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Pacemakers and Implantable Cardioverter-Defibrillators

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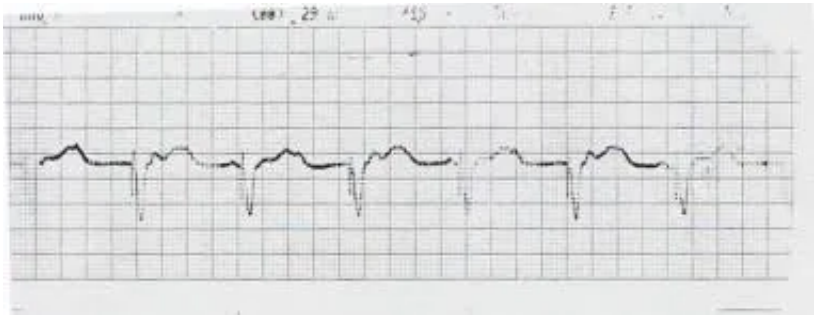
Practice Essentials

An implantable cardioverter-defibrillator (ICD) is a specialized device designed to directly treat many dysrhythmias, and it is specifically designed to address ventricular tachyarrhythmias. ICDs have revolutionized the treatment of patients at risk for sudden cardiac death due to ventricular tachyarrhythmias. A permanent pacemaker is an implanted device that provides electrical stimuli, thereby causing cardiac contraction when intrinsic myocardial electrical activity is inappropriately slow or absent. See the image below. All modern ICDs also function as pacemakers.



100% ventricular paced rhythm.

Indications for ICD placement

Indications for ICD implantation can be divided into two broad categories: secondary prophylaxis against sudden cardiac death and primary prophylaxis. For secondary prophylaxis, ICD placement is indicated as initial therapy in survivors of cardiac arrest due to ventricular fibrillation (VF) or hemodynamically unstable ventricular tachycardia (VT). Published guidelines exclude cases in which there are “transient or reversible causes,”[1] although in the specifics of its application this exclusion is somewhat controversial.

Currently, indications for primary prophylaxis account for most ICD implants. Measurable quantitative benefit is smaller in the primary prophylaxis population than in the secondary prophylaxis population. Class I indications (ie, the benefit greatly outweighs the risk, and the treatment should be administered) are as follows:

- Structural heart disease, sustained VT
- Syncope of undetermined origin, inducible VT or VF at electrophysiologic study (EPS)
- Left ventricular ejection fraction (LVEF) ≤35% due to prior MI, at least 40 days post-MI, NYHA class II or III
- LVEF ≤35%, NYHA class II or III

- LVEF ≤30% due to prior MI, at least 40 days post-MI
- LVEF ≤40% due to prior MI, inducible VT or VF at EPS

Class IIa indications (ie, the benefit outweighs the risk and it is reasonable to administer the treatment) are as follows:

- Unexplained syncope, significant LV dysfunction, nonischemic cardiomyopathy
- Sustained VT, normal or near-normal ventricular function
- Hypertrophic cardiomyopathy with one or more major risk factors
- Arrhythmogenic right ventricular dysplasia/cardiomyopathy (ARVD/C) with one or more risk factors for sudden cardiac death (SCD)
- Long QT syndrome, syncope or VT while receiving beta-blockers
- Nonhospitalized patients awaiting heart transplant
- Brugada syndrome, syncope or VT
- Catecholaminergic polymorphic VT, syncope or VT while receiving beta-blockers
- Cardiac sarcoidosis, giant cell myocarditis, or Chagas disease

Pacemaker indications

The most certain indications for permanent pacing include the following:

- Symptomatic bradycardia due to sinus node dysfunction (sick sinus syndrome)
- Symptomatic chronotropic incompetence
- Symptomatic sinus bradycardia due to necessary drug therapy for other medical conditions, such as atrial fibrillation or coronary artery disease
- Symptomatic AV block
- Symptomatic bradycardia due to complete heart block or second-degree AV block or when ventricular arrhythmias are presumed to arise from AV block
- Symptomatic bradycardia due to complete heart block or second-degree AV block that is due to necessary drug therapy for another medical condition
- Second- or third-degree AV block in asymptomatic awake patients in sinus rhythm resulting in periods of asystole longer than 3.0 seconds or ventricular rates less than 40 beats per minute
- Second -or third-degree AV block in asymptomatic awake patients in atrial fibrillation resulting in pauses of at least 5 seconds
- To facilitate AV node ablation
- Advanced second- or third-degree AV block associated with neuromuscular diseases such as myotonic dystrophy, Kearns-Sayer syndrome, Erb dystrophy (limb-girdle muscular dystrophy), and peroneal muscular atrophy
- Second- or third-degree AV block during exercise in the absence of myocardial ischemia
- Recurrent syncope caused by spontaneously occurring carotid sinus stimulation and demonstrable ventricular pauses of greater than 3 seconds with purposeful carotid sinus pressure

- Cardiac resynchronization therapy with biventricular pacing

In the setting of chronic bifascicular block, permanent pacing is indicated for:

- Advanced second-degree AV block or intermittent third-degree AV block
- Type II second-degree AV block
- Alternating bundle branch block

Temporary emergency pacing is indicated for therapy of significant and hemodynamically unstable bradyarrhythmias and for prevention of bradycardia-dependent malignant arrhythmias.

Magnet Inhibition

Features of magnet inhibition are as follows:

- In most devices, placing a magnet over a permanent pacemaker temporarily "reprograms" the pacer into an asynchronous pacing mode; it does not turn the pacemaker off
- If the device company parameters are known, application of a magnet can determine whether the pacer's battery needs to be replaced
- Although many different branded pacemaker/ ICD magnets are available, in general, any pacemaker/ICD magnet can be used to inhibit the device
- Magnet use inhibits further ICD discharge; it does not, however, inhibit pacing
- In some devices, "magnet function" can be disabled

Indications for ICD deactivation are as follows:

- End-of-life care (after a discussion with the patient and family)
- Inappropriate shocks
- During resuscitation
- During surgical procedures that involve electrocautery

ICD complications and malfunctions

Acute surgical complications include the following:

- Pain
- Bleeding
- Pneumothorax
- Hemothorax
- Cardiac perforation with or without pericardial effusion and tamponade (sometimes requiring urgent drainage)
- Pulseless electrical activity following intraoperative defibrillation threshold testing

Subacute ICD complications include the following:

- Pain

- Infection
- Pocket hematoma
- Wound dehiscence
- Lead dislodgment
- Deep venous thrombosis
- Upper extremity edema
- Degradation of lead function

Chronic complications include the following:

- Device-related pain
- Lead fracture
- Inappropriate shocks
- Erosion of device through skin
- Immunologic rejection – Rare

Pacemaker complications and malfunctions

Pacemaker complications include the following:

- Pneumothorax
- Pericarditis
- Infection
- Skin erosion
- Hematoma
- Lead dislodgment
- Venous thrombosis

Major pacemaker malfunctions include the following:

- Failure to output
- Failure to capture
- Failure to sense
- Pacemaker-mediated tachycardia (PMT)
- Pacemaker syndrome
- Twiddle syndrome
- Cardiac monitor pseudomalfunction
- Pacemaker pseudomalfunction

Inpatient Care

Reasons for admission may include the following:

- Device investigation: To determine if there is an imminent battery failure (multiple shocks will deplete battery life)
- Addition of antiarrhythmic medications
- Treatment of MI (which may be linked to the initial discharge)
- Treatment of patient discomfort
- Provision of psychological support: Up to 35% of people develop anxiety disorder following ICD placement, although disabling problems necessitating admission are fairly uncommon[2]

Overview

An implantable cardioverter-defibrillator (ICD) is a specialized implantable electronic device designed to directly treat a cardiac tachyarrhythmia, whereas a permanent pacemaker is an implanted device that provides electrical stimuli, thereby causing cardiac contraction when intrinsic myocardial electrical activity is inappropriately slow or absent. A pacemaker senses intrinsic cardiac electric potentials, and, if these are too infrequent or absent, transmits impulses to the heart to stimulate myocardial contraction.

All modern ICDs are equipped with a demand pacing system and serve the dual functions of emergency defibrillation and backup pacing. If a patient has a ventricular ICD and the device senses a ventricular rate that exceeds the programmed threshold, the device may be programmed to deliver antitachycardia pacing therapy (ATP) or defibrillation. With antitachycardia pacing, the device delivers a preset number of rapid pulses in succession in an attempt to terminate the ventricular tachycardia. If antitachycardia pacing therapy is ineffective within a prespecified number of trials or a prespecified time period, or if the ventricular rate exceeds a preset rate, the device delivers a high-energy electric shock to reset the heart’s electrical activity.

Evolution of the ICD

The implantable cardioverter-defibrillator (ICD) has revolutionized the treatment of patients at risk for sudden cardiac death due to ventricular tachyarrhythmias. Initially introduced in humans in 1980[3] and approved by the US Food and Drug Administration (FDA) in 1985, the ICD has evolved from a treatment of last resort to a first-line treatment and prophylactic therapy for patients at risk for ventricular tachycardia (VT) or ventricular fibrillation (VF).[1] Michel Mirowski conceived of and developed the ICD almost single-handedly. Prompted by the sudden death of a colleague, Mirowski conceived of an automatic, fully implantable defibrillator. Initially, lead systems were epicardial, requiring a thoracotomy for implantation, and pulse generators were large and bulky, requiring abdominal implantation.

Remarkable technologic advances have made ICDs easier and safer to implant and better accepted by patients and physicians. The development of transvenous lead systems, more effective biphasic defibrillation waveforms, and "active can" technology allows implantation in nearly all patients without the need for thoracotomy.[4]

Significant miniaturization of the capacitors and other components has reduced the size of the pulse generator tremendously, permitting subcutaneous pectoral implantation in most patients.[5, 6] A new generation of subcutaneously implanted devices has obviated the need for transvenous leads in a select group of patients. On September 28, 2012, the FDA approved the first subcutaneous ICD (SCD) for ventricular tachyarrhythmias which allows the lead to be placed under the skin rather than through a vein into the heart.[7] These devices are again larger, as there is a requirement for increased current delivery and, in their present form, have very limited pacing capabilities.

In addition to being considerably smaller than early generations of ICDs, current ICDs have markedly progressed in their therapeutic and diagnostic functions. Early devices were simple “shock boxes,” offering only high-energy shocks when the patient's heart rate exceeded a cut-off point. Diagnostic information was limited to the number of shocks delivered. Current devices offer tiered therapy with programmable antitachycardia pacing schemes, as well as low-energy and high-energy shocks in multiple tachycardia zones.

Dual-chamber, rate-responsive bradycardia pacing is now available in all ICDs, and sophisticated discrimination algorithms minimize shocks for atrial fibrillation, sinus tachycardia, and other non–life-threatening supraventricular tachyarrhythmias. Diagnostic functions, including stored electrograms, allow for verification of shock appropriateness. Device battery longevity has also increased; early devices lasted 2 years or less, while current devices are expected to last 8 years or longer.

ICD Clinical Trials

Early data regarding the implantable cardioverter-defibrillator (ICD) was drawn primarily from uncontrolled series of patients for whom antiarrhythmic drug therapy for ventricular tachycardia (VT) or ventricular fibrillation (VF) had failed. Even in these refractory patients, initial series suggested a markedly reduced risk of sudden, presumed arrhythmic, death. Subsequent randomized, controlled trials also focused on secondary prevention of sustained VT, VF, and sudden cardiac death.[8] In the early to-mid 1990s, three clinical trials were conducted in patients who had survived life-threatening ventricular tachyarrhythmias.

The Antiarrhythmics Versus Implantable Defibrillators (AVID) trial, conducted in the United States, enrolled patients with prior cardiac arrest or hemodynamically significant sustained VT and randomized patients to either ICD implantation or antiarrhythmic drug therapy, including primarily amiodarone and, in a few cases, sotalol.[9] The Canadian Implantable Defibrillator Study (CIDS) trial in Canada had a similar structure.[10, 11] The Cardiac Arrest Study Hamburg (CASH) trial in Hamburg enrolled cardiac arrest survivors and randomized them to amiodarone, metoprolol, propafenone, or ICD implantation.[12]

The AVID trial, although sponsored by the National Institutes of Health (NIH), was extremely controversial in its conception, because many electrophysiologists maintained that a randomized trial was not necessary to prove the effectiveness of the ICD. This trial was terminated prematurely because of improved survival rates in the ICD-treated patients. The CIDS trial yielded similar results. In the CASH trial, the propafenone arm of the study was terminated prematurely due to an increased mortality rate.[13] Ultimately, in the CASH trial, ICD therapy proved superior when compared with either amiodarone or metoprolol therapy.

These trials firmly established the ICD as preferred therapy in patients who have survived cardiac arrest or hemodynamically significant, sustained VT.

Subgroup analyses of results from the AVID trial, CIDS trial, and primary prevention Multicenter Automatic Defibrillator Implantation Trial (MADIT)[14] suggested that the survival benefit of the ICD is realized primarily by a well-defined subgroup of patients; specifically, those with greater impairment of left ventricular systolic function, as measured by left ventricular ejection fraction (LVEF), without end-stage (New York Heart Association [NYHA] class IV) heart failure. In these three trials, the ICD had less apparent benefit in patients with better-preserved left ventricular systolic function.

Several important trials have subsequently been performed examining the role of ICDs as primary therapy for patients who are at risk for but who have not yet manifested sustained ventricular arrhythmias. These trials include the MADIT (Multicenter Automatic Defibrillator Implantation Trial), MUSTT (Multicenter Unsustained Tachycardia Trial), MADIT II, SCD-HeFT (Sudden Cardiac Death in Heart Failure Trial), and the COMPANION (Comparison of Medical Therapy, Pacing, and Defibrillation in Chronic Heart Failure) trial.

MADIT study

Similar to the AVID trial, MADIT was terminated prematurely because of a significant survival benefit seen in patients treated with ICDs. MADIT in 1996 had enrolled patients with ischemic cardiomyopathy (LVEF ≤35%) and asymptomatic, nonsustained VT who had inducible sustained VT or VF not suppressible with procainamide infusion during electrophysiology study (EPS). Enrolled patients were randomized to either ICD implantation or to therapy considered appropriate by the treating physician. Antiarrhythmic drug therapy was administered in both arms as considered appropriate

by the treating physician.[14]

MUSTT

MUSTT, reported in 1999, also showed a survival benefit to ICD therapy. The trial had similar inclusion criteria to MADIT (prior infarct, LVEF ≤40%, nonsustained VT inducible at EPS) and randomized patients to EPS-guided therapy versus no specific antiarrhythmic therapy.

Early in the trial, EPS-guided therapy consisted of antiarrhythmic drug therapy guided by EPS testing, with ICD implantation reserved for patients with ventricular arrhythmias refractory to antiarrhythmic drugs. Later in the trial, ICDs were used earlier in patients who were randomized to the EPS-guided therapy arm of the trial. MUSTT showed a survival benefit in the EPS-guided group. The survival benefit was attributable to the ICD. Patients who were randomized to EPS-guided therapy and treated with antiarrhythmic drugs fared no better or worse than patients assigned to the control arm of the trial.[15]

In MUSTT, a registry was maintained of patients who met the clinical criteria for the study but were noninducible in the electrophysiology laboratory. During follow-up, the survival rate in this group was better than in the inducible patients assigned to the control group but not as good as in inducible patients who received ICDs. Although MUSTT was not designed to determine the optimal treatment in noninducible patients, many have concluded that, in the population studied, EPS testing may be used to stratify high-risk and moderate-risk patients rather than high-risk and low-risk patients.[16, 17]

MADIT II

The Multicenter Automatic Defibrillator Implantation Trial II (MADIT II) in 2002 markedly expanded the potential pool of ICD recipients. MADIT II randomized patients with prior myocardial infarction and LVEF at or below 30% to ICD therapy or a control group. Nonsustained VT or inducible VT at EPS was not required. Patients who received an ICD had a 31% reduction in mortality rate. An important aspect of MADIT II was that subjects in both arms of the trial were well managed medically with a high rate of beta-blocker, angiotensin-converting enzyme (ACE) inhibitor, and cholesterol-lowering medication usage.[18]

Information regarding cost implications of ICDs continues to emerge. The MADIT II study showed that prophylactic implantation of a defibrillator reduced the rate of mortality in patients with a previous myocardial infarction and low LVEF. The cost analysis phase of the study showed that during the 3.5-year period of the study, the average survival gain for the defibrillator arm was 0.167 years (2mo), the additional costs were $39,200, and the incremental cost-effectiveness ratio (iCER) was $235,000 per year-of-life saved. In 3 alternative projections to 12 years, this ratio ranged from $78,600 to $114,000. Estimated cost per life-year saved is relatively high at 3.5 years, but projected costs are substantially lower over the course of longer time horizons.[19]

COMPANION trial

In the COMPANION trial, pacemakers and, to an even greater extent, ICDs, were found to reduce the risk of death in patients with advanced heart failure, even when there was no indication for pacemaker or ICD treatment.

In the COMPANION trial, patients with advanced heart failure, NYHA functional class III or IV, an LVEF of 35% or less, and intraventricular conduction delay with QRS duration of over 120 milliseconds, but with no indication for pacemaker or ICD implant, were randomized to optimal medical therapy alone or in combination with cardiac resynchronization therapy with either a biventricular pacemaker or biventricular pacemaker-defibrillator.

Risk of hospitalization or death from heart failure was reduced by 34% in the pacemaker group and by 40% in the defibrillator group. Risk of death from any cause was reduced by 24% in the pacemaker group and by 36% in the defibrillator group.[20]

SCD-HeFT

In SCD-HeFT, a primary prevention trial reported in 2005 in which subjects with an LVEF of 35% or less and symptoms in NYHA functional class II or III were randomly assigned to 1 of 3 treatment groups—conventional heart failure therapy plus placebo, conventional heart failure therapy plus amiodarone, or conventional heart failure therapy plus ICD implant—ICD therapy, as compared with placebo, was associated with a 23% reduction in the risk of death from any cause and an

absolute 7% decrease in mortality over 5 years.

No difference in mortality benefit was shown between subjects with ischemic cardiomyopathy (70% of enrollees) and those with nonischemic cardiomyopathy. ICD therapy benefited only NYHA functional class II subjects.[21]

Other studies

A meta-analysis of 5 clinical trials that included 4317 patients with NYHA functional class I/II heart failure performed by Adabag et al suggested that asymptomatic patients with NYHA functional class I/II heart failure may benefit from cardiac resynchronization therapy.[22] This therapy has been shown to decrease all-cause mortality, reduce heart failure hospitalizations, and improve LVEF in these patients. The authors did caution that risks versus benefits do need to be taken into consideration for this group of patients.

Levy et al found that in patients with moderately symptomatic heart failure with an ejection fraction of 35% or less, primary prevention with an ICD provides no benefit in some cases but substantial benefit in others, and that ICD benefit can be predicted. Analysis of data from the placebo arm of SCD-HeFT showed that patients could be classified into 5 groups on the basis of predicted 4-year mortality. In the treatment arm, ICD implantation decreased relative risk of sudden cardiac death by 88% in patients with the lowest baseline mortality risk, versus 24% in the highest-risk group. ICD treatment decreased relative risk of total mortality by 54% in the lowest-risk group but provided no benefit (2%) in the highest-risk group.[23]

Women enrolled in primary prevention ICD trials have had the same mortality compared with men, while experiencing significantly fewer appropriate ICD interventions, thus suggesting a smaller impact of sudden cardiac death on overall mortality in women with dilated cardiomyopathy.[24]

Trials Showing No Benefit From ICD Therapy

At least four notable, published implantable cardioverter-defibrillator (ICD) trials have failed to demonstrate a significant survival benefit to ICD therapy over optimal medical therapy. Two of these trials examined patients with ischemic cardiomyopathy and two examined patients with nonischemic cardiomyopathy.

DEFINITE

The Defibrillators in Non-Ischemic Cardiomyopathy Treatment Evaluation Trial (DEFINITE) enrolled subjects with nonischemic cardiomyopathy and showed a trend toward mortality benefit in the ICD arm, with mortality at 2 years being 14.1% in the medical therapy arm and 7.9% in the ICD arm.[25]

CAT

The Cardiomyopathy Trial (CAT), in which 104 subjects with recent (< 9 mo) onset of nonischemic cardiomyopathy were randomized to ICD therapy or control group, was terminated early, at 1 year, due to lower than expected mortality in the control group (3.7% vs the expected 30%). At a mean follow-up of 5.5 years, no significant difference was shown in cumulative survival.[26]

CABG-Patch trial

In the Coronary Artery Bypass Graft (CABG)-Patch trial, ICD implantation improved the sudden cardiac death mortality rate but not the total mortality rate. In the study, subjects undergoing CABG who had decreased left ventricular function (ejection fraction < 35%) and an abnormal signal-averaged electrocardiogram (SAECG) were randomized to epicardial ICD implantation at the time of CABG or to a control group.[27] The lack of effectiveness of IDCs on the mortality rate apparently resulted from the poor predictive value of a preoperative SAECG in identifying patients at risk for arrhythmic death or the salutatory effects of coronary revascularization in reducing the risk of arrhythmic death.

DINAMIT

In the Defibrillator in Acute Myocardial Infarction Trial (DINAMIT)—which enrolled subjects within 40 days of an acute

myocardial infarction and randomized them to optimal medical therapy with or without a defibrillator—no difference was shown in mortality at a mean follow-up of 2.5 years. The CABG-Patch and DINAMIT trials each involved a possible confounder of revascularization.[28]

AMIOVIRT

A small trial that directly compared ICD therapy with medical therapy of amiodarone found no mortality difference between the 2 treatments at 3 years. The Amiodarone Versus Implantable Cardioverter-Defibrillator Randomized Trial (AMIOVIRT) studied 103 subjects with nonischemic cardiomyopathy and was stopped early when a prespecified rule for futility was reached.[29] (The SCD-HeFT trial provided only an indirect comparison between amiodarone therapy and ICD therapy because the trial was designed only to compare each of these therapies individually with optimal medical therapy.)[21]

CRT-D treatment study

A study by Barsheshet et al suggested that patients with ischemic cardiomyopathy have a higher risk of heart failure or death that is directly related to cardiac resynchronization therapy with defibrillator (CRT-D) and the elapsed time from myocardial infarction.[30]

ICD treatment post-myocardial infarction

In the case of post-myocardial infarction (MI) patients, no mortality benefit was observed from placing ICDs in patients with reduced ejection fraction until after 40 days post-MI and patient reassessment. This is likely due to the fact that death in the first 40 days post-MI may be attributed to causes other than arrhythmia.[31]

Age-related studies

Some data suggest that not all age groups benefit equally from the protective effect of ICDs. One pooled analysis of primary prevention trials found that the elderly do not derive a clinically significant benefit from ICDs.[32] Moreover, there was an overall 17% complication rate associated with ICD treatments. In this analysis, elderly was defined as older than 60 for some studies and older than 65 for others.

A pooled analysis of secondary prevention ICD trials also found no reduction in all-cause and arrhythmic mortality in elderly patients.[33] However, current recommendations do not exclude ICD implantation on the basis of age.

ICD Indications

Indications for implantable cardioverter-defibrillator (ICD) implant can be divided into two broad categories: secondary prophylaxis against sudden cardiac death and primary prophylaxis. Multiple studies have shown the ICD to be superior to antiarrhythmic drug therapy in patients with a history of life-threatening ventricular tachycardia (VT) and ventricular fibrillation (VF). Therefore, the indications for secondary prophylaxis are well supported by clinical evidence gained from randomized clinical trials.[1] Currently, however, indications for primary prophylaxis account for most of ICD implants, even though the evidence for such implants is often less well established. Measurable quantitative benefit is smaller in the primary prophylaxis population than in the secondary prophylaxis population.

Wilcox et al investigated the clinical effectiveness of cardiac resynchronization (CRT) and ICD therapy as a function of sex from data in 8936 outpatients with heart failure and reduced ejection fraction (≤35%) and found a substantially reduced 24-month mortality in eligible men and women with heart failure and reduced ejection fraction.[34] The data was obtained from the Registry to Improve the Use of Evidence-Based Heart Failure Therapies in the Outpatient Setting (IMPROVE HF) for vital status (alive/dead) at 24 months. The clinical benefit associated with ICD/CRT-D therapy was similar in both sexes.[34]

Using data from the Get With The Guidelines-Heart Failure Registry and the National Cardiovascular Data Registry's ICD Registry, Pokorney et al compared the relationship of primary prevention ICDs with mortality in 852 Medicare, racial/ethnic minority patients (nonwhite race or Hispanic ethnicity) and 2070 white non-Hispanic patients.[35] Minority ICD patients had a lower adjusted 3-year mortality rate (44.9%) than their non-ICD counterparts (54.3%). Similarly, white non-Hispanic ICD patients had a lower adjusted 3-year mortality rate (47.8%) than their non-ICD counterparts (57.3%). The investigators found

no significant interaction between race/ethnicity and lower mortality risk with ICD; they indicated that these results support the use of a similar approach in selecting ICD patients, irrespective of race or ethnicity.[35]

Investigators have looked at ongoing indications for ICD therapy at the time of elective device replacement. In one prospective cohort study, 21% of patients received appropriate ICD therapy within 3 years following device replacement, even if they had never received appropriate therapy from their originally implanted device. For patients who had received appropriate therapy from their first device, 48% received appropriate therapy over the same 3-year period.[36]

Secondary prophylaxis

An ICD is recommended as initial therapy in survivors of cardiac arrest due to VF or hemodynamically unstable VT. Published guidelines exclude cases in which there are “completely reversible causes.”[1]

The exclusion for completely reversible causes is somewhat controversial. As an example, an acute myocardial infarction (MI) predisposes to polymorphic VT, and the culprit lesion may be reversed with intracoronary stenting. However, we know that any patient who presents with an MI is at increased risk of recurrent MI, which may again precipitate an unstable ventricular arrhythmia. One school of thought suggests that such patients should undergo ICD implant, even though the cause of cardiac arrest is completely reversible, because the risk of recurrence is increased. However, current standard of care is that cardiac arrest during the initial hours of a define acute MI is not considered and indication for ICD implantation.

In another example, consider cardiac arrest secondary to transient prolongation of the QT interval, perhaps secondary to drug therapy. QT interval prolongation increases the risk of torsades de pointes, a potentially life-threatening arrhythmia. Withdrawal of the offending agent may normalize the QT interval, thereby reversing the cause of cardiac arrest. However, such a patient remains at risk of recurrent QT prolongation and subsequent cardiac arrest, perhaps from an electrolyte disturbance or as a result of ingestion of a different QT-prolonging agent.

Primary prophylaxis

Indications for an ICD implant as primary prophylaxis against sudden cardiac death are listed in the Table 1, below. The indications are listed as class I or class IIa, as classified by the 2008 American College of Cardiology and American Heart Association (ACC/AHA) guidelines. Class I means that the treatment is useful, that its benefit greatly outweighs the risk, and that it should be administered.

Class IIa means that the benefit outweighs the risk and it is reasonable to administer the treatment. Class IIb means that the benefit probably outweighs the risk and that the treatment may be considered. Class III means that the risk outweighs the benefit, and the treatment should not be performed. Only Class I and Class IIa indications are included in the table. For a complete list, the reader is referred to the 2008 ACC/AHA guidelines.[1]

The greatest predictors of risk for sudden cardiac death include left ventricular systolic function and heart failure symptoms. The vast majority of investigational studies have quantified left ventricular systolic function using the measure of left ventricular ejection fraction (LVEF). The most widely used form of heart failure symptom classification is the New York Heart Association (NYHA) functional class classification system, which classifies mild to no symptoms as Class I, and the most severe symptoms as Class IV.

Table 1. Indications for ICD Implant (Open Table in a new window)

Indication	Classification	Supporting Studies
Structural heart disease, sustained VT	Class I	AVID, CASH, CIDS
Syncope of undetermined origin, inducible VT or VF at	Class I	CIDS

EPS		
LVEF ≤35% due to prior MI, at least 40 days post-MI, NYHA Class II or III	Class I	SCD-HeFT
LVEF ≤35%, NYHA Class II or III	Class I	SCD-HeFT
LVEF ≤30% due to prior MI, at least 40 days post-MI	Class I	MADIT II
LVEF≤40% due to prior MI, inducible VT or VF at EPS	Class I	MADIT, MUSTT
Unexplained syncope, significant LV dysfunction, nonischemic CM	Class IIa	Expert opinion
Sustained VT, normal or near-normal ventricular function	Class IIa	Expert opinion
Hypertrophic cardiomyopathy (CM) with 1 or more major risk factors	Class IIa	Expert opinion
Arrhythmogenic right ventricular dysplasia/cardiomyopathy (ARVD/C) with 1 or more risk factors for sudden cardiac death (SCD)	Class IIa	Expert opinion
Long QT syndrome, syncope or VT while receiving beta blockers	Class IIa	Zareba et al,[37] Viskin et al,[38] Goel et al,[39] Monnig et al,[40] Goldenberg et al,[41] Hobbs et al[42]
Nonhospitalized patients awaiting heart transplant	Class IIa	Expert opinion
Brugada syndrome, syncope	Class IIa	Expert opinion

Brugada syndrome, VT	Class IIa	Expert opinion
Catecholaminergic polymorphic VT, syncope or VT while receiving beta blockers	Class IIa	Expert opinion
Cardiac sarcoidosis, giant cell myocarditis, or Chagas disease	Class IIa	Expert opinion

CM = cardiomyopathy; EPS = electrophysiologic studies; ICD = implantable cardioverter-defibrillator; LVEF = left ventricular ejection fraction; MI = myocardial infarction; NYHA = New York Heart Association; VF = ventricular fibrillation; VT = ventricular tachycardia.

Pacemaker Indications

The most certain indications for permanent pacing include the following:

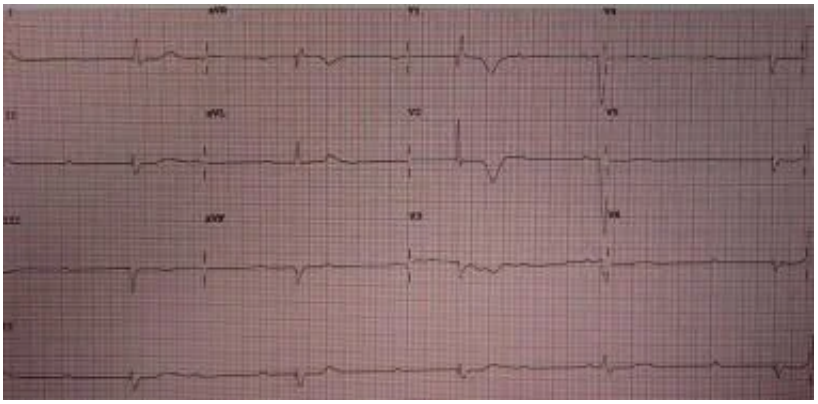
- Symptomatic bradycardia due to sinus node dysfunction (sick sinus syndrome)
- Symptomatic chronotropic incompetence
- Symptomatic sinus bradycardia due to necessary drug therapy for other medical conditions, such as atrial fibrillation or coronary artery disease
- Symptomatic AV block
- Symptomatic bradycardia due to complete heart block or second-degree AV block or when ventricular arrhythmias are presumed to arise from AV block
- Symptomatic bradycardia due to complete heart block or second-degree AV block that is due to necessary drug therapy for another medical condition
- Second- or third-degree AV block in asymptomatic awake patients in sinus rhythm resulting in periods of asystole longer than 3.0 seconds or ventricular rates less than 40 beats per minute
- Second -or third-degree AV block in asymptomatic awake patients in atrial fibrillation resulting in pauses of at least 5 seconds
- To facilitate AV node ablation
- Advanced second- or third-degree AV block associated with neuromuscular diseases such as myotonic dystrophy, Kearns-Sayer syndrome, Erb dystrophy (limb-girdle muscular dystrophy), and peroneal muscular atrophy
- Second- or third-degree AV block during exercise in the absence of myocardial ischemia
- Recurrent syncope caused by spontaneously occurring carotid sinus stimulation and demonstrable ventricular pauses of greater than 3 seconds with purposeful carotid sinus pressure

- Cardiac resynchronization therapy with biventricular pacing

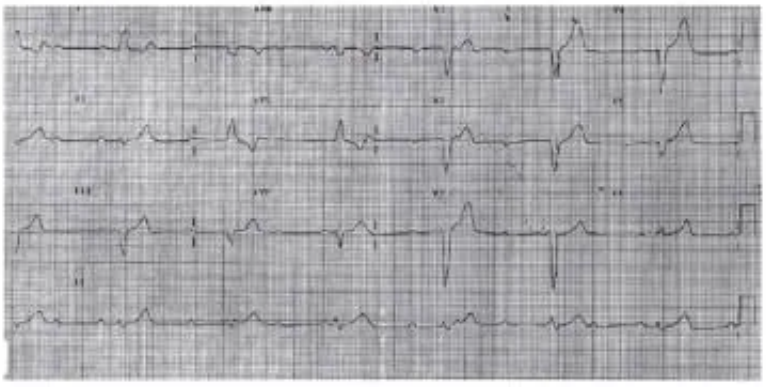
In the setting of chronic bifascicular block, permanent pacing is indicated for:

- Advanced second-degree AV block or intermittent third-degree AV block
- Type II second-degree AV block
- Alternating bundle branch block

Temporary emergency pacing is indicated for therapy of significant and hemodynamically unstable bradyarhythmias and for prevention of bradycardia-dependent malignant arrhythmias. Examples include refractory symptomatic sinus node dysfunction, complete heart block (see the image below), alternating bundle-branch block, new bi-fascicular block, and bradycardia-dependent ventricular tachycardia.



Third-degree heart block. Image courtesy of James Heilman, MD, via Wikimedia Commons.



Electrocardiogram showing complete heart block. The ventricular rate is 25 per minute, the QRS complex is very wide, and there is atrioventricular dissociation. Image courtesy of Case Reports in Cardiology ((Jafar NS, et al. Rapidly Progressive Atrioventricular Block in a Patient with Sarcoidosis. Case Rep Cardiol. 21 Aug 2014; Article ID 372936).

Examples of indications for prophylactic temporary pacing include for the purpose of transcatheter aortic valve replacement (TAVR), use of medications that may cause or exacerbate hemodynamically significant bradycardia, prophylaxis during the perioperative period surrounding cardiac valvular surgery, Lyme disease or other infections (Chagas disease) that cause interval changes.

Device Insertion

Pacing systems consist of a pulse generator and pacing leads. With permanent systems, endocardial leads are inserted transvenously and advanced to the right ventricle and/or atrium, where they are affixed to the myocardial tissue, typically with a helical screw tip, most of which are extendable and retractable. The pulse generator is placed subcutaneously or submuscularly in the chest wall.

Pulse generators contain a battery, as well as sensing, timing, and output circuits. The battery (most commonly lithium-iodide) typically has a lifespan of around 10 years, although technologic advances are constantly improving performance. Pulse generators can be set to fixed-rate (asynchronous) or demand (synchronous) modes. In the asynchronous mode, impulses are produced at a set rate that is independent of intrinsic cardiac activity. This mode carries a small, but inherent, danger of initiating lethal arrhythmias should the impulse coincide with the vulnerable period of the T-wave. In the synchronous mode, the sensing circuit searches for an intrinsic depolarization potential. If this is absent, a pacing response is generated. This mode closely mimics intrinsic myocardial electrical activity.

At the time of pacemaker implantation, and at regular intervals afterward, signal amplitude and width are set high enough to reliably achieve myocardial capture, yet low enough to maximize battery life.

Temporary systems use an external pulse generator with leads placed either transcutaneously or transvenously. Transcutaneous leads are the easiest and most convenient to use for rapid application of temporary pacing, and they are the method of choice during emergency department (ED) resuscitation. Transcutaneous pacing may be uncomfortable, and patients may require mild sedation (eg, benzodiazepine). Transcutaneous pacing may also fail to reliably induce cardiac contraction. Often, transvenous pacing must be established to reliably stabilize cardiac rhythm. Once the central venous access is gained, transvenous leads provide the most reliable and comfortable pacing mechanism and are a good transition to permanent systems.

Under ideal circumstances, most temporary pacing catheters are inserted with fluoroscopic guidance in a catheterization laboratory. In more emergent situations, flexible, balloon-tipped catheters may be positioned using only electrocardiographic monitoring.

Many patients who undergo implantable cardioverter-defibrillator or pacemaker implantation are anticoagulated with warfarin. A strategy of implanting devices during uninterrupted warfarin therapy has a lower bleeding risk than a strategy of temporarily discontinuing warfarin and bridging with heparin.[43] We await similar data for newer anticoagulants such as dabigatran, rivaroxaban, apixaban, and edoxaban.

Pacing Codes

The Heart Rhythm Society and the British Pacing and Electrophysiology Group (BPEG) developed a code to describe various pacing modes.[44] (See Table 2, below.)

Table 2. Pacemaker Code Used to Describe Various Pacing Modes (Open Table in a new window)

1st Position	2nd Position	3rd Position	4th Position	5th Position
Chamber Paced	Chamber Sensed	Response to Sensing	Rate Modulation	Multisite Pacing
A	A	T	O	O

V	V	I	R	A
D	D	D		V
	O	O		D

A = atrium, D = dual (both chambers), I = inhibited, O = none, R = rate adaptive, T = triggered, V = ventricle.

Pacing code explanation:

A typical pacing code consists of 3-5 letters. The first letter indicates the chamber(s) paced, as follows:

- A - Atrial pacing
- V - Ventricular pacing
- D - Dual-chamber (atrial and ventricular) pacing

The second letter indicates the chamber in which electrical activity is sensed, as follows:

- A, V, or D
- O is used when pacemaker discharge is not dependent on sensing electrical activity.

The third letter refers to the response to a sensed electric signal, as follows:

- T - Triggering of pacing function
- I - Inhibition of pacing function
- D - Dual response (ie, any spontaneous atrial and ventricular activity will inhibit atrial and ventricular pacing, and lone atrial activity will trigger a paced ventricular response)
- O - No response to an underlying electric signal (usually related to the absence of associated sensing function)

The fourth letter represents rate modulation, as follows:

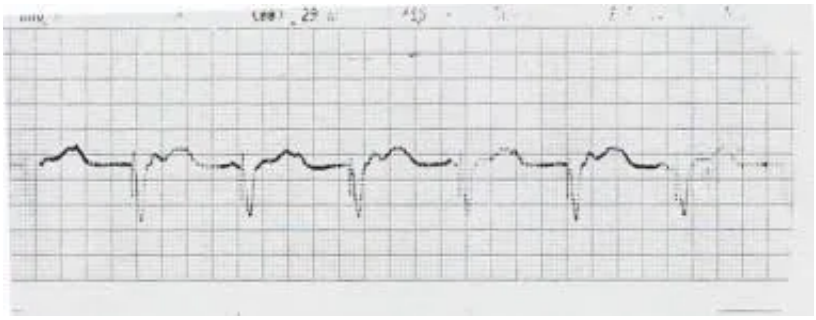
- R - Rate-response ("physiologic") pacing
- O - No programmability or rate modulation

The fifth letter represents multisite pacing, as follows:

- A - Atrial
- V - Ventricular
- D - Dual (pacing + shock)

Although the first 3 letters of the pacing code are used most commonly, a 5-position code is currently in use.

Modern pacemakers have multiple functions. The simplest settings are VVI and AAI. The VVI mode senses and paces the ventricle and is inhibited by a sensed ventricular event. Alternatively, the AAI mode senses and paces in the atrium, and each sensed event triggers the generator to fire within the P wave. (See the image below.)



100% ventricular paced rhythm.

The most common setting, the DDD mode, denotes that both chambers are capable of being sensed and paced. This requires two functioning leads, one in the atrium and the other in the ventricle. On the electrocardiogram (ECG), if both atrium and ventricle are being paced, there will be a pacing artifact before the P wave and preceding the QRS. The first pacing artifact indicates the atrial depolarization, and the second indicates the initiation of the QRS complex. Given that one of the leads is in the right ventricle, a left bundle-branch pattern may be evident on ECG.

Note that a 2-wired system does not necessarily need to be in DDD mode, since the atrial or ventricular leads can be programmed off. Additionally, single tripolar lead systems are available that can sense atrial impulses and either sense or pace the ventricle. Thus, this system provides for atrial tracking without the capability of atrial pacing and can be used in patients with AV block and normal sinus node function.

Pacemaker programming can be performed noninvasively by an electrophysiology technician or cardiologist. Because of the myriad of pacemaker types, patients should carry a card with them providing information about their particular model. Most pacemaker generators have an x-ray code that can be seen on a chest radiograph; however, the chest radiography may need to be zoomed onto the pacemaker generator for better resolution. The markings, along with the shape of the generator, may assist with deciphering the manufacturer of the generator and pacemaker battery.

For further information or locations of technicians for pacemaker devices, the device company can be contacted at the following 24-hour help-line telephone numbers below[45] :

- Boston Scientific (formerly Guidant) - 800-CARDIAC (800-227-3422)
- Medtronic - 800-633-8766
- Abbott (formerly St Jude Medical) - 800-PACE-ICD (800-722-3433)
- Biotronik - 800-547-0394
- LivaNova (formerly Sorin) - 877-663-7674

Magnet Inhibition

In most devices, placing a magnet over a permanent pacemaker temporarily "reprograms" the pacer into asynchronous mode; it does not turn the pacemaker off. Each pacemaker type has a unique asynchronous rate for beginning of life (BOL), elective replacement indicator (ERI), and end of life (EOL). Therefore, if the device company parameters are known, application of a magnet can determine if the pacer's battery needs to be replaced. Further interrogation or manipulating of the device should be performed by an individual skilled in the technique.

Although many different branded pacemaker/implantable cardioverter-defibrillator (ICD) magnets are available, emergency physicians should be aware that, in general, any pacemaker/ICD magnet can be used to inhibit delivery of shock therapy

from the device. When a magnet is applied to an ICD, pacing therapy is not inhibited.

The majority of devices have a magnet response; however, some devices can be programmed to not respond to magnet application and thus will need a device programmer to change the parameters.

In some devices, application of a magnet produces a soft beep for each QRS complex. If the magnet is left on for approximately 30 seconds, the ICD is disabled and a continuous tone is generated. To reactivate the device, the magnet must be lifted off the area of the generator and then replaced. After 30 seconds, the beep returns for every QRS complex.

Indications for ICD deactivation are as follows:

- End-of-life care (after a discussion with the patient and family)
- Inappropriate shocks
- During resuscitation
- During surgical procedures that employ electrocautery

Adjunctive Care in ICD Therapy

Although implantable cardioverter-defibrillators (ICDs) are extremely effective in terminating life-threatening arrhythmias, many patients require adjunctive therapy to reduce the frequency of arrhythmic events that require therapy. This generally consists of pharmacologic therapy, and, particularly in cases of failure of drug therapy, radiofrequency catheter ablation.

Inappropriate shocks may be delivered for atrial fibrillation, sinus tachycardia, and other types of supraventricular tachycardia, prompting ICD reprogramming or adjunctive therapy.

See The Heart Rhythm Society's Expert Consensus Statement on the Management of Cardiovascular Implantable Electronic Devices (CIEDs) in Patients Nearing End of Life or Requesting Withdrawal of Therapy.[46]

ICD Complications and Malfunctions

Several complications of implantable cardioverter-defibrillators (ICD) implant have been described, some of which are currently tracked in a national database of ICD implants. Acute surgical complications include the following:

- Pain
- Bleeding
- Pneumothorax
- Hemothorax
- Cardiac perforation with or without pericardial effusion and tamponade (sometimes requiring urgent drainage)
- Pulseless electrical activity following intraoperative defibrillation threshold testing

An analysis of more than 350,000 ICD implantations included in the National Cardiovascular Data Registry–ICD Registry revealed 3.1% of patients experienced inhospital adverse events, 1.2% experienced major adverse events, and 0.4% died. Adverse events were lower (1.9%) with single-chamber ICD implants than with dual-chamber ICD implants (2.9%) or with biventricular ICD implants (4.1%). Specific adverse event rates included lead dislodgement (1%), hematoma (0.9%), pneumothorax (0.4%), and cardiac arrest (0.3%).

Physician level of training and level of specialty certification have been shown to affect the risk of adverse events associated with ICD implant. An ICD Registry analysis found that physicians who implant more ICDs have lower rates of procedural

complications and in-hospital mortality[47] Implant volume may partially explain the difference in adverse events among physicians with different specialty certifications. However, no inverse relationship was found between procedure volume and adverse event rate observed within the board certified category.

Subacute and chronic complications

Subacute ICD complications include the following:

- Pain
- Infection
- Pocket hematoma
- Wound dehiscence
- Lead dislodgment
- Deep venous thrombosis
- Upper extremity edema
- Degradation of lead function

Chronic complications include the following:

- Device-related pain
- Lead fracture
- Inappropriate shocks
- Erosion of device through skin
- Immunologic rejection - Rare

Infection

ICD infection rates are higher in patients undergoing generator replacement compared with de novo implant.[48] A prospective study revealed an infection rate of 1.3% in patients undergoing device replacement.[49] In this study, postoperative hematoma significantly increased the risk of infection (22.7% vs 0.98%).

Inappropriate shocks

One of the risks of ICD implant is that of inappropriate ICD shocks. An inappropriate ICD shock is one that is not precipitated by accurate detection of a malignant ventricular arrhythmia, ventricular tachycardia (VT), or ventricular fibrillation (VF).[50] Typically, inappropriate ICD shocks result when atrial arrhythmias, such as atrial fibrillation, atrial tachycardia, or atrial flutter, accelerate the ventricular rate beyond the set limit for delivery of ICD shock therapy.

However, inappropriate shocks may also result from sinus tachycardia, supraventricular tachycardia (SVT), illicit drug use (as with cocaine and methamphetamine), and ventricular oversensing. Ventricular oversensing may occur due to T-wave oversensing, electromagnetic interference (EMI), a loose setscrew in the ICD header, or ICD lead fracture.

Analysis of the MADIT II trial data revealed that 11.5% of the ICD patients received inappropriate ICD shocks and that 31.2% of all ICD shocks were deemed inappropriate. Inappropriate ICD shocks were attributed to atrial fibrillation (44%), supraventricular tachycardia (36%), and abnormal sensing (20%). Patients with inappropriate shocks had greater all-cause mortality.[51]

Drug therapy with hydroxymethylglutaryl-coenzyme A reductase inhibitors, or so-called statins, has been shown to reduce,

by more than half, the frequency of inappropriate ICD shocks secondary to occurrence of atrial fibrillation and atrial flutter.[52] However, this finding has not been reproduced in other studies.

There is some indirect evidence that the incidence of inappropriate shocks may be lower in patients with dual-chamber devices compared with patients who receive single-chamber devices.[53]

Failure to shock and ineffective cardioversion

Failure to deliver a shock may be caused by failure to sense, lead fracture, EMI, and inadvertent ICD deactivation. Management includes external defibrillation or cardioversion and antidysrhythmic medications.

Ineffective cardioversion may result from inadequate energy output, rise in defibrillation threshold (possibly due to an antiarrhythmic medication, such as amiodarone, flecainide, or phenytoin), myocardial infarction at the lead site, lead fracture, insulation breakage, scarring at the lead implantation site, and lead dislodgment.

Many ICDs deliver a programmed set of therapies per dysrhythmic episode. The number of therapies per episode is programming specific. If a delivered therapy does not terminate the arrhythmia, the device proceeds to the next programmed therapy. For example, a total of 6 attempts at defibrillation are attempted per episode of ventricular fibrillation. The device attempts defibrillation and then reevaluates the cardiac rhythm. If the arrhythmia persists, it delivers therapy number 2 and so on until all 6 attempts have been delivered. Once this occurs, the device does not deliver therapy until a new episode is declared. Initial therapy for VT may be antitachycardia pacing (also known as overdrive pacing) rather than cardioversion.

ICDs do not prevent all sudden deaths, and acknowledging that cardiac arrest is not necessarily an ICD malfunction is important. The device may have properly delivered the required shocks for the triggering rhythm but still have been ineffective in resolving it.

Sprint Fidelis lead fracture

In July 2007, a higher than expected rate of Sprint Fidelis model 6949 ICD lead fractures were reported. Six patients presented with lead failure 4-23 months after implant. A subsequent database search for similar reports revealed that 33% of affected patients had inappropriate ICD shocks. Analysis of affected leads revealed 33% with high lead impedance and a 35% rate of pace-sense and high-voltage conductor fracture.

The lead manufacturer, Medtronic, Inc, issued an advisory in October 2007 with suggested ICD programming changes aimed at early detection of lead failure and reduction of inappropriate ICD shocks. Medtronic also discontinued sales of the affected leads, which include Sprint Fidelis models 6930, 6931, 6948, and 6949.[54]

A subsequent report found a 3.3% rate (17 of 514) of Sprint Fidelis lead failure 11-35 months after implant. Of the failures in this report, 88% were caused by pace-sense conductor fractures and 12% by high-voltage conductor defects. Of patients with pace-sense conductor fractures, 80% received inappropriate shocks. Notably, impedance monitoring did not prevent inappropriate shocks in two thirds of patients with lead failure.[55]

The Canadian Heart Rhythm Society issued a report on outcomes of the Medtronic Sprint Fidelis family of leads.[56] Lead failure was seen in 1.29% (80 of 6181) of patients at 21 months of observation. Inappropriate shocks were experienced in 56% of patients with lead failure. No deaths were attributed to lead failure. ICD interrogation prior to lead failure revealed evidence of altered lead function in only 10% of failing leads, consistent with the findings of Kallinen et al.[55]

The advisory issued by Medtronic suggested that, in general, the risks of lead replacement surgery outweigh the benefits.[54] Nonetheless, many centers are replacing leads for patients who are pacemaker dependent or who have received prior appropriate ICD shock therapy for treatment of appropriately detected, malignant ventricular arrhythmias.[56]

The Medtronic Sprint Fidelis leads (models 6949, 6948, 6931 and 6930) are subject to an increasing and problematic rate of lead failure.[57] Specific device programming can enhance lead diagnosis, but many lead failures and the consequences of those failures remain unpredicted.[58]

Diagnostic information tracked and recorded in most devices may be used to distinguish between lead fracture and lead connection problems.[59] Current tools and technologies can be used to address Fidelis lead fractures, including a high success rate for lead extraction in specific centers; however, the optimal treatment strategy is evolving.[60]

A study by Morrison et al that sought to compare all-cause mortality in patients with Fidelis and Quattro leads with those with a nonadvisory lead found that of 2671 study patients, adjusted survival was similar with the Fidelis and Quattro leads.[61]

Other leads may also be problematic. For example, the Riata family of ICD leads were recalled on November 28, 2011, due to premature erosion of the insulation around the electrical conductor wires (ie, insulation failure).[62] The manufacturer (St Jude Medical; now, Abbott) estimated approximately 79,000 Riata leads remained implanted in US patients since 2011.[62]

Implantation risk evaluation

The indication for ICD implantation represents a balance between potential benefit and likely risk. The acute risk of ICD implantation is small but is increased by multiple factors. The following are risk factors established by an ICD registry risk score model:[63]

- Age greater than 70 years - 1 point
- Female - 2 points
- New York Heart Association (NYHA) class III - 1 point
- NYHA class IV - 3 points
- Atrial fibrillation - 1 point
- Prior valve surgery - 3 points
- Chronic lung disease - 2 points
- Blood urea nitrogen (BUN) > 30 mg/dL - 2 points
- Reimplantation for reasons other than battery change - 6 points
- Dual chamber ICD type - 2 points
- Biventricular ICD type - 4 points
- Nonelective ICD implant -3 points

The risk of any inhospital complication increases from 0.6% among patients with a score of less than 5 to 8.4% among the patients with greater than 19 risk points.

The American College of Cardiology (ACC) and American Heart Association (AHA), in collaboration with the American Association for Thoracic Surgery (AATS) and the Society of Thoracic Surgeons (STS), have developed an extensive set of guidelines for ICD implantation. These guidelines represent a consensus statement that is largely evidence-based and that summarizes the available clinical evidence as of the time of its publication in May 2008.[1]

Pacemaker Complications

Pacemaker complications include malfunction due to mechanical factors such as pneumothorax, pericarditis, infection, skin erosion, hematoma, lead dislodgment, and venous thrombosis. Treatment depends on the etiology. Pneumothoraces may require medical observation, needle aspiration, or even chest tube placement.

Erosion of the pacer through the skin, while rare, requires device replacement and systemic antibiotics. Hematomas may be treated with direct pressure and observation, rarely requiring surgical drainage.

Lead dislodgment generally occurs within 2 days of device implantation pacer and may be seen on chest radiography. Alternatively, fluctuating impedance may be a subtle clue, as the patient may have normal impedance when the lead is in contact with the endocardium, but infinite (or very high) impedance when the lead is dislodged.

Free-floating ventricular leads may trigger malignant arrhythmias. Device-associated venous thrombosis is rare but generally presents as unilateral arm edema. Treatment includes extremity elevation and anticoagulation.

Advanced life support protocols, including defibrillation, may safely be performed for patients with pacemakers in place. Sternal paddles are placed at a safe distance (10 cm) from the pulse generator. Temporary pacing may become necessary in cases of myocardial infarction, as the current pacemaker discharge settings may be insufficient to stimulate ventricular contraction.

Pacemaker Malfunctions

Major modes of pacemaker malfunctions include the following:

- Failure to output
- Failure to capture
- Failure to sense
- Pacemaker-mediated tachycardia
- Pacemaker syndrome
- Twiddler syndrome
- Cardiac monitor pseudomalfunction
- Pacemaker pseudomalfunction

Failure to output

Failure to output occurs when no pacing artifact is present despite an indication to pace. This may be due to battery failure, lead fracture, fractured lead insulation, oversensing (inhibiting pacer output), poor lead connection at the takeoff from the pacer, and "cross-talk" (ie, a phenomenon occurring when atrial output is sensed by a ventricular lead in a dual-chamber pacer).

Management of pacer output complications includes medications to increase the intrinsic heart rate and placement of a temporary pacer. A chest radiograph is warranted to check pacer leads and to evaluate for possible lead fracture, which occurs most commonly at the clavicle or first rib. The patient's pacer identification card should be obtained and his or her electrophysiologist or cardiologist consulted.

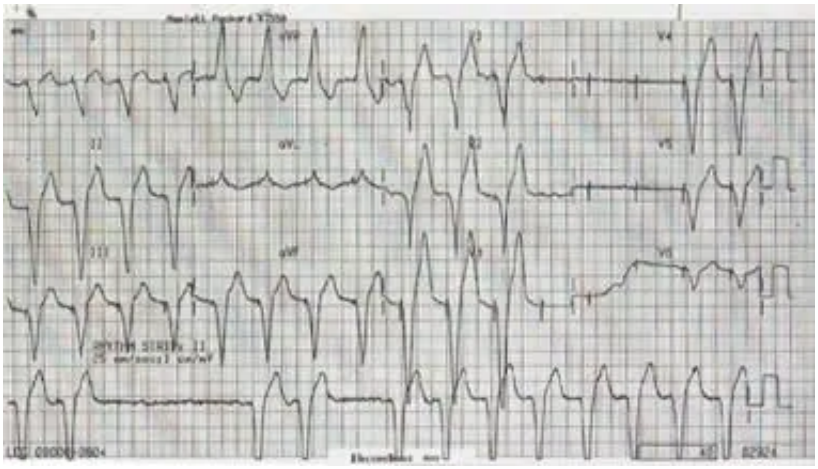
Lead impedance (resistance) may also be an indicator of lead malfunction. Very low impedance may signify a fracture of the insulation (ie, the energy is dissipating into the surrounding tissue), whereas infinite (or very high) impedance may signify either complete lead fracture or a lead tip dislodged from the endocardium.

Failure to capture

Failure to capture occurs when a pacing artifact is not followed by an atrial or a ventricular complex (see the image below). This may be due to the following:

- Lead fracture
- Lead dislodgement
- Fractured lead insulation
- Elevated pacing threshold

- MI at the lead tip
- Drugs (eg, flecainide)
- Metabolic abnormalities (eg, hyperkalemia, acidosis, alkalosis)
- Cardiac perforation
- Poor lead connection at the takeoff from the generator
- Improper amplitude or pulse-width settings



Intermittent periods of ventricular capture.

Fibrosis at the endocardial surface where leads were implanted may also occur in the weeks following pacemaker implantation. The fibrosis may create an electrical resistance barrier preventing ventricular depolarization. This may detected as an abnormally high change in impedance (Δ impedance).

Managing pacer-capture complications is similar to treating output complications, with extra consideration given to treating metabolic abnormalities and potential myocardial infarction (MI). Temporary pacing is used to stabilize the patient until an electrophysiology technician or cardiologist can further evaluate the pacemaker.

Oversensing

Oversensing occurs when a pacer incorrectly senses noncardiac electrical activity and is inhibited from pacing. This may result in a heart rate lower than the preset rate. This form of output failure may be due to muscular activity (particularly of the diaphragm or pectoralis muscles), electromagnetic interference (EMI) (from magnetic resonance imaging [MRI]), or fractured lead insulation. Oversensing is one condition that is diagnosable and treatable with magnet application. As mentioned before, magnet application will convert the pacemaker to asynchronous mode, and it will then operate at the preset rate.

Of note, it has been reported that cellular phones held within 10 cm of the pulse generator may elicit this response.[64]

Individual implantable cardioverter-defibrillator (ICD) manufacturers also have recommendations for unsafe devices that may interact with the ICD (eg, Medtronic's "Electromagnetic Compatibility Guide"[65]).

Undersensing

Undersensing occurs when a pacer incorrectly misses intrinsic depolarization and paces despite intrinsic activity. The pacemaker is more or less operating in asynchronous mode. This may be due to poor lead positioning, lead dislodgment, magnet application, low battery, or MI. Management is similar to that for other types of failures.

Pacemaker-mediated tachycardia

A premature ventricular contraction (PVC) in a dual-chamber pacemaker may precipitate a pacemaker-mediated tachycardia (PMT). If a PVC is transmitted in a retrograde manner through the atrioventricular node, it may, in turn, depolarize the atria. This atrial depolarization is detected by the atrial sensor, which then stimulates the ventricular leads to fire, hence creating an endless loop.

Although the maximum rate is limited by the pacemaker's programmed upper limit, the possibility of developing ischemia exists in susceptible patients. This is another opportunity to use a magnet to diagnose and treat the arrhythmia. The magnet will place the pacemaker into asynchronous mode and sensing will be deactivated, thus preventing continuation of the reentrant dysrhythmia.

Runaway pacemaker

A malfunction of the pacemaker generator resulting in a life-threatening rapid tachycardia (up to 200 beats per minute [bpm]) is known as runaway pacemaker. The generator may malfunction from various causes, although most commonly it is a battery failure or external damage.

This rare medical emergency requires immediate action. An external magnet may induce slower pacing, but it is possible that the device will not respond to magnet application and more aggressive measures may be necessary. If a patient becomes unstable, treatment involves making an incision in the chest wall over the pacemaker and severing the pacemaker leads from the generator. Note that the patient may require temporary pacing as a result.

Pacemaker syndrome

Pacemaker syndrome is a phenomenon in which a patient feels symptomatically worse after pacemaker placement and presents with progressively worsening symptoms of congestive heart failure (CHF). This is mainly due to the loss of atrioventricular synchrony whereby the pathway is reversed and now has a ventricular origin. The atrial contribution to the preload is lost and cardiac output, as well as blood pressure, falls.

Immediate treatment is mainly supportive, whereas long-term treatment involves altering the pacemaker to restore atrioventricular synchrony and possible ventricular synchrony. For example, this may require changing the pacemaker from single-chamber to dual-chamber pacing or to dual-ventricular pacing.

Twiddler syndrome

Some patients will persistently disturb and manipulate the pacemaker generator, resulting in malfunction. A chest radiograph may reveal twisting or coiling or may show lead fracture, dislodgement, or migration. This situation will require surgical correction, with further patient education and counseling.

Cardiac monitor pseudomalfunction

From time to time, cardiac monitors will report an incorrect heart rate, too low or too high, due to inappropriate interpretation of pacing artifacts. Clinicians faced with this issue should first palpate the pulse and correlate with a pulse oximeter plethysmogram to verify the findings on a cardiac monitor. New monitors have settings to adapt for patients with pacemakers and provide more accurate heart rates.

Pacemaker pseudomalfunction

In some clinical settings, an apparent pacing system malfunction is suggested; however, the apparent malfunction is a normal, programmed pacer function. Such pseudomalfunctions are partly due to new algorithms to preserve intrinsic conduction and more physiologic pacing. These can sometimes be corrected by changing the programming; in other cases, the patient may need to have the device changed.

Magnet use inhibits further ICD discharge. It does not, however, inhibit pacing. In some devices, application of a magnet produces a soft beep for each QRS complex. If the magnet is left on for approximately 30 seconds, the ICD is disabled and a continuous tone is generated. To reactivate the device, the magnet must be lifted off the area of the generator and then replaced. After 30 seconds, the beep returns for every QRS complex. Indications for ICD deactivation are as follows:

- End-of-life care - After a discussion with the patient and family
- Inappropriate shocks
- During resuscitation
- With transcutaneous pacing - External pacing can cause an ICD to fire
- During procedures such as central lines or surgery with electrocautery

Inpatient Care

One of the most difficult decisions after a patient presents to the emergency department (ED) complaining of an implantable cardioverter-defibrillator (ICD) discharge is to determine if the discharge was appropriate. Whenever possible, the device should be interrogated, since, unless the shock and the rhythm that preceded it were witnessed, it is not possible to determine shock appropriateness without investigation.

Reasons for admission may include the following:

- Device investigation - To determine whether there is an imminent battery failure (multiple shocks will deplete battery life)
- Addition of antiarrhythmic medications
- Treatment of myocardial infarction (which may be linked to the initial discharge)
- Treatment of patient discomfort
- Provision of psychological support - Up to 35% of patients develop anxiety disorder following ICD placement, although disabling problems necessitating admission are fairly uncommon[2]

Resuscitation of Patients With an ICD

If a patient enters a life-threatening cardiac arrhythmia, advanced cardiac life support (ACLS) protocols should be initiated immediately. Although an implantable cardioverter-defibrillator (ICD) will attempt defibrillation, chest compressions should be continued. Note that some of the current may enter the rescuer; aside from some mild discomfort, however, there has never been a reported case of rescuer injury from this.[45]

Ventricular tachycardia (VT) and ventricular fibrillation (VF) refractory to ICD defibrillation will require external defibrillation and/or antiarrhythmic medications as dictated by ACLS protocols. If external defibrillation is required, attempt to keep the generator at least 10 cm away and out of the shock wave. Defibrillation that affects the generator may cause total device failure. However, do not withhold therapy for fear of damaging the ICD.

If rescuers are uncomfortable with ICD discharge during resuscitation, deactivation of the ICD with a magnet is indicated.

Central Venous Catheter Placement in ICD Patients

Pacemaker or implantable cardioverter-defibrillator (ICD) leads placed in the venous system often have surrounding thrombosis, with 20% of patients having complete occlusion at 2 years.[66] If a metal guidewire contacts the lead system during central line placement, there may be enough noisy artifact to trigger an inappropriate shock.

Consideration should be given to either avoid a metal guidewire or deactivate the ICD during central line placement. Although the contralateral subclavian or internal jugular vein can be cannulated with care, femoral vein access is a much

safer option.

Consultations and Monitoring

Some patients with implantable cardioverter-defibrillator (ICDs) require emotional or psychological support for anxiety, depression, and difficulties in adjusting to life with an ICD. The dedicated ICD clinic staff can help with many of these issues. Referral to a psychologist or psychiatrist may also be helpful. Support groups are available for patients and their families, some of which are accessible online.

Patients with ICDs should be observed in a dedicated ICD clinic. Patients are seen more frequently early after implant—generally, 1 week after implant for a wound check, 1 month after implant for device interrogation, and 3 months after implant for repeat device interrogation. The follow-up interval generally can be increased to every 6 months in patients who are clinically stable.

Imaging safety and artifacts

Magnetic resonance imaging (MRI) scans were safely performed in a study of 15 patients with subcutaneous ICDs (S-ICD System). All scans (5 of the brain, 4 of the heart, 6 of the L-spine, and 2 of the C-spine) were performed at least 6 weeks after implantation of the ICD, which was turned off during imaging. There was no evidence of movement or rotation of the ICD, and no malfunctions were found on postscan checks up to 12 months later. However, as MRI-conditional devices become specifically approved, MRI studies of nonconditional devices is generally avoided at many centers—or performed only on specific protocols. Protocols for the imaging of conditional devices are also typically specified, and they may dictate certain programming adjustments, lead parameters, or delay after implantation.

MRI imaging of the brain, C-spine, and L-spine was free of artifacts. Heart imaging showed no major artifacts in the right chamber, but interference in the left chamber view. The procedure was repeated with reduced radiofrequency field exposure, reduced turbo factors, increased repetition time, and reduced flip angle in two patients who complained of heating over the subcutaneous pocket caused by the thermistor during their first L-spine scan.[67]

Schukro and Puchner investigated the safety and diagnostic efficiency of routine low-field MRI in 338 patients with pacemakers and ICDs. All completed scans could be analyzed efficiently and no induction of arrhythmia or inhibition of pacemaker function occurred. They concluded low-field MRI examinations (0.2 T) were safe and efficient for patients with cardiac rhythm management devices.[68]

Rashid and colleagues developed a modified wideband late gadolinium enhancement (LGE) MRI technique that can overcome hyperintensity image artifacts caused by implanted cardiac devices. In their study of 12 patients with ICDs, use of the wideband LGE sequence eliminated the severe, uninterpretable hyperintensity artifacts in the left ventricular wall that occurred with conventional LGE technique, thereby enabling confident evaluation of myocardial viability.[69]

Patient Education

Although technologic advances have greatly reduced the potential effects of electromagnetic interference (EMI), patients should be advised to avoid strong electromagnetic fields because of potential interference with sensing circuitry. Examples of potential hazards include arc welders, large generators, and magnetic resonance imaging (MRI) magnets. Household appliances, microwave ovens, cell phones, and hand-held metal detectors (used for security screening) should not pose a serious threat.[70]

A shock from an implantable cardioverter-defibrillator (ICD) is generally painful. Patients should be advised of this in advance. Advise patients and their families that someone touching them is not harmed if the ICD discharges. Issues regarding driving can be problematic. In the absence of specific state laws, many electrophysiologists recommend that patients be shock-free for 6 months before resuming driving. Loss of driving privileges imposes an enormous burden and change of lifestyle on patients with this restriction. Rules and recommendations regarding commercial driving typically are more stringent.

Questions & Answers

Overview

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References

1. [Guideline] Epstein AE, DiMarco JP, Ellenbogen KA, et al. ACC/AHA/HRS 2008 Guidelines for Device-Based Therapy of Cardiac Rhythm Abnormalities: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the ACC/AHA/NASPE 2002 Guideline Update for Implantation of Cardiac Pacemakers and Antiarrhythmia Devices) developed in collaboration with the American Association for Thoracic Surgery and Society of Thoracic Surgeons. J Am Coll Cardiol. 2008 May 27. 51(21):e1-62. [QxMD MEDLINE Link].

2. Kamphuis HC, de Leeuw JR, Derksen R, Hauer RN, Winnubst JA. Implantable cardioverter defibrillator recipients: quality of life in recipients with and without ICD shock delivery: a prospective study. Europace. 2003 Oct. 5(4):381-9. [QxMD MEDLINE Link].

3. Mirowski M, Reid PR, Mower MM, et al. Termination of malignant ventricular arrhythmias with an implanted automatic defibrillator in human beings. N Engl J Med. 1980 Aug 7. 303(6):322-4. [QxMD MEDLINE Link].

4. Robinson LA, Windle JR, Easley AR. Options in managing the patient with high defibrillation thresholds. Ann Thorac Surg. 1994 May. 57(5):1184-92. [QxMD MEDLINE Link].

5. Bardy GH, Poole JE, Kudenchuk PJ, et al. A prospective randomized comparison in humans of biphasic waveform 60-microF and 120-microF capacitance pulses using a unipolar defibrillation system. Circulation. 1995 Jan 1. 91(1):91-5. [QxMD MEDLINE Link].

6. Kroll MW, Swerdlow CD. Optimizing defibrillation waveforms for ICDs. J Interv Card Electrophysiol. 2007 Apr. 18(3):247-63. [QxMD MEDLINE Link].

7. U.S. Food and Drug Administration. FDA approves first subcutaneous heart defibrillator. Available at http://www.fda.gov/NewsEvents/Newsroom/PressAnnouncements/ucm321755.htm. Accessed: September 28, 2012.

8. Wever EF, Hauer RN, van Capelle FL, et al. Randomized study of implantable defibrillator as first-choice therapy versus conventional strategy in postinfarct sudden death survivors. Circulation. 1995 Apr 15. 91(8):2195-203. [QxMD MEDLINE Link].

9. The Antiarrhythmics versus Implantable Defibrillators (AVID) Investigators. A comparison of antiarrhythmic-drug therapy with implantable defibrillators in patients resuscitated from near-fatal ventricular arrhythmias. The Antiarrhythmics versus Implantable Defibrillators (AVID) Investigators. N Engl J Med. 1997 Nov 27. 337(22):1576-83. [QxMD MEDLINE Link].

10. Connolly SJ, Gent M, Roberts RS, et al. Canadian implantable defibrillator study (CIDS) : a randomized trial of the implantable cardioverter defibrillator against amiodarone. Circulation. 2000 Mar 21. 101(11):1297-302. [QxMD MEDLINE Link].

11. Bokhari F, Newman D, Greene M, Korley V, Mangat I, Dorian P. Long-term comparison of the implantable cardioverter defibrillator versus amiodarone: eleven-year follow-up of a subset of patients in the Canadian Implantable Defibrillator Study (CIDS). Circulation. 2004 Jul 13. 110(2):112-6. [QxMD MEDLINE Link].

12. Kuck KH, Cappato R, Siebels J, Rüppel R. Randomized comparison of antiarrhythmic drug therapy with implantable defibrillators in patients resuscitated from cardiac arrest : the Cardiac Arrest Study Hamburg (CASH). Circulation. 2000 Aug 15. 102(7):748-54. [QxMD MEDLINE Link].

13. Siebels J, Kuck KH. Implantable cardioverter defibrillator compared with antiarrhythmic drug treatment in cardiac arrest survivors (the Cardiac Arrest Study Hamburg). Am Heart J. 1994 Apr. 127(4 Pt 2):1139-44. [QxMD MEDLINE Link].

14. Moss AJ, Hall WJ, Cannom DS, et al. Improved survival with an implanted defibrillator in patients with coronary disease at high risk for ventricular arrhythmia. Multicenter Automatic Defibrillator Implantation Trial (MADIT) Investigators. N Engl J Med. 1996 Dec 26. 335(26):1933-40. [QxMD MEDLINE Link].

15. Buxton AE, Lee KL, Fisher JD, et al. A randomized study of the prevention of sudden death in patients with coronary artery disease. Multicenter Unsustained Tachycardia Trial (MUSTT) Investigators. N Engl J Med. 1999 Dec 16. 341(25):1882-90. [QxMD MEDLINE Link].

16. Buxton AE, Lee KL, Hafley GE, et al. Relation of ejection fraction and inducible ventricular tachycardia to mode of death in patients with coronary artery disease: an analysis of patients enrolled in the multicenter unsustained tachycardia trial. Circulation. 2002 Nov 5. 106(19):2466-72. [QxMD MEDLINE Link].

17. Fisher JD, Buxton AE, Lee KL, et al. Designation and distribution of events in the Multicenter UnSustained Tachycardia Trial (MUSTT). Am J Cardiol. 2007 Jul 1. 100(1):76-83. [QxMD MEDLINE Link].

18. Moss AJ, Zareba W, Hall WJ. Prophylactic implantation of a defibrillator in patients with myocardial infarction and reduced ejection fraction (MADIT II). N Engl J Med. 2002 Mar 21. 346(12):877-83. [QxMD MEDLINE Link].

19. Zwanziger J, Hall WJ, Dick AW, et al. The cost effectiveness of implantable cardioverter-defibrillators: results from the Multicenter Automatic Defibrillator Implantation Trial (MADIT)-II. J Am Coll Cardiol. 2006 Jun 6. 47(11):2310-8. [QxMD MEDLINE Link]. [Full Text].

20. Bristow MR, Saxon LA, Boehmer J, et al. Cardiac-resynchronization therapy with or without an implantable defibrillator in advanced chronic heart failure. N Engl J Med. 2004 May 20. 350(21):2140-50. [QxMD MEDLINE Link].

21. Bardy GH, Lee KL, Mark DB, et al. Amiodarone or an implantable cardioverter-defibrillator for congestive heart failure. N Engl J Med. 2005 Jan 20. 352(3):225-37. [QxMD MEDLINE Link].

22. Adabag S, Roukoz H, Anand IS, Moss AJ. Cardiac resynchronization therapy in patients with minimal heart failure a systematic review and meta-analysis. J Am Coll Cardiol. 2011 Aug 23. 58(9):935-41. [QxMD MEDLINE Link].

23. Levy WC, Lee KL, Hellkamp AS, et al. Maximizing survival benefit with primary prevention implantable cardioverter-defibrillator therapy in a heart failure population. Circulation. 2009 Sep 8. 120(10):835-42. [QxMD MEDLINE Link]. [Full Text].

24. Santangeli P, Pelargonio G, Russo AD, et al. Gender differences in clinical outcome and primary prevention defibrillator benefit in patients with severe left ventricular dysfunction: A systematic review and meta-analysis. Heart Rhythm. 2010 Jul. 7(7):876-82. [QxMD MEDLINE Link].

25. Kadish A, Dyer A, Daubert JP, et al. Prophylactic defibrillator implantation in patients with nonischemic dilated cardiomyopathy. N

Engl J Med. 2004 May 20. 350(21):2151-8. [QxMD MEDLINE Link].

26. Bansch D, Antz M, Boczor S, et al. Primary prevention of sudden cardiac death in idiopathic dilated cardiomyopathy: the Cardiomyopathy Trial (CAT). Circulation. 2002 Mar 26. 105(12):1453-8. [QxMD MEDLINE Link]. [Full Text].

27. Bigger JT Jr. Prophylactic use of implanted cardiac defibrillators in patients at high risk for ventricular arrhythmias after coronary-artery bypass graft surgery. Coronary Artery Bypass Graft (CABG) Patch Trial Investigators. N Engl J Med. 1997 Nov 27. 337(22):1569-75. [QxMD MEDLINE Link]. [Full Text].

28. Hohnloser SH, Kuck KH, Dorian P, et al. Prophylactic use of an implantable cardioverter-defibrillator after acute myocardial infarction. N Engl J Med. 2004 Dec 9. 351(24):2481-8. [QxMD MEDLINE Link].

29. Strickberger SA, Hummel JD, Bartlett TG, et al. Amiodarone versus implantable cardioverter-defibrillator:randomized trial in patients with nonischemic dilated cardiomyopathy and asymptomatic nonsustained ventricular tachycardia--AMIOVIRT. J Am Coll Cardiol. 2003 May 21. 41(10):1707-12. [QxMD MEDLINE Link].

30. Barsheshet A, Moss AJ, Eldar M, et al. Time-dependent benefit of preventive cardiac resynchronization therapy after myocardial infarction. Eur Heart J. 2011 Jul. 32(13):1614-21. [QxMD MEDLINE Link].

31. Pouleur AC, Barkoudah E, Uno H, et al. Pathogenesis of sudden unexpected death in a clinical trial of patients with myocardial infarction and left ventricular dysfunction, heart failure, or both. Circulation. 2010 Aug 10. 122(6):597-602. [QxMD MEDLINE Link]. [Full Text].

32. Santangeli P, Di Biase L, Dello Russo A, et al. Meta-analysis: age and effectiveness of prophylactic implantable cardioverter-defibrillators. Ann Intern Med. 2010 Nov 2. 153(9):592-9. [QxMD MEDLINE Link].

33. Healey JS, Hallstrom AP, Kuck KH, et al. Role of the implantable defibrillator among elderly patients with a history of life-threatening ventricular arrhythmias. Eur Heart J. 2007 Jul. 28(14):1746-9. [QxMD MEDLINE Link].

34. Wilcox JE, Fonarow GC, Zhang Y, et al. Clinical effectiveness of cardiac resynchronization and implantable cardioverter-defibrillator therapy in men and women with heart failure: findings from IMPROVE HF. Circ Heart Fail. 2014 Jan. 7(1):146-53. [QxMD MEDLINE Link]. [Full Text].

35. Pokorney SD, Hellkamp AS, Yancy CW, et al. Primary prevention implantable cardioverter-defibrillators in older racial and ethnic minority patients. Circ Arrhythm Electrophysiol. 2015 Feb. 8(1):145-51. [QxMD MEDLINE Link]. [Full Text].

36. Erkapic D, Sperzel J, Stiller S, et al, for the INSURE Investigators. Long-term benefit of implantable cardioverter/defibrillator therapy after elective device replacement: results of the INcidence free SUrvival after ICD REplacement (INSURE) trial--a prospective multicentre study. Eur Heart J. 2013 Jan. 34(2):130-7. [QxMD MEDLINE Link].

37. Zareba W, Moss AJ, Daubert JP, et al. Implantable cardioverter defibrillator in high-risk long QT syndrome patients. J Cardiovasc Electrophysiol. 2003 Apr. 14(4):337-41. [QxMD MEDLINE Link].

38. Viskin S. Implantable cardioverter defibrillator in high-risk long QT syndrome patients. J Cardiovasc Electrophysiol. 2003 Oct. 14(10):1130-1; reply 1131. [QxMD MEDLINE Link].

39. Goel AK, Berger S, Pelech A, et al. Implantable cardioverter defibrillator therapy in children with long QT syndrome. Pediatr Cardiol. 2004 Jul-Aug. 25(4):370-8. [QxMD MEDLINE Link].

40. Monnig G, Kobe J, Loher A, et al. Implantable cardioverter-defibrillator therapy in patients with congenital long-QT syndrome: a long-term follow-up. Heart Rhythm. 2005 May. 2(5):497-504. [QxMD MEDLINE Link].

41. Goldenberg I, Mathew J, Moss AJ, et al. Corrected QT variability in serial electrocardiograms in long QT syndrome: the importance of the maximum corrected QT for risk stratification. J Am Coll Cardiol. 2006 Sep 5. 48(5):1047-52. [QxMD MEDLINE Link].

42. Hobbs JB, Peterson DR, Moss AJ, et al. Risk of aborted cardiac arrest or sudden cardiac death during adolescence in the long-QT syndrome. JAMA. 2006 Sep 13. 296(10):1249-54. [QxMD MEDLINE Link].

43. Ghanbari H, Phard WS, Al-Ameri H, et al. Meta-analysis of safety and efficacy of uninterrupted warfarin compared to heparin-based bridging therapy during implantation of cardiac rhythm devices. Am J Cardiol. 2012 Nov 15. 110(10):1482-8. [QxMD MEDLINE Link].

44. Bernstein AD, Daubert JC, Fletcher RD, et al. The revised NASPE/BPEG generic code for antibradycardia, adaptive-rate, and multisite pacing. North American Society of Pacing and Electrophysiology/British Pacing and Electrophysiology Group. Pacing Clin Electrophysiol. 2002 Feb. 25(2):260-4. [QxMD MEDLINE Link].

45. McMullan J, Valento M, Attari M, Venkat A. Care of the pacemaker/implantable cardioverter defibrillator patient in the ED. Am J Emerg Med. 2007 Sep. 25(7):812-22. [QxMD MEDLINE Link].

46. Lampert R, Hayes DL, Annas GJ, et al. HRS expert consensus statement on the management of Cardiovascular Implantable Electronic Devices (CIEDs) in patients nearing end of life or requesting withdrawal of therapy. Heart Rhythm. 2010 Jul. 7(7):1008-26. [QxMD MEDLINE Link].

47. Freeman JV, Wang Y, Curtis JP, Heidenreich PA, Hlatky MA. Physician procedure volume and complications of cardioverter-defibrillator implantation. Circulation. 2012 Jan 3. 125(1):57-64. [QxMD MEDLINE Link].

48. Uslan DZ, Gleva MJ, Warren DK, et al. Cardiovascular implantable electronic device replacement infections and prevention: results from the REPLACE Registry. Pacing Clin Electrophysiol. 2012 Jan. 35(1):81-7. [QxMD MEDLINE Link].

49. Curtis JP, Luebbert JJ, Wang Y, et al. Association of physician certification and outcomes among patients receiving an implantable cardioverter-defibrillator. JAMA. 2009 Apr 22. 301(16):1661-70. [QxMD MEDLINE Link].

50. Fischer A, Ousdigian KT, Johnson JW, Gillberg JM, Wilkoff BL. The impact of atrial fibrillation with rapid ventricular rates and device programming on shocks in 106,513 ICD and CRT-D patients. Heart Rhythm. 2012 Jan. 9(1):24-31. [QxMD MEDLINE Link].

51. Daubert JP, Zareba W, Cannom DS, et al. Inappropriate implantable cardioverter-defibrillator shocks in MADIT II: frequency, mechanisms, predictors, and survival impact. J Am Coll Cardiol. 2008 Apr 8. 51(14):1357-65. [QxMD MEDLINE Link].

52. Bhavnani SP, Coleman CI, White CM, et al. Association between statin therapy and reductions in atrial fibrillation or flutter and inappropriate shock therapy. Europace. 2008 Jul. 10(7):854-9. [QxMD MEDLINE Link].

53. Almendral J, Arribas F, Wolpert C, et al. Dual-chamber defibrillators reduce clinically significant adverse events compared with single-chamber devices: results from the DATAS (Dual chamber and Atrial Tachyarrhythmias Adverse events Study) trial. Europace. 2008 May. 10(5):528-35. [QxMD MEDLINE Link].

54. Hauser RG, Kallinen LM, Almquist AK, et al. Early failure of a small-diameter high-voltage implantable cardioverter-defibrillator lead. Heart Rhythm. 2007 Jul. 4(7):892-6. [QxMD MEDLINE Link]. [Full Text].

55. Kallinen LM, Hauser RG, Lee KW, et al. Failure of impedance monitoring to prevent adverse clinical events caused by fracture of a recalled high-voltage implantable cardioverter-defibrillator lead. Heart Rhythm. 2008 Jun. 5(6):775-9. [QxMD MEDLINE Link].

56. Krahn AD, Champagne J, Healey JS, et al. Outcome of the Fidelis implantable cardioverter-defibrillator lead advisory: a report from the Canadian Heart Rhythm Society Device Advisory Committee. Heart Rhythm. 2008 May. 5(5):639-42. [QxMD MEDLINE Link].

57. Faulknier BA, Traub DM, Aktas MK, et al. Time-dependent risk of Fidelis lead failure. Am J Cardiol. 2010 Jan 1. 105(1):95-9. [QxMD MEDLINE Link].

58. Kallinen LM, Hauser RG, Tang C, et al. Lead integrity alert algorithm decreases inappropriate shocks in patients who have Sprint Fidelis pace-sense conductor fractures. Heart Rhythm. 2010 Aug. 7(8):1048-55. [QxMD MEDLINE Link].

59. Swerdlow CD, Sachanandani H, Gunderson BD, et al. Preventing overdiagnosis of implantable cardioverter-defibrillator lead fractures using device diagnostics. J Am Coll Cardiol. 2011 Jun 7. 57(23):2330-9. [QxMD MEDLINE Link].

60. Maytin M, Love CJ, Fischer A, et al. Multicenter experience with extraction of the Sprint Fidelis implantable cardioverter-defibrillator lead. J Am Coll Cardiol. 2010 Aug 17. 56(8):646-50. [QxMD MEDLINE Link].

61. Morrison TB, Friedman PA, Kallinen LM, et al. Impact of implanted recalled Sprint Fidelis lead on patient mortality. J Am Coll Cardiol. 2011 Jul 12. 58(3):278-83. [QxMD MEDLINE Link]. [Full Text].

62. US Food & Drug Administration. Premature insulation failure in recalled Riata implantable cardioverter defibrillator (ICD) leads manufactured by St. Jude Medical, Inc.: FDA safety communication [archived]. Available at http://www.fda.gov/MedicalDevices/Safety/AlertsandNotices/ucm314930.htm. January 12, 2015; Accessed: February 13, 2017.

63. Haines DE, Wang Y, Curtis J. Implantable cardioverter-defibrillator registry risk score models for acute procedural complications or death after implantable cardioverter-defibrillator implantation. Circulation. 2011 May 17. 123(19):2069-76. [QxMD MEDLINE Link].

64. Thaker JP, Patel MB, Jongnarangsin K, Liepa VV, Thakur RK. Electromagnetic interference with pacemakers caused by portable media players. Heart Rhythm. 2008 Apr. 5(4):538-44. [QxMD MEDLINE Link].

65. Medtronic. Electromagnetic Compatibility Guide. Medtronic. Available at http://www.medtronic.com/rhythms/downloads/UC200602918EN.pdf.

66. Pavia S, Wilkoff B. The management of surgical complications of pacemaker and implantable cardioverter-defibrillators. Curr Opin Cardiol. 2001 Jan. 16(1):66-71. [QxMD MEDLINE Link].

67. Johnson K. MRI safe in patients with subcutaneous defibrillators. Medscape Medical News. March 11, 2014. Available at http://www.medscape.com/viewarticle/821808. Accessed: March 17, 2014.

68. Schukro C, Puchner SB. Safety and efficiency of low-field magnetic resonance imaging in patients with cardiac rhythm management devices. Eur J Radiol. 2019 Sep. 118:96-100. [QxMD MEDLINE Link].

69. Rashid S, Rapacchi S, Vaseghi M, et al. Improved late gadolinium enhancement MR imaging for patients with implanted cardiac devices. Radiology. 2014 Jan. 270(1):269-74. [QxMD MEDLINE Link]. [Full Text].

70. Jilek C, Tzeis S, Vrazic H, et al. Safety of screening procedures with hand-held metal detectors among patients with implanted cardiac rhythm devices: a cross-sectional analysis. Ann Intern Med. 2011 Nov 1. 155(9):587-92. [QxMD MEDLINE Link].

71. PRNewswire. Boston Scientific announces FDA approval of innovative new defibrillators and heart failure devices [news release]. Boston Scientific. Available at http://news.bostonscientific.com/2014-04-15-Boston-Scientific-Announces-FDA-Approval-Of-Innovative-New-Defibrillators-And-Heart-Failure-Devices. April 15, 2014; Accessed: April 27, 2014.

72. Bardy GH, Smith WM, Hood MA, et al. An entirely subcutaneous implantable cardioverter-defibrillator. N Engl J Med. 2010 Jul 1. 363(1):36-44. [QxMD MEDLINE Link]. [Full Text].

73. Buxton AE, Lee KL, DiCarlo L, et al. Electrophysiologic testing to identify patients with coronary artery disease who are at risk for sudden death. Multicenter Unsustained Tachycardia Trial Investigators. N Engl J Med. 2000 Jun 29. 342(26):1937-45. [QxMD MEDLINE Link].

74. [Guideline] Gregoratos G, Abrams J, Epstein AE, et al. ACC/AHA/NASPE 2002 guideline update for implantation of cardiac pacemakers and antiarrhythmia devices: summary article: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (ACC/AHA/NASPE Committee). Circulation. 2002 Oct 15. 106(16):2145-61. [QxMD MEDLINE Link]. [Full Text].

75. [Guideline] Gregoratos G, Cheitlin MD, Conill A, et al. ACC/AHA guidelines for implantation of cardiac pacemakers and antiarrhythmia devices: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Pacemaker Implantation). J Am Coll Cardiol. 1998 Apr. 31(5):1175-209. [QxMD MEDLINE Link].

76. Mirowski M. The automatic implantable cardioverter-defibrillator: an overview. J Am Coll Cardiol. 1985 Aug. 6(2):461-6. [QxMD MEDLINE Link].

77. Myerburg RJ. Implantable cardioverter-defibrillators after myocardial infarction. N Engl J Med. 2008 Nov 20. 359(21):2245-53. [QxMD MEDLINE Link].

78. Scott PA, Silberbauer J, McDonagh TA, Murgatroyd FD. Impact of prolonged implantable cardioverter-defibrillator arrhythmia detection times on outcomes: a meta-analysis. Heart Rhythm. 2014 May. 11(5):828-35. [QxMD MEDLINE Link].

79. Stiles S. Cutting inappropriate ICD shocks: Long arrhythmia-detection time strategy confirmed. Heartwire from Medscape. February 21, 2014. Available at http://www.medscape.com/viewarticle/820918. Accessed: March 17, 2014.

80. Winkle RA, Mead RH, Ruder MA, et al. Long-term outcome with the automatic implantable cardioverter-defibrillator. J Am Coll Cardiol. 1989 May. 13(6):1353-61. [QxMD MEDLINE Link]. [Full Text].

81. US Food and Drug Administration. FDA approves first leadless pacemaker to treat heart rhythm disorders [news release]. April 6, 2016. Available at http://www.fda.gov/NewsEvents/Newsroom/PressAnnouncements/ucm494417.htm. Accessed: June 2, 2016.

82. Maeda R, Minami Y, Haruki S, et al. Implantable cardioverter defibrillator therapy and sudden death risk stratification in hypertrophic cardiomyopathy patients with midventricular obstruction: A single-center experience. Int J Cardiol. 2016 Jul 1. 214:419-22. [QxMD MEDLINE Link].

83. Migliore F, Silvano M, Zorzi A, et al. Implantable cardioverter defibrillator therapy in young patients with cardiomyopathies and channelopathies: a single Italian centre experience. J Cardiovasc Med (Hagerstown). 2016 Jul. 17(7):485-93. [QxMD MEDLINE Link].

84. Hikage T, Nojima T, Fujimoto H. Active implantable medical device EMI assessment for wireless power transfer operating in LF and HF bands. Phys Med Biol. 2016 Jun 21. 61(12):4522-36. [QxMD MEDLINE Link].

85. Hayashi Y, Takagi M, Kakihara J, et al. Safety and efficacy of high-rate cutoff and long detection interval ICD programming in secondary prevention patients. Heart Vessels. 2017 Feb. 32(2):175-85. [QxMD MEDLINE Link].

86. Maurizi N, Tanini I, Olivotto I, et al. Effectiveness of subcutaneous implantable cardioverter-defibrillator testing in patients with hypertrophic cardiomyopathy. Int J Cardiol. 2017 Mar 15. 231:115-9. [QxMD MEDLINE Link].

87. Younes A, Al-Kindi SG, Alajaji W, Mackall JA, Oliveira GH. Presence of implantable cardioverter-defibrillators and wait-list mortality of patients supported with left ventricular assist devices as bridge to heart transplantation. Int J Cardiol. 2017 Mar 15. 231:211-5. [QxMD MEDLINE Link].

88. Giammaria M, Cerrato E, Imazio M, Curnis A. Active implanted cardiac devices and magnetic resonance: results of a survey among cardiologists of Piedmont. J Cardiovasc Med (Hagerstown). 2017 Mar. 18(3):185-95. [QxMD MEDLINE Link].

89. Ip JE, Wu MS, Kennel PJ, et al. Eligibility of pacemaker patients for subcutaneous implantable cardioverter defibrillators. J Cardiovasc Electrophysiol. 2017 May. 28(5):544-8. [QxMD MEDLINE Link].

90. Farid TA, Omer MA, Gosch K, et al. Stability of pacing indices and need for pacing in cardiac transplant patients over 1 year of follow-up. J Interv Card Electrophysiol. 2017 Jun. 49(1):27-32. [QxMD MEDLINE Link].

91. Padmanabhan D, Kella DK, Deshmukh AJ, et al. Safety of thoracic magnetic resonance imaging for patients with pacemakers and defibrillators. Heart Rhythm. 2019 May 29. [QxMD MEDLINE Link].