The implantable cardioverter defibrillator

Michael Glikson, Paul A Friedman

Implantable cardioverter defibrillators (ICDs) have evolved from the treatment of last resort to the gold standard therapy for patients at high risk for ventricular tachyarrhythmias. High-risk patients include those who have survived life-threatening arrhythmias, and individuals with cardiac diseases who are at risk for such arrhythmias, but are symptomless. Use of an ICD will affect the patient’s quality of life. Some drugs can substantially affect defibrillator function and efficacy, and possible drug-device interactions should be considered. Patients with ICDs may encounter cell phones, antitheft detectors, and many other sources of potential electromagnetic interference. In addition to treating ventricular tachyarrhythmias, new defibrillators provide full featured dual chamber pacing, and could treat atrial arrhythmias, and congestive heart failure by means of biventricular pacing.

Development of the implantable cardioverter defibrillator was pioneered by Michel Mirowski in the late 1960s after the death of a close friend and mentor who had been admitted to hospital with recurrent ventricular tachyarrhythmias. His frustration with the limitations of available treatments for high-risk individuals led to the idea of an implantable device that would continuously monitor the cardiac rhythm and deliver defibrillating shocks when ventricular tachyarrhythmias occurred. During the 1970s, experimental models were built and refined by Mirowski and Morton Mower, leading to the first implantation of a defibrillator in a patient, with two previous cardiac arrests was in 1980.¹ For several years implantable cardioverter defibrillator (ICD) treatment was limited to patients with documented cardiac arrest due to ventricular fibrillation and was available in few centres. However, in 1985 the US Food and Drug Administration approved commercial devices. Experience with these devices expanded as did evidence of the effectiveness of defibrillators in terminating malignant ventricular arrhythmias. By 1991, when two expert panels published independent recommendations, indications had broadened to include ventricular fibrillation or ventricular tachycardia not amenable to drug treatment.² Since that time, because of further refinements in device technology, loss of faith in the universal effectiveness of drug treatment, and accumulating evidence from randomised clinical trials, ICDs have become the treatment of choice for patients at high risk for life-threatening arrhythmias.³

**Implantable defibrillator overview**

The implantable defibrillator has two components, the pulse generator and leads. The defibrillator generator houses the battery and the circuitry used for pacing pulse and shock generation, for signal filtering and analysis, and for data storage (figure 1). Battery depletion is the most common indication for replacement, and today’s devices will last 5–7 years, depending on shock and pacing frequency. Before a high-energy shock can be delivered, the charge must be accumulated in a capacitor, because the battery itself is not capable of delivering the amount of charge required in the short time-frame of a defibrillation shock. Capacitors are bulky but innovations in capacitor technology have contributed to the reduction in defibrillator size over the past 10 years. Because of the decrease in pulse-generator size, defibrillators are almost always implanted in the pectoral area rather than in the abdomen, making it convenient for the patient, lowering implant morbidity, and diminishing long-term lead-related complications.⁴,⁵

Defibrillator leads transmit electrical signals from the heart to the pulse generator for analysis, and deliver pacing and shocking pulses to the myocardium. Early systems used contoured epicardial patches for defibrillation because of their superior distribution of the.
defibrillating electric field. Despite their defibrillation efficacy, epicardial systems had the disadvantage of necessitating a thoracotomy for implantation with its attendant risks. This led to the development of transvenous lead systems, which eliminated the need for thoracotomy, diminished implant morbidity, and increased long-term lead reliability.6 Despite clear advantages because of their uneven distribution of the electric field, non-thoracotomy leads often needed greater energy for defibrillation than epicardial systems. In order to achieve adequate defibrillation, separate shocking elements were often needed in the form of superior vena cava leads, coronary sinus leads, or subcutaneous patches or arrays.7 Within a few years, however, advances in waveform and lead design nearly eliminated the need for this added complexity.7 Biphasic shock waveforms, for reasons that remain poorly understood, resulted in substantial improvements in defibrillation efficacy. The use of the pulse generator shell with its large surface area as one of the leads for defibrillation also enhanced defibrillation efficacy.8

Defibrillation functionality has increased with each new generation of device. The realisation that many shocks are followed by prolonged asystole led to the addition of simple back-up pacing (VVI) to the initial shock-only devices. Introduction of low-energy shocks for treatment of slower ventricular tachycardias, based on discrimination between tachycardia and fibrillation by rate criteria, resulted in greater treatment specificity and tolerance by patients in second-generation devices. Third-generation devices introduced antitachycardia overdrive pacing, allowing painless termination of most episodes of ventricular tachycardia by avoidance of shock treatment altogether.9,10 Subsequent generations added extensive stored information on arrhythmia episodes, including endocardial electrocardiography recordings (electrograms) and measurements of cycle-length intervals.11,12 Sophisticated data on individual episodes and the status of the device could be retrieved non-invasively by radiofrequency telemetry. Dual-chamber implantable defibrillators able to provide physiological (DDD or DDDR) pacing have emerged. Dual-chamber devices may enhance discrimination of ventricular from supraventricular arrhythmias by analysis of atrial electrical activity. The latest generation of devices provide treatment for atrial fibrillation and other supraventricular arrhythmias that frequently occur in device recipients.11,12 Devices that support mechanical pump function via biventricular pacing are under assessment.13

**Principles of operation**

All implantable defibrillator systems have four essential functions: arrhythmia detection, arrhythmia treatment, bradycardia pacing, and episode-data storage.

**Detection**

Detection of a ventricular arrhythmia is based mainly on two rhythm characteristics—heart rate and arrhythmia duration (typically 1–3 s) that are programmable to meet the needs of the individual patient.14 The rate criterion distinguishes a tachyarrhythmia from a normal rhythm, and the duration requirement prevents detection of non-sustained episodes. Determination of intracardiac ventricular rate presents unique challenges—it requires appropriate sensing of QRS complexes of large amplitude, avoidance of detection of the subsequent mid-sized T wave (which would result in double counting of a single contraction), while maintaining adequate sensitivity to detect small-amplitude fibrillatory electrograms.15 These challenges have been overcome by means of a dynamic gain or sensitivity threshold, which alters the amplitude of the intracardiac signal the device can detect throughout the cardiac cycle, permitting QRS detection, avoidance of T-wave counting, and a high sensitivity for fibrillation (figure 2).15,16

Because a patient’s tolerance of ventricular tachycardia is often a function of the heart rate, devices offer several detection zones, with independently programmable treatments for each zone.17 The fastest tachycardias (such as ventricular fibrillation) can thus be treated more aggressively with immediate shock while slower ventricular tachycardias can be treated with painless overdrive pacing, often eliminating the need for a shock. Different types of treatment can be applied to ventricular tachycardias of different rates, by further dividing the ventricular tachycardia zone into slow and fast (figure 3).17,18

Supraventricular rhythms with a rapid ventricular response (including sinus tachycardia, atrial flutter, atrial fibrillation, and other supraventricular tachycardias) can erroneously be detected as tachycardia or fibrillation, resulting in inappropriate treatment, including shocks. This effect was reported in up to 30% of patients with defibrillators once electrograms became available, permitting arrhythmia diagnosis. This led to the addition of detection enhancements to the ventricular tachycardia detection zones to improve specificity. When programmed on, detection enhancements prevent treatment delivery despite a fast heart rate in the tachycardia zone if other factors (eg, a narrow QRS complex) suggest a supraventricular mechanism. Because the overlap in heart rate between ventricular and supraventricular arrhythmias occurs mainly in the slower tachycardia zones, and since the very fast rhythms detected in the ventricular-fibrillation zone (usually >200/min) are more likely to be unstable and need immediate treatment, detection enhancements typically affect only the ventricular-tachycardia zone (figure 3).

Single-chamber ICDs use enhancements based on rhythm patterns or QRS morphology to differentiate between ventricular and supraventricular arrhythmias. Sudden onset of the arrhythmia helps to differentiate ventricular tachycardia from sinus tachycardia, which has a gradual onset.19,20 Stability of the arrhythmia cycle length (figure 4) helps to differentiate ventricular tachycardia from atrial fibrillation, which is usually irregular.19,20 Morphology discrimination compares the intracardiac electrogram of the tachyarrhythmia QRS
complex with the patient’s QRS morphology during
normal sinus rhythm; similarity favours a rapid
supraventricular tachycardia, whereas dissimilarity
favours ventricular tachycardia. The intracardiac QRS
width has also been used to distinguish ventricular from
supraventricular tachycardias; a wide complex
tachycardia is probably ventricular.

Dual-chamber detection algorithms take advantage
of information acquired simultaneously from the atrial
and the ventricular leads.12 Although algorithms vary
from device to device most share the following
properties: atrioventricular dissociation is diagnostic of
ventricular tachycardia when the ventricular rate exceeds
the atrial rate; atrioventricular association favours a
supra-ventricular rhythm; and an irregular ventricular
rhythm can only be due to atrial fibrillation when
fibrillation is diagnosed in the atria. Detection
enhancements have improved rhythm discrimination
and decreased inappropriate treatments by correctly
diagnosing 60–95% of supraventricular arrhythmia
episodes, without missing ventricular tachycardia
episodes.12,19–21

Therapies

Ventricular arrhythmias can be terminated by a
defibrillator in three ways: by antitachycardia (overdrive)
pacing by cardioversion (synchronised shock), or by
defibrillation (non-synchronised shocks). The therapy
delivered is independently programmable based on
device rhythm determination. Tachyarrhythmias in the
ventricular-fibrillation range are treated by immediate
defibrillation, whereas ventricular tachycardias,
particularly the slower ones, are often treated by
sequences of overdrive pacing, low-energy cardioversion
when overdrive pacing fails, or defibrillation should the
rhythm deteriorate or prove refractory to less aggressive
measures.

Defibrillation is the mainstay of therapy for ventricular
fibrillation or rapid ventricular tachycardia. The efficacy
of defibrillation in ventricular fibrillation is higher than
98%. Between four and eight successive shocks are
available and maximum shock energies range between
25 J and 42 J in current devices, which suffices to
defibrillate most patients by the use of an endocardial
approach with biphasic waveforms and modern leads.
The mean energy needed for successful defibrillation is
about 10 J. To ensure high success rates, the system has
to be tested thoroughly during implantation, with
prompt reproducible conversion of induced fibrillation
with an energy that is at least 10 J lower than the
maximum device output.12,23 If defibrillation with a 10 J
safety margin cannot be achieved, changes in lead
configuration, repositioning of electrodes, or addition of
defibrillating elements will be needed.14 However, with
biphasic waveforms, such changes are infrequently
required.17

Low energy cardioversion is often used for termination
of ventricular tachycardias that do not respond to
overdrive pacing. Many monomorphic tachycardias can
be ended with shocks of 1 J or less.17 Low-energy
cardioversion should always be backed up by successive
high-energy shocks, since arrhythmias sometimes
accelerate after low-energy shocks.17

Antitachycardia pacing consists of short bursts of
pacing impulses at rates 10–20% greater than the
tachycardia, and can terminate the arrhythmia in
60–90% of episodes eliminating the need for shocks.17,25
In about 10% of patients antitachycardia pacing may
accelerate arrhythmia,17 necessitating more aggressive
treatment. Overall, this is a very effective and well
tolerated mode of treatment in patients with recurrent
stable or semistable monomorphic ventricular
tachycardia.

Antibradycardia pacing

Concomitant pacemakers were necessary in almost 20%
of patients with first-generation ICDs, which lacked
antibradycardia pacing capability. With today’s
implantable defibrillators with full feature pacing a
concomitant pacemaker is no longer needed.26 Between
30 and 40% of defibrillator recipients are estimated to
need dual-chamber pacing, and an additional few, who
are in chronic slow atrial fibrillation, may need VVIR
functions.11

Figure 3: Defibrillator detection zones

Arrhythmia diagnosis is based predominantly on the heart rate. Heart rate is never allowed into the bradycardia zone, as pacing will occur at the
programmed lower rate limit, up to the programmed pacing upper rate limit. Heart rates in the ventricular tachycardia zone can be treated with
antitachycardia pacing, lower energy shock, or high energy shock, whereas arrhythmias fast enough to be detected in the ventricular fibillation zone
typically are treated immediately with high energy defibrillation. When programmed on, detection enhancements to discriminate slow tachycardia from
tachycardia (such as the stabilility criterion) are only applied to arrhythmias in the tachycardia zone.
Figure 4: Ventricular tachycardia detection and stability enhancement

Upper: Detection of an episode of tachycardia. Dashed lines show tachycardia detection interval (TDI) and fibrillation detection interval (FDI). Intervals are labelled as VS in the normal heart rate zone and TS, when they are shorter than the TDI. The third electrogram occurs at a cycle length of 300 ms, which is less (faster) than the programmed TDI of 400 ms, incrementing the counter to 1. At A, the counter is reset to zero by a sensed interval of 600 ms, which is longer than the TDI. At B, tachycardia is detected, since the eight consecutive intervals that are faster than the TDI mean that the counter reaches the programmed number of intervals to detect tachycardia (NID). Depending upon the type of treatment programmed, antitachycardia pacing or shock delivery would begin at this point. Lower: The stability criterion to prevent inappropriate detection of tachycardia for fast atrial fibrillation. Tachycardia counting begins with the first fast interval (300 ms). At point A the counter is reset to zero since the cycle length of 375 ms, although less (faster) than the TDI, is more than 60 ms greater.
Diagnostics and storage of information

All modern defibrillators store information that can be retrieved non-invasively by telemetry. Stored episode data are important to confirm the appropriateness of treatments delivered and to guide reprogramming. Stored electrograms will tell the clinician whether shocks were delivered for supraventricular arrhythmias (including atrial fibrillation and sinus tachycardia) inappropriately detected as ventricular tachycardia or because of system malfunction (particularly lead failure). Moreover, since many episodes of ventricular tachycardia are symptomless because they are ended painlessly by overdrive pacing, these data could be the only source of information about their occurrence.

Current defibrillators store electrograms of arrhythmias before and after treatment plus masses of additional information—eg, hundreds (sometimes thousands) of intervals preceding arrhythmia episodes, a heart rate histogram, and a event log for atrial arrhythmia and non-sustained arrhythmia episodes. In order to record morphology information from within the heart, far-field electrograms recorded between the widely spaced right ventricular coil and the pulse generator are used. By contrast, near-field electrograms, recorded between the lead tip and the adjacent ring or coil, provide little morphology information but are used for heart-rate determination (figure 5).

Information is also available on lead impedance and battery status, and remaining device longevity. Some modern ICDs automatically check batteries and leads every day. If a problem is encountered, the device generates an audible tone to alert the patient to seek medical evaluation.

ICD indications

The goal of ICD therapy is to prevent premature arrhythmic death in an individual who might otherwise enjoy long-term survival, free from imminent death from coexisting cardiac or non-cardiac disease. This is either primary prevention (ie, to be used in patients at risk for but not having had a serious ventricular arrhythmia) or secondary prevention—ie, to prevent recurrence in a...
patient who has had a serious ventricular arrhythmia. This distinction is useful in determining which patients will benefit from ICDs.

**Secondary prevention**

Individuals who have a cardiac arrest due to ventricular fibrillation or ventricular tachycardia in the absence of acute myocardial infarction or other clearly reversible cause are at high risk for recurrent sudden cardiac death. Early natural-history studies in these patients showed recurrence rates of life-threatening arrhythmias ranging from 30% to 50% at 2 years’ follow-up mandating therapy in this population.7 In the Antiarrhythmics versus Implantable Defibrillators (AVID) trial, which was the first and largest randomised trial of ICDs for secondary prevention, 1016 patients with resuscitated ventricular fibrillation or symptomatic ventricular tachycardia and ejection fraction of 40% or less were assigned to defibrillator or antiarrhythmic drug treatment.26 Defibrillators were predominantly transvenous (93%). Drug treatment was with empiric amiodarone in 96% of patients; a small subset was randomised to electrophysiological or Holter-guided sotalol. At 3 years follow-up, mortality was reduced by 29% in patients treated with implantable defibrillators. A post-hoc subgroup analysis suggested that the improved survival in the ICD group was limited to patients with ejection fraction below 0.35.27

The Canadian Implantable Defibrillator Study (CIDS) compared secondary prevention by ICD with amiodarone in 659 patients with clinical characteristics similar to the AVID patients. ICDs resulted in a 20% decrease in all-cause mortality and a 33% decrease in arrhythmic mortality over 5 years, which was not significant (p=0.14 and p=0.09, respectively). The investigators noted that the 95% CI for risk reduction by ICD treatment in the CIDS Study overlapped that in the AVID Study, suggesting similar results with the difference between studies probably attributable to chance.28 A post-hoc subgroup analysis showed ICD benefit only in the highest risk quartile (composed of older patients with ejection fraction <35% or class III heart failure).29

In the Cardiac Arrest Study Hamburg (CASH), a 23% fall in all-cause mortality with ICD treatment was noted compared with a composite drug group (patients treated with amiodarone or metoprolol, pooled) in 288 cardiac arrest survivors followed for a mean of 57 months (p=0.08). Notably, the mean ejection fraction (0.46) was higher in CASH than in AVID, and about 10% of patients in CASH had no structural heart disease. Additionally, over half the CASH patients received epicardial lead systems (which require thoracotomy), resulting in a higher perioperative mortality than is found with current practice. Both factors probably led to an underestimated benefit of the main clinical side effect of device therapy.29

Taken together, these studies confirm that ICD implantation is more effective than medical treatment in survivors of sudden cardiac death and in patients with haemodynamically unstable ventricular arrhythmia. Patients with depressed ventricular function and more advanced heart failure derive the greatest benefit. The benefit of these devices over amiodarone in survivors with normal or near normal ejection fraction has not been proven, but where readily available, ICDs are widely used in this population as well.

Patients with syncope of undetermined origin with clinically relevant, haemodynamically significant, sustained ventricular tachycardia or ventricular fibrillation induced during electrophysiological study are also at high risk for recurrent clinical events and for sudden death—particularly if there is a history of myocardial infarction or if depressed ventricular function is present. This population also benefits from defibrillators.30 In the setting of substantial structural heart disease and inducible arrhythmias, syncope is a clinically important event, and ICD use in this setting can loosely be categorised as secondary prevention.

In primary prevention, accurate assessment of underlying cardiac disease is important for prediction of a symptomless individual’s risk of developing life-threatening arrhythmias, and for providing appropriate therapy. Whereas for some specific disease states established methods of risk assessment exist, for many others predictors of arrhythmic events are poorly defined and the approach remains controversial. Nonetheless, it has become increasingly clear that the underlying disease has an important role in prognosis, and will affect the decision whether or not to implant an ICD.

Patients with previous myocardial infarction, depressed ventricular function (ejection fraction <0.35%) and non-sustained ventricular tachycardia are at increased risk for ventricular tachyarrhythmias, despite the absence of arrhythmia symptoms, with a 2 year case fatality rate of about 30%. In the Multicentre Automatic Defibrillator Implant Trial (MADIT), such patients underwent electrophysiology for risk stratification. Those whose inducible sustained ventricular tachyarrhythmias were not suppressed by procainamide were randomised to an implantable defibrillator or conventional treatment, which for 80% of patients was amiodarone. At 2 years of follow-up, the ICD group had a significant 54% decrease in risk of death from any cause.31 Posthoc analysis showed greatest benefit in patients with the most significant ventricular dysfunction (ejection fraction below 0.26).31

The Multicentre Unsustained Tachycardia Trial (MUSTT) included patients after myocardial infarction with an ejection fraction of 0.40 or less and non-sustained ventricular tachycardia.32 This study was not designed as an ICD trial, but as a comparison of an electrophysiological study-guided approach with no specific treatment in high-risk symptom-free patients. An electrophysiological study was done in 2202 patients. Those who were non-inducible (65%) were followed up via a registry (figure 6). The 765 patients (35%) with inducible sustained ventricular tachycardia were randomised to no antiarrhythmic treatment (n=353) or electrophysiological-guided treatment (n=351). This second group was further randomised to one of several antiarrhythmic drugs. If the initial drug did not prevent induction of ventricular tachycardia during electrophysiological study, there was an additional randomisation between other antiarrhythmic drugs and an implantable defibrillator, until ultimately patients in this group received either a defibrillator or a effective drug (figure 6). At 5 years of follow-up, mortality from arrhythmia or cardiac arrest was 32% in patients receiving no antiarrhythmic therapy and 25% in the electrophysiological-guided group (hazard ratio 0.73, p=0.04). However, more impressive was the 9% 5-year mortality in inducible patients who received defibrillators, compared with 34% in inducible patients treated with drugs, and 32% in those not treated with antiarrhythmics (p<0.001).33

The third primary prevention trial, CABG-Patch, recruited 900 patients scheduled for elective coronary
bypass surgery with ejection fraction less than 0·36 and abnormal signal-averaged electrocardiogram, and randomised them to ICD or no treatment. Over an average 32 month follow-up there was no difference in overall or cardiac mortality. This study differed from two other primary prevention trials by enrolling patients who had no ventricular arrhythmias (spontaneous or induced) and were undergoing revascularisation at the time of enrolment. This led to lower long-term mortality than for the entire study population.

Taken together, MADIT and MUSTT confirm that patients with previous myocardial infarction, depressed ventricular function, and non-sustained ventricular tachycardia who prove inducible at electrophysiological study fare better with devices than with medical treatment. Some have recommended, therefore, ambulatory electrocardiography after myocardial infarction in patients with left ventricular dysfunction to screen for non-sustained ventricular tachycardia, with an electrophysiological study for further risk stratification offered when such tachycardia is found. For patients with inducible ventricular tachycardia, ICD implantation is recommended.

Because of their demonstrated efficacy and low implant morbidity, ICDs have been increasingly used in patients with uncommon conditions that predispose to sudden death, such as hypertrophic cardiomyopathy, familial long QT syndrome, Brugada syndrome, idiopathic ventricular fibrillation, and arrhythmogenic right ventricular dysplasia. Since in many of these conditions, young people with preserved ventricular function are affected, devices could offer years of added life. Indeed, in a multicentre series of 128 patients with hypertrophic cardiomyopathy who underwent ICD implantation for secondary prevention (34%) and primary (66%) prevention, the annual rate of appropriate device treatment (presumably reflecting termination of potentially lethal arrhythmias) was 5% in the prophylactic (primary prevention) group and 11% in the secondary prevention group.

In the primary prevention group the time from implant to first appropriate discharge was at times as long as 9 years.

The challenge in these uncommon disorders remains patient risk assessment and selection. In a young individual who survives an episode of cardiac arrest, device implantation is usually recommended. In symptomless individuals with high-risk characteristics, ICDs can also be used, given the frequent lack of effective alternatives, the guaranteed compliance, and the low morbidity with non-thoracotomy devices. Moreover, in some conditions (long QT syndrome and a subset of hypertrophic cardiomyopathy), the dual-chamber pacing function of ICDs can be of benefit beyond sudden death risk prevention. Ultimately, because these conditions are rare and because of ethical concerns about randomised studies in young patients with potentially lethal disorders, investigation of ICD efficacy will probably remain observational. The criteria and controversies surrounding the selection of high-risk symptom-free patients with one of these conditions for implantation of devices can be found elsewhere.

Although there is little disagreement about formal indications for ICD implantation among the various national guidelines, ICD use differs hugely from country to country. The annual ICD implantation rate in the USA approaches 200 per million population, whereas in Europe it ranges from about one per million in eastern Europe, to ten per million in the UK, and 30–40 per million in Germany, Scandinavia, Belgium, Austria, and Israel. When data from US studies are applied it is essential to realise that the US is unique in its high implantation rate.

Complications in ICD therapy
Multicentre trials of new implantable defibrillators, in anticipation of the US Food and Drug Administration review, have used strict criteria to define adverse events, classified as adverse observations or complications. Adverse observations are clinical events, with or without symptoms, which are correctable by reprogramming—eg, myopotential oversensing or inappropriate detection of a supraventricular tachycardia as a ventricular tachycardia. Adverse observations are not uncommon in clinical trials, showing the need for careful monitoring of patients.

Complications are adverse events that need another intervention after implantation, and can be divided into acute procedure-related complications or late complications. In one study involving 303 implants at 49 centres, complications included: atrial lead dislodgment (2·4%); ventricular lead dislodgment (1%); device failure (1·7%); pocket infection (1·3%); device migration (1%); change in lead position (0·3%); diaphragmatic stimulation (0·3%); and subclavian stenosis (0·3%). Other complications are haematoma, seroma, pneumothorax, or perforation. System infection can be the most important complication, because it may require system removal. There was infection in 1·3–3·4% of abdominal systems, though it is less frequent than in pectoral systems. Procedural mortality—the most important complication—has traditionally been defined as death within 30 days of surgery. Implant mortality is generally low; the AVID trial enrolled over 1000 patients, with a 30-day mortality of 2·4% in the defibrillator group, compared with 3·5% in the antiarrhythmic drug group (p=0·27).

Late complications are most commonly related to lead malfunction, which can include conductor fracture, breach of insulation, or tip dislodgment any of which could disrupt arrhythmia detection or treatment of delivery. The frequency of lead failure has diminished with technological advances. With today’s pectoral systems, the lead failure rate at 5 years is 12%; importantly, most can be detected before clinical manifestation by routine clinical evaluation.

Quality of life
Defibrillator implantation can make the patient confident of an improved life expectancy, anxious about receiving a shock without warning, while others may worry about failed shocks, or about altered body image, and they may avoid exercise and sexual activity because of fears that the ICD will discharge. However, most recipients are pleased with the device. In a subgroup of up to 15% of patients there is a risk of psychological distress, especially soon after the implantation and with those patients with frequent shocks at particularly high risk. Caregivers should ask about anxiety, depression, and sexual or physical inactivity, and recommend a support group or psychiatric referral for patients at risk.

The ability to drive a car is an important part of quality of life. Theoretical calculations and survey results indicate that the risk to society posed by ICD recipients driving a personal automobile is extremely low. Although rules vary between countries (and even between states within the USA), some general principles have been adopted with variation by both American and European expert panels. These include a ban on driving...
commercial vehicles and temporary prohibition of driving of any kind for 6 months after implantation in patients presenting with syncope and after ICD discharge associated with clinically significant symptoms.44,45

Many patients with ICDs avoid intense physical activity because they are fearful of the arrhythmia or the resultant shocks. The misconception that patients with ICDs (as well as pacemakers) cannot take exercise is very common. Patients with structural heart disease should be encouraged to exercise but exercise testing to measure peak heart rate with activity is a wise precaution.

ICDs and the non-specialist

Because ICD use is becoming more and more common, general cardiologists, internists, and general practitioners are sometimes faced with questions about the management of ICD patients, such as the possibility of drug interactions with defibrillators. Most recipients of implantable defibrillators are treated with a pharmacological drug of one form or another due to the high frequency of the concomitant medical conditions in this population.

Potentially life-threatening drug-device interactions are predominantly associated with membrane-active antiarrhythmic agents (Vaughan-Williams class I or class III drugs). These drugs are frequently used to treat supraventricular arrhythmias that might otherwise lead to inappropriate shocks, to suppress ventricular tachyarrhythmias and prevent excessive appropriate shocks, to slow ventricular tachyarrhythmias to mitigate their haemodynamic effects, and to increase antitachycardia pacing responsiveness. In defibrillator trials, membrane-active drug use has ranged from 12% to 31%.9,24,34 Device-drug interactions (panel 1) require assessment by a specialist electrophysiologist when such agents are initiated or their dose is increased.49

Membrane-active antiarrhythmic drugs slow the rate of ventricular tachycardia. If the rate falls below the programmed cut-off, the tachycardia will remain undetected by the device. Also detection enhancements meant to improve specificity may be affected by antiarrhythmic drugs. Sodium-channel-blocking drugs (especially class IC agents such as flecainide and propafenone) increase the QRS width, which may lead to rhythm misclassification by the device and inappropriate treatment, in patients whose devices use electrogram width or electrogram template enhancements. These drugs could also increase pacing thresholds, leading to loss of effective pacing.

The most potentially dangerous effect of drugs on implantable defibrillator function is an alteration in defibrillation shock efficacy. Drug-defibrillation interactions are complex, and published work is confounded by anaesthetic effects, heterogeneity in defibrillator lead type and waveform, and variability in the species tested (ie, human vs canine or porcine). However, in general, substances that impede the fast inward sodium current (such as lidocaine) tend to raise the defibrillation threshold, whereas drugs that block repolarising potassium currents (such as sotalol) lower the defibrillation threshold. Advances in defibrillator technology have mitigated these adverse pharmacological effects. Nonetheless, one agent, chronic oral amiodarone, warrants special attention since it could increase defibrillation thresholds to a clinically relevant degree, even in modern systems. Defibrillation threshold testing should be considered when drugs that can raise the threshold (especially amiodarone) are initiated in patients with defibrillators.

With the frequency of coronary artery disease and left-ventricular dysfunction among patients with defibrillators, it is important to note that drugs used to treat these conditions, such as β adrenergic blocking agents, angiotensin-converting-enzyme inhibitors, angiotensin-receptor antagonists, diuretics, digitalis, aspirin, and coumadin do not pose a special risk to patients with defibrillators as long as standard care is used in administering these agents. In addition, atrioventricular nodal slowing agents may control the tachycardia response during atrial fibrillation, maintaining a heart rate below the tachycardia detection cut-off, preventing inappropriate shocks. In patients with ventricular tachycardia storms or with frequent ICD discharges, there is also benefit from β blockers when added to other antiarrhythmic agents, probably due to the arrhythmogenic nature of excessive sympathetic activity provoked by recurrent shocks.

A patient with an ICD may present with frequent recurrent device discharges. This situation is a true emergency, since multiple shocks are very painful and frightening to the patient and family members, and could also result in proarrhythmia when delivered inappropriately. Whereas some multiple ICD shocks might be due to ventricular tachycardia storm, most episodes in which more than two shocks are delivered result from inappropriate treatment (panel 2).

Although definitive treatment for multiple shocks may require specialist referral, non-specialists may need to intervene immediately. Irrespective of the cause of the shock, the patient should be sedated, the device

---

**Panel 1: Drug-ICD Interactions**

<table>
<thead>
<tr>
<th>ICD function</th>
<th>Potential drug effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sensing and detection</td>
<td>Diminished slew could affect detection (rare)</td>
</tr>
<tr>
<td>Ventricular tachycardia rate slowed below detection cutoff rate</td>
<td></td>
</tr>
<tr>
<td>Pacing</td>
<td>Increase pacing threshold</td>
</tr>
<tr>
<td>Increase threshold at rapid pacing rates, as in antitachycardia pacing (use dependency, particularly IC agents)</td>
<td></td>
</tr>
<tr>
<td>Induce bradycardia or atrioventricular block</td>
<td></td>
</tr>
<tr>
<td>Necessitating antibradycardia pacing</td>
<td></td>
</tr>
<tr>
<td>Defibrillation</td>
<td>Proarrhythmia with increased shock frequency</td>
</tr>
<tr>
<td>Increase or decrease defibrillation threshold</td>
<td></td>
</tr>
</tbody>
</table>
inactivated, and the patient continuously monitored with an external defibrillator at hand (preferably with adhesive defibrillation pads placed far from the implanted device). Monitoring will readily determine whether shocks are appropriately delivered for ventricular tachycardia or inappropriately delivered during sinus rhythm or supraventricular arrhythmia. Pharmacological treatment can be directed toward the documented arrhythmia (eg, β-blockers for rapid atrial fibrillation; lidocaine for ventricular tachycardia, amiiodarone, and β-blockers for ventricular tachycardia storms, &c). A magnet placed over an ICD will immediately inactivate the tachycardia sensing function without affecting bradycardia pacing function. Not all ICDs resume function as soon as the magnet is removed.45,46 So patients should remain monitored until the device is confirmed to be working properly.

Following emergency measures, and while the patient is closely monitored, an electrophysiologist should be consulted.

External electromagnetic interference

External sources of electromagnetic interference could mimic cardiac signals resulting in inappropriate treatment, interfere with antibradycardia pacing, reset programmable variables, disable arrhythmia detection, or in extreme cases permanently damage device electrical circuits. All common household articles are safe for use by patients with ICDs, although there have been anecdotal reports of interference by television remote controls and electric razors (ie, carrying them or bending over them) should be avoided because they have magnets, which after long-term close contact may inactivate some devices.

Although patients with ICDs are advised to keep away from antitheft surveillance systems, these monitors are generally safe when passed through quickly. If a patient stands between the detector bars ICD function could be generally safe when passed through quickly. If a patient contact may inactivate some devices.

Magnetic resonance imaging should be avoided in patients with ICDs, despite anecdotal reports to the contrary.47 Gallstone or kidney stone lithotripsy is not contraindicated in patients with pectoral ICDs, but could damage abdominal implants. The ICD should be checked before and immediately after the procedure.

Surgical cautery could trigger shock treatment or inhibit pacing function. ICDs should be inactivated before surgery; this necessitates monitoring and access to an external defibrillator during the procedure. Other medical procedures, such as transcatheter nerve stimulation, can be done after real-time check of the device’s response to stimulation with termination of the application immediately if any interference is detected. Radiotherapy can damage circuits, and defibrillators should be shielded if they lie within the irradiation field.48

The working environment, especially if equipped with electric motors, ignition systems, or magnets, could pose some risk.49 We usually recommend on-site real-time interrogation of the ICD in a worst-case scenario when this is a concern.

Although numerous interactions with pacemakers and ICDs have been described, in practical terms there is no risk with digital or analogue handheld cellular phones that are kept at least 15 cm from the site of the device.50,51

Patients with pectoral defibrillators should not put a cell phone in a shirt pocket, and they should use the contralateral ear when phoning.

The next generation of ICDs

Implantable defibrillators have evolved from simple shock boxes into sophisticated multi-function devices. New and emerging devices have new applications unrelated to ventricular tachyarrhythmia and bradyarrhythmia management. Since many patients with defibrillators have concomitant atrial fibrillation or congestive heart failure,52 device-based approaches to these disorders have been developed.

A history of atrial fibrillation is reported in 25% of defibrillator recipients,53 and 33% of recipients without an apparent history of atrial fibrillation have paroxysmal supraventricular arrhythmias noted in device logs. Atrial fibrillation is a frequent cause of inappropriate shocks.54 Moreover, it can aggravate heart failure, and at times can be the triggering mechanism of ventricular arrhythmias.55

Therefore, a defibrillator that is also capable of prevention and termination of atrial fibrillation episodes is desirable.

Such a device has become available. The atrial and ventricular defibrillator has dual chamber pacing and uses an atrial pacing rate-stabilisation algorithm to eliminate pauses after premature atrial complexes that can be proarhythmic.56 It also includes a high atrial rate overdrive feature to prevent recurrence immediately after an atrial arrhythmia. Additionally, this device is capable of 50 Hz burst pacing in the atrium, which might be able to terminate atrial fibrillation, though this is still under investigation. By contrast with ventricular arrhythmias, atrial fibrillation is not immediately life-threatening and devices can be programmed to treat it only after sustained episodes, or shocks can be restricted to only certain hours of the day. Alternatively, a patient activator is available, so that when a patient develops symptomatic atrial fibrillation, the ICD can be activated. If the device confirms atrial fibrillation, a shock is delivered. The pain from atrial defibrillation shocks is variable, but some patients prefer it to ongoing atrial fibrillation.57 One randomised study showed a large decrease in atrial fibrillation burden, with the use of prevention and termination therapies, and over 50% of atrial tachyarrhythmia episodes were terminated with painless pacing.58 For patients with paroxysmal atrial arrhythmias, the arrhythmia management device could have advantages over traditional defibrillators.

Congestive heart failure is common among defibrillator recipients (34% in one series).59 A new technique of biventricular pacing, which uses a coronary sinus lead for left-ventricular pacing, has been introduced. In patients with conduction defects, pacing both ventricles restores ventricular synchrony and substantially improves pulmonary capillary wedge pressures, mitral regurgitation, and cardiac output acutely.60 In pilot studies and small randomised trials this technique has been shown to diminish symptoms in patients with congestive heart failure and advanced intraventricular conduction defects.61 Because of the high frequency of sudden death in patients with heart failure initially enrolled in the biventricular pacemaker studies, implantable defibrillators with biventricular pacing are being assessed.62 7–12% of ICD recipients have been estimated to benefit from biventricular pacing.63 Although this approach is promising, its long-term effects remain to be established.

Advances in ICD technology have greatly diminished implant morbidity, and have led to widespread patient...
and physician acceptance of the treatment. Future devices should offer increased longevity, diminished size, and possibly treatment for atrial arrhythmias and for congestive heart failure.

References


24. Luria D, Chugh S, Lexvold N, Hammill S, Friedman P.


