ICD implantation rates exceed 30,000 per year and will continue to grow as clinical indications evolve.9 The majority of physicians will be involved in the care of ICD recipients. In this review, we provide the generalist with an overview of ICD function and the management of ICD patients in clinical practice.

**The Modern ICD**

**Early Beginnings**

The first human ICD implant occurred in 1980.10 Approval for general use was granted by the US Food and Drug Administration in 1985. These early devices consisted of a large pulse generator (PG)
(Fig 1) and patch electrodes for defibrillation placed directly on the heart or pericardium. Epicardial screw-in leads also were placed for rate sensing (Fig 2). Implant procedures required general anesthesia, thoracotomy, were associated with longer hospital stays, and had a perioperative mortality rate in the range of 4%.11

In addition to the limitations imposed by size, early devices offered few options for patient-specific programming. The ICD was ordered by the physician specifying a detection heart rate and delivered by the manufacturer to perform defibrillation should the patient’s ventricular rate exceed this value. No programming options existed for changing the pre-specified detection rate. Therefore, should an anti-arrhythmic agent be required and have the effect of slowing ventricular tachycardia (VT) below the preset detection rate, ICD therapy would not be delivered. A further limitation of early devices was the inability to discriminate rapid ventricular rhythms of supraventricular origin from ventricular-based rhythms, leading to a high incidence of inappropriate shocks. Analysis of stored electrograms (EGMs) in later device models confirmed a rate of inappropriate shocks in the range of 25 to 40%.12–15

Advances in ICD lead systems and defibrillation waveforms allowed for successful transvenous, pectoral ICD implants (Fig 3). ICD systems are now implanted in a manner similar to cardiac pacemakers and without the need for general anesthesia. Perioperative mortality is < 1%.16 Complications such as infection, pneumothorax, pericardial tamponade, and pocket hematoma occur at rates similar to those seen with pacemaker implantation (< 3%).17,18 As ICD system hardware has evolved, so too has the internal circuitry responsible for the specialized features of the device. The modern ICD may now be programmed to detect several specified tachycardia zones, with the alternative of less aggressive antitachycardia pacing (ATP) therapy for slower, hemodynamically stable VT. Further, sophisticated rhythm-discrimination algorithms now exist to discern VT from rapid supraventricular arrhythmias. This has resulted in the decline of the inappropriate shock rate to < 5%.19,20

**ICD Rhythm Discrimination: Rapid Supraventricular Tachycardia vs Ventricular Arrhythmia**

Current ICDs allow for the programming of at least three tachyarrhythmia detection zones. The highest rate zone is referred to as the ventricular fibrillation (VF) zone. In the VF zone, the risk of sudden hemodynamic collapse mandates 100% sensitivity in detection. This is achieved by employing an XY algorithm that requires a certain proportion (eg, 12/16) of R-R intervals to be within the programmed zone for detection. Additional slower VT zones may be programmed for detection of more hemodynamically stable ventricular arrhythmias. In contrast to the more rapid rate of the VF zone, rate criterion as the sole method for detection in VT zones results in a significant number of inappropriate shocks.
Most unnecessary therapies are due to sinus tachycardia or atrial fibrillation with ventricular rates overlapping with programmed VT detection zones. Therefore, rhythm-discrimination algorithms have been developed to prevent inappropriate therapy and to increase specificity for VT within a programmed VT zone. Unfortunately, increasing specificity is at the expense of diminishing sensitivity. Thus, all rhythm-discrimination algorithms are programmable only for the presumed more hemodynamically stable, slower VT zones.

The two most commonly employed rhythm-discrimination algorithms utilize onset and stability reference criteria. The onset algorithm monitors for abrupt changes in the R-R interval from beat to beat. In the scenario of sudden-onset tachycardia within a VT zone, should the R-R interval of the first two beats of tachycardia decrease (reflecting an increased heart rate) by more than the programmed onset criterion (e.g., 40 ms), then the tachycardia is considered to be ventricular in origin. The onset algorithm is intended to prevent inappropriate therapy for sinus tachycardia rates that progress into the slow VT zone. Thus, a gradual change in R-R interval entering a VT zone, as is the case in sinus tachycardia, would not satisfy onset criteria and would be considered supraventricular in origin, averting therapy. The limitation of this feature is the potential failure to detect VT that arises during exercise and lacks abrupt onset. The onset criterion has been reported to underdetect 5 to 13% of VTs.

Stability refers to the R-R interval variability of the detected tachycardia. Ventricular rates due to atrial fibrillation would be expected to show a wide range of R-R intervals, as opposed to monomorphic VT. A programmable R-R stability algorithm would detect as VT any rhythm with R-R intervals varying less than the programmed value, for example, 40 ms. A potential difficulty is polymorphic VT, although this rhythm tends to be faster and detected within the VF zone.

The availability of dual-chamber ICD systems with the capability of A-V sequential pacing and sensing has allowed the design of newer enhancement-detection algorithms. The sensing of atrial EGMs permits comparison of atrial and ventricular rates. Rapid tachycardias with an atrial rate more than the ventricular rate would appropriately be detected as supraventricular in origin. Similarly, dissociation of atrial and ventricular rates during tachycardia may be recognized, accurately identifying the rhythm as VT.

The assessment of ventricular EGMs has been incorporated as another method to differentiate VTs from supraventricular tachycardias. The EGM width criterion is based on the assumption that ventricular-based rhythms will have a wider EGM compared to EGMs derived from normal AV conduction. While the addition of this criterion to onset and stability significantly lowers inappropriate shocks, it is known that EGM width can change significantly at high sinus rates and over time. Morphology discrimination (MD; Ventritex; Sunnyvale, CA) is a novel algorithm whereby the device stores a template of the baseline (sinus) ventricular EGM morphology (Fig 4). During tachycardia detection, each complex is compared to the template, and the algorithm determines the percent match. Should the tachyarrhythmia EGM complex be less than a programmable percent match, therapy is initiated. Limitations of
EGM morphology exist for patients with underlying bundle branch block or rate-related bundle branch block.

Most electrophysiologists will utilize rhythm-discrimination algorithms individually or in combination, depending on the clinical history of the patient. Although these features increase detection specificity and lower inappropriate shock rates to < 5%, underdetection and inhibition of therapy for true VT may occur. A programmable safety feature known as sustained rate duration can apply therapy if the heart rate remains in a VT zone over a programmed duration of time, thereby returning sensitivity for true VT to near 100%.19,20,26

**ICD Therapy**

All ICDs use electrical defibrillation as the only therapeutic option for heart rates detected in the VF zone. Multiple or tiered therapeutic options are available for VT detection zones. A sequence of therapy is programmable and is followed as needed until an episode is terminated. These programmable options include ATP, low-energy synchronized cardioversion, and defibrillation.

The objective of ATP is to terminate monomorphic VT promptly with little discomfort to the patient. Delivery of ATP is applied in successive paced beats at a rate faster than the tachycardia cycle length. ATP therapy successfully terminates approximately 90% of episodes of spontaneous VT.29 In addition, the use of this therapy is associated with a statistically significant 36 to 28% reduction of first ICD shocks over a 2-year follow-up period.30 The risk of this therapy is the potential for accelerating VT to VF, which may occur in up to 10% of attempts.31 Therefore, tiered therapy must always include the programming of back-up defibrillation.

**Diagnostic Storage**

Early ICDs stored little information, noting only the number of device discharges. Current devices store and display extensive data. An updated therapy history is provided on each interrogation. A large number of episodes may be stored and their details viewed individually. Specifics regarding the date/time, therapy delivered, detection zone and criteria satisfied, and total length of the episode are displayed. Intracardiac EGMs of episodes requiring therapy are retrievable, including the onset segment. Marker annotations indicating R-R intervals and the device’s interpretation of the ongoing rhythm are displayed. Simultaneous EGMs from atrial and ventricular leads may be viewed, assisting the physician in determining the appropriateness of the applied therapy (Fig 5).

**Clinical Trials of the ICD**

**Secondary Prevention of SCD**

The impact of the ICD on survival in patients with a history of life-threatening arrhythmias has now been assessed in three randomized trials (Table 1).

The Antiarrhythmics Versus Implantable Defibrillator Trial (AVID)2 evaluated the efficacy of the ICD in reducing total mortality in patients with an ejection fraction (EF) < 40% and a history of SCD or sustained VT with syncope or hemodynamic compromise. In the control arm of the study, 90% of patients received empiric amiodarone, and the remainder received Holter-guided sotalol. This trial was terminated early due to a clear benefit in the ICD-treated group. At follow-up after 3 years, survival rates were 75% for the ICD group vs 61% for the antiarrhythmic group. Although β-blocker use
was more prevalent in the ICD group, adjusting for this imbalance did not alter the mortality rate reduction attributable to the ICD.

The Cardiac Arrest Study-Hamburg Trial\(^4\) randomly assigned survivors of cardiac arrest to ICD or to treatment with propafenone, metoprolol, or amiodarone. The propafenone arm was withdrawn early due to an observed excess mortality rate. The all-cause mortality rate was 12.1% in the ICD arm vs 19.6% in the combined arms of amiodarone and metoprolol at follow-up after 2 years, a statistically significant reduction. Interestingly, no difference was observed between the amiodarone and metoprolol arms.

**Figure 5.** Intracardiac EGMs. **Top:** the panel illustrates the successful termination of VT by a single burst of ATP. **Middle:** the panel demonstrates the electrical cardioversion of VT. **Bottom:** the panel indicates a rapid supraventricular tachycardia with ventricular rates detected in a VT zone. Rhythm-discrimination algorithms correctly identified this as atrial fibrillation/flutter (AF) and prevented inappropriate therapy. AR = atrial sensed beats during pacing refractory periods; AS = atrial sense; CD = charge delivered; TD = tachycardia detected; TP = antitachycardia paced beat; TS = tachycardia sense; VR = ventricular sensed beats during pacing refractory periods; VS = ventricular sense VP = ventricular paced beat.
The Canadian Implantable Defibrillator Study (CIDS) randomized a patient population, which was similar to the one in the AVID trial, to an ICD or to empiric amiodarone. At 5 years, a trend in favor of reduced all-cause mortality in the ICD treatment group was present, although this was not statistically significant. The trial design of CIDS may have lessened the impact of ICD therapy in this study. In contrast to the AVID trial, this study did not mandate a poor EF for all patients enrolled. Included in enrollment were patients with syncope without spontaneous VT who subsequently underwent electrophysiologic (EP) study. Evidence from an EP study of inducible VT satisfied enrollment criteria, without regard to EF. This is reflected in the difference between the average EF for the CIDS patient population (34%) and that for the AVID population (27%). The difference in EF between the patient populations of the CIDS and the AVID trial also extends to New York Heart Association (NYHA) heart failure class, a known correlate to the risk of SCD. In CIDS, >50% of patients were of asymptomatic heart failure status, whereas in the AVID trial >70% of patients had NYHA class II-III heart failure. Thus, the results of CIDS suggest that patients with symptomatic VT and milder degrees of left ventricular (LV) dysfunction (EF, >35%) may be adequately treated with amiodarone, whereas patients experiencing episodes of VT who have poorer EF values and NYHA status are best treated with ICD implantation. Indeed, a subgroup analysis of CIDS indicates a much stronger trend to improved survival in ICD-treated patients with EFs <35%.

Presently, treatment with an ICD is a class I indication for secondary prevention in survivors of cardiac arrest not due to a reversible cause, for patients with syncope of unknown etiology and inducible VT/VF, and in patients with spontaneous, sustained VT (Table 2).8

Primary Prevention of SCD

Since only 2 to 30% of persons who have cardiac arrest survive, a strong impetus to identify high-risk patients for the primary prevention of SCD ex-
The recently completed Multicenter Unsustained Tachycardia Trial (MADIT) was the first prospective, randomized trial assessing the value of the ICD. Patients with prior myocardial infarction (MI), LV EFs < 35%, evidence of documented nonsustained VT (NSVT) and inducible VT on EP study that was not suppressed by IV procainamide randomly received an ICD or conventional therapy. A statistically significant difference in the total mortality rate was observed, 15% in the ICD group and 39% in the conventional therapy group over an average follow-up period of 2.5 years. Although a striking benefit for the ICD was present, the trial was criticized for the lack of a unified approach to drug therapy in the conventional treatment group. At last patient contact, only 5% of patients in the conventional group were receiving β-blocker therapy vs 27% in the ICD group. The proportion of participants in the conventional group receiving therapy with angiotensin-converting enzyme inhibitors was 51%, and the proportion receiving amiodarone was 45%.

The recently completed Multicenter Unsustained Tachycardia Trial randomized a patient population similar to that in the MADIT. Patients with inducible VT received medical therapy alone (excluding antiarrhythmic drugs) or an EP-guided approach. Patients in the EP-guided group whose conditions were suppressible by antiarrhythmic drug therapy were maintained on regimens with their respective drugs. Patients with VT not suppressible by antiarrhythmic drug therapy received an ICD. At a median follow-up of 39 months, a statistically significant benefit was evident with respect to the primary end point of SCD or resuscitated cardiac arrest in the EP-guided group (25%) compared to that in the medical therapy group (32%). Further analyses indicated that the benefit seen with the EP-guided approach was due solely to the ICD group (p < 0.001). Patients receiving EP-guided antiarrhythmic drug therapy did not show improved outcomes compared to medically treated patients not receiving antiarrhythmic drug therapy. Patients enrolled in this trial who were not inducible at EP study, and therefore were considered to be at low risk for SCD, were followed-up in a registry. Interestingly, this patient population had an unexpectedly high mortality rate of 24% at follow-up.

The Coronary Artery Bypass Graft-Implantable Cardioverter-Defibrillator Study (CABG-PATCH) study randomized patients prior to coronary surgery with EFs < 35% and positive signal-averaged ECGs to either the ICD group or the control group.
difference in overall mortality was observed. The study did not require NSVT or inducible VT for trial enrollment. It is suggested that patients with a similar degree of LV dysfunction but without inducible VT may be at a lower risk for SCD. In addition, revascularization may have decreased the risk for ischemia-induced arrhythmias in this patient population. These factors may help to explain the much lower mortality rate in the CABG-PATCH control arm (18%) than in the MADIT control group (39%).

The ongoing Sudden Cardiac Death in Heart Failure Trial will present the most stringent control intervention thus far vs the ICD. Patients with symptomatic (ie, NYHA class II-III) ischemic or nonischemic cardiomyopathy and EFs < 35% are being randomized to treatment with ICD, amiodarone, or placebo. A key component to this trial is the strong encouragement for β-blocker use, targeting 70% of the patient population. The relevance of this is highlighted by recent trials indicating a significant reduction in all-cause and sudden death mortality in congestive heart failure patients randomized to treatment with β-blockers. Furthermore, the combination of β-blocker and amiodarone has been suggested in post hoc analyses to have a significant impact on reducing the number of deaths from arrhythmia.

In summary, the current evidence suggests that patients with a history of cardiac arrest or sustained VT and syncope are best treated with an ICD. Patients with EFs < 35%, coronary artery disease, and NSVT should be referred for EP study. If inducible, they should receive an ICD.

MANAGING PATIENTS WITH ICDs
Troubleshooting Inappropriate ICD Function

The first issue in assessing a patient with a recent defibrillation discharge is to determine whether therapy was appropriate. Despite the sophisticated rhythm-discrimination algorithms that are in current use, rapid atrial fibrillation remains the most common cause of inappropriate shocks. The main reason for this is atrial fibrillation ventricular rates that meet VF zone criteria in which detection is based on the ventricular rate alone and the programming of rhythm-discrimination algorithms is not permitted. Preventive options include increasing the VF detection rate, adding AV nodal blocking agents, or considering AV nodal ablation. Inappropriate therapy also may result from the sensing of electrical chatter. Electrical chatter develops in the presence of lead fractures or insulation breaks, or may result from a loose connection to the PG. These problems may be evident on analysis of stored EGMs in which nonphysiologic R-R intervals (ie, those of < 150 ms) may be observed. Lead problems should be suspected on device interrogation when marked variation of R-wave sensing, pacing thresholds, and impedance measurements exist. Occasionally, having the patient perform physical maneuvers such as straining or arm movements may reproduce electrical chatter while observing ICD telemetry. An overpenetrated chest radiograph may localize a lead fracture. Issues related to lead failure or to loose connections require surgical intervention. Acute management of a patient with inappropriate, incessant shocks not caused by a ventricular arrhythmia should include placing a magnet over the device. This maneuver will disable therapy delivery but will have no effect on required pacing.

The inability of therapy to convert an arrhythmia may have dire consequences. Lead failure or malposition may be a culprit, resulting in insufficient energy delivery. Altered tissue substrate may render ATP unsuccessful due to failure to adequately capture. Defibrillation thresholds (DFTs) may be increased due to amiodarone initiation or to severe electrolyte/acid-base abnormalities. Increasing the programmed shock energy level may suffice, but a thorough device evaluation is required. Managing an acute ventricular arrhythmia not successfully terminated by ICD therapy should not differ from the approach used in patients without ICDs. An ICD discharge during patient contact by a resuscitating team member will not harm or pose a risk to the individual.

Antiarrhythmic Drug-ICD Interactions

ICD implantation may obviate the need for long-term antiarrhythmic drug therapy in a large number of patients. However, frequent shocks due to atrial tachyarrhythmias and/or ventricular arrhythmias will require the initiation of drug therapy in the management of certain patients. In addition to the possibility of decreasing VT rates out of programmed detection zones, therapy with antiarrhythmic drugs also may cause proarrhythmia. An increase in ICD therapies correlating with recent initiation of an antiarrhythmic agent should raise this suspicion.

The effect of antiarrhythmic drugs on the minimum energy requirement for successful defibrillation, or DFT, requires special attention. At ICD implantation, the DFT is determined and a 10-J safety margin typically is added to ensure defibrillation efficacy. Some antiarrhythmic agents may alter the DFT as a result of their EP properties (Table 3). In general, class IA antiarrhythmic drugs, such as quinidine and procainamide, appear to have little
effect on the DFT at therapeutic doses. The short-term administration of class IB agents, including lidocaine, phenytoin, and mexiletine, has been shown to increase the DFT. The class III agents amiodarone and sotalol are commonly used for the maintenance of sinus rhythm in patients with paroxysmal atrial fibrillation. Their effects on the DFT are disparate. Amiodarone has been shown to falsely detect tachycardia and to deliver inappropriate therapy. In the home environment, there have been no reports of normally functioning domestic appliances causing any inappropriate shocks in modern ICDs. Microwave ovens, portable telephones, or personal computers have not been linked to interference in current ICD systems. Inappropriate shocks from electric razors have been reported rarely. Hand-held radiofrequency remote controls may produce inappropriate sensing when held in close proximity to the ICD device but have not been shown to adversely affect device function when held > 10 cm from the chest wall.

Patients should be advised to avoid the strong magnetic fields of electronic theft surveillance systems or to walk through them without pausing. Similarly, patients should present their device identification card to airport security personnel and walk briskly through the security gate. The ICD device may trigger the alarm. In these instances, brief passage of a hand-held metal detector over the device is innocuous; however, prolonged exposure (ie, > 30 s) should be avoided as this may inactivate programmed therapies in some devices. Caution is needed in the use of cellular telephones. When used, the phone should be held on the side opposite to the device. The avoidance of close contact with the PG and lead system is recommended, and, therefore, phones should not be placed in nearby pockets.

Patients with occupational exposures to large magnetic fields may have their work environment assessed by an ICD company representative for the possibility of inappropriate device sensing.

In the hospital setting, MRI imaging is generally contraindicated. The use of electrocautery in surgery may cause electrical chatter and oversensing. The device is best turned off in this setting with appropriate monitoring and an external defibrillator nearby. In addition, electrocautery should be avoided in close proximity to the device to prevent the risk of damage to internal circuitry.

ICDs and Driving

The privilege of driving is a quality-of-life issue for ICD recipients. Although the presence of an ICD will terminate sudden, malignant ventricular arrhythmias, loss of consciousness may not be prevented in up to 15% of episodes. Current data suggest that the risk of fatal motor vehicle accidents involving ICD recipients is low. European data indicate that only 1.5 to 3.4% of road accidents are attributed to sudden driver incapacity. A study observing the driving history of 291 ICD patients over an average of 3 years noted only 11 traffic accidents. Of these accidents, no fatalities occurred and there were no accidents associated with driver syncope or defibrillation therapy. In the remaining patient cohort, 5% of patients received ICD therapy while driving, which was not associated with syncope or accident.

At present, most US states have no specific laws regarding driving for ICD patients. A consensus statement providing recommendations has been published by the North American Society of Pacing and Electrophysiology/American Heart Association. These guidelines recommend against noncommercial driving for a period of 6 months following ICD implantation for survivors of life-threatening arrhythmias. Similarly, following successful ICD therapy for a ventricular arrhythmia, the patient should refrain from driving for 6 months. The basis for these time frames stems from data indicating that the risk of recurrent arrhythmia is greatest.
soon after an index event and decreases to < 0.7% per month after the seventh month.\(^6\) It is recommended that aircraft piloting and commercial driving be prohibited in patients receiving an ICD. Similar precautions should be followed for patients involved in the handling of heavy machinery.

**Cost-Effectiveness**

Advancements in ICD technology leading to non-thoracotomy, pectoral implantation have resulted in the vast majority of procedures being undertaken in the EP laboratory. This transition from the operating room has significantly reduced the cost associated with ICD implantation. A recent cost-analysis for ICD procedures compared the expense of procedures in an operating-room setting vs an EP laboratory.\(^5\) Total costs were significantly less in the EP laboratory ($4,541) than in the operating room ($9,431). This lower cost was attributable to lower physician fees, hospital charges, and the shorter length of postprocedural convalescence.

Present data analyzing the cost-effectiveness of the ICD relative to conventional medical therapy are obscured given the rapid technological advances of the ICD. Owens et al\(^6\) provided an economic model estimating that a 30% reduction in mortality by the ICD relative to amiodarone would satisfy the current standards of cost-effectiveness (<$50,000 per life-year gained). This estimate compares favorably to the observed risk reduction of 31% at 3 years in the AVID trial and the 59% risk reduction at 2 years in MADIT.

Prior clinical information regarding the cost-effectiveness of ICDs has been limited by small patient numbers and trial design.\(^6\) Completion of MADIT in 1996 has allowed for an adequate duration of patient follow-up to assess the costs accumulated by each treatment group.\(^6\) The expenses of recurrent hospitalizations, physician visits, medications, laboratory tests, and procedures were analyzed. In view of the significant mortality reduction in MADIT, the resulting cost-effectiveness ratio was $27,000 per life-year gained. In patients who received nonthoracotomy procedures, this ratio was reduced to $23,000 per life-year gained.

Advances in ICD technology will reduce the cost of this therapeutic strategy further. Improved batteries are increasing device longevity, better diagnostic features avoid inpatient or ambulatory Holter monitoring, and dual-chamber pacing capabilities prevent the need for separate pacemaker implantation when required.

**Future Developments**

Future advances in ICD technology are motivated by the demographics of an aging population and by the success of various medical interventions in improving the survival of cardiac patients prone to SCD. A goal of newer-generation ICD devices will be to provide intervention prior to arrhythmia onset, avoiding discomforting cardioversion or defibrillation therapy. ECG or physiologic parameters known to increase ventricular arrhythmia risk, such as long-short R-R intervals, T-wave alternans, heart rate variability, or hemodynamic instability, may be intervened on by novel ICD therapies. Such programmable therapies may include pacing to avoid long-short coupling, intermittent antiarrhythmic drug infusion, or multisite pacing to improve hemodynamics.\(^6\) Further advances in device technology will undoubtedly expand the role of the ICD in the primary and secondary prevention of SCD.

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