
The Implantable Cardioverter Defibrillator: Technology, Indications, And Impact On Cardiovascular Survival

**Atul Bhatia, MD, Ryan Cooley, MD,
Marcie Berger, MD, Zalmen Blanck, MD,
Anwer Dhala, MD, Jasbir Sra, MD,
Kathleen Axtell-McBride, RN,
Cheryl VanderVort, RN, and Masood Akhtar, MD**

Abstract: Since the introduction of the implantable cardioverter defibrillator (ICD) for the management of patients with high risk of arrhythmic SCD, there has been increasing use of this device. Its basic promise to effectively terminate ventricular tachycardia (VT)–ventricular fibrillation (VF) has been repeatedly met. In several randomized trials, the ICD has been shown to be superior to conventional anti-arrhythmic therapy, both in patients with documented VT-VF (secondary prevention) and those with high risk such as left ventricular ejection fraction and no prior sustained VT-VF (primary prevention). In both groups, the ICD showed overall and cardiac mortality reduction.

The device now can more accurately detect VT-VF and differentiate these from other arrhythmias through a series of algorithms and direct-chamber sensing. Therapy options include painless antitachycardia pacing, low-energy cardioversion, and high-energy defibrillation. The technique implant is now simple as a pacemaker with one lead attached to an active (hot) can functioning as the other electrode.

Among other improvements is its weight, volume, multiprogrammability, and storage of information,

dual-chamber pacing and sensing, dual-chamber defibrillation, and addition of biventricular pacing for cardiac synchronization.

It is anticipated that further improvement in ICD technology will take place and the list of indications will grow. (Curr Probl Cardiol 2004;29:293-356.)

Ventricular tachycardia (VT)–ventricular fibrillation (VF) have posed a great challenge for all clinicians and other individuals involved in the care of patients with these arrhythmias. The magnitude of morbidity, mortality, and economic impact related to VT-VF makes it an important public health issue.¹⁻¹⁰

By some estimates VT-VF are responsible for roughly half of the deaths secondary to cardiac disease. In many cases, the deaths are premature and instantaneous, with little chance of intervention. In terms of sheer numbers, there are at least 250,000 annual arrhythmic deaths in the United States alone. These deaths are generally covered under the category of sudden cardiac death (SCD), which whenever recorded from the onset, is usually the result of VT-VF.

There is a realization, resulting from a vast experience of out-of-hospital and in-hospital defibrillation (DF) experience, that DC shock invariably terminates these arrhythmias when applied instantaneously. Delay in delivery of DF has disastrous consequences to the point that a 10-minute delay results in a nearly 100% mortality or permanent brain damage (M.A., personal observation, 2003). The rapidity of problem recognition and immediate delivery of therapy, therefore, are of the essence if improvement is to be expected.

A variety of approaches has been used during the last century, particularly the last two decades, to impact arrhythmia mortality including the emphasis on underlying causes such as coronary artery disease (CAD).^{11,12} Because the outcome of out-of-hospital resuscitation had a limited success for most of the population at risk, prophylactic intervention is often seen as a more practical solution. One such approach has been the use of anti-ischemic drugs such as beta-blockers, as well.^{12,13} Because ambient ventricular ectopy has often been associated with cardiac arrest, anti-arrhythmic drug prophylaxis in the postmyocardial infarction population was also tested.^{14,15} Adverse outcome noted in CAST and CAST II with Class Ic anti-arrhythmic agents compared to placebo shifted the focus to alternate therapy. These included Class III anti-arrhythmic agents and renewed interest in beta-blocker therapy.^{13,16,17}

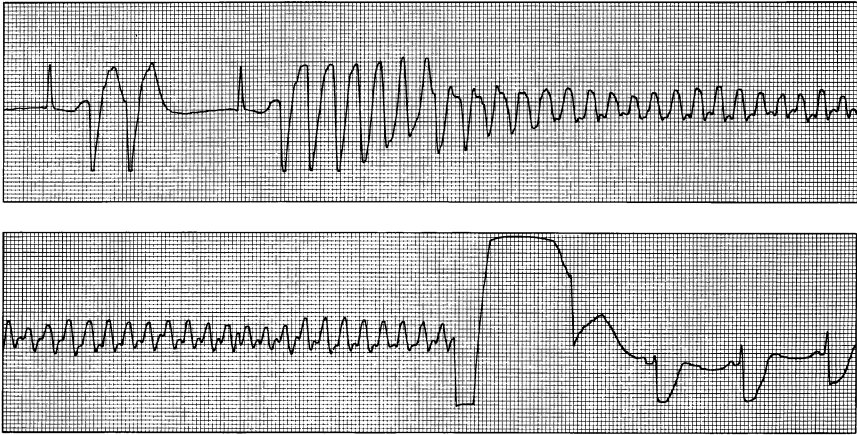
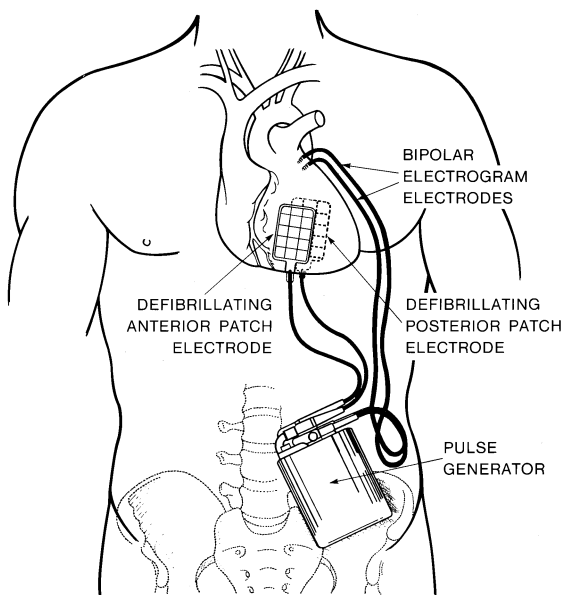


FIG 1. Surface ECG of a functioning ICD system in response to VT. The onset of spontaneous rapid polymorphic VT is depicted. The device detects and terminates the arrhythmia to patient surprise because the VT had not produced any symptoms yet. Reproduced with permission from Tchou PJ, Kadri N, Anderson J, Caceres JA, Jazayeri M, Akhtar M. Automatic implantable cardioverter defibrillators and survival of patients with left ventricular dysfunction and malignant ventricular arrhythmias. *Ann Intern Med* 1988;109:529-34.

B. J. Gersh: I agree that the results of out-of-hospital resuscitation are somewhat discouraging, but a recent study from Olmstead County, Minnesota, demonstrated that 40% of patients undergoing cardiopulmonary resuscitation for witness ventricular fibrillation left the hospital neurologically intact, and late survival was excellent. This shows what can be done in a relatively small community among whom a high proportion are CPR-trained (Bunch TJ, White RD, Gersh BJ. Long-term outcomes of out of hospital cardiac arrests after successful early defibrillation. *N Engl J Med* 2003;348:2626-33).

Concurrently, however, implantable cardioverter defibrillator (ICD) treatment for VT-VF (Fig 1) was getting more popular, primarily due to unreliability of anti-arrhythmic agents for prevention of SCD.^{18,19} Nonetheless, due to the lack of any randomized data comparing anti-arrhythmics to defibrillator efficacy there still remained a lot of skepticism. A central point of contention was whether all of these therapies alone or in combination had any significant positive impact on overall mortality. If the high-risk population already had limited survival, expensive therapy such as ICD would ultimately play a limited role. There was also a growing sense that due to a variety of factors, the incidence of cardiac and



EPICARDIAL LEAD SYSTEM

FIG 2. Epicardial lead system. The figure is a schematic display of epicardial leads used in early experience of ICD implantation. The 2 patches (high voltage shocking leads) placed anteriorly and posteriorly (dotted) are shown. Separate leads placed in a healthy part of the muscle were used for bipolar sensing. All 4 leads are then tunneled to an abdominal or subcostal area and connected to the ICD generator; this position of the ICD was necessary due to the size of the generator. Although this combination of patches and lead are seldom used now, many patients still have functioning epicardial leads that have gone through multiple ICD generator changes. Patients undergoing thoracic or cardiac surgery who have had such devices previously placed should have proper questioning about it and a PA and lateral chest radiograph must be obtained.

sudden death was declining and additional survival benefits from ICD and other anti-arrhythmic therapy may not materialize. Conversely, however it has been argued that if SCD accounts for half of cardiovascular deaths and that the latter being the number one cause of mortality in the United States, reduction in the overall death rate from heart disease should be demonstrable if SCD was eliminated or markedly reduced. From the randomized trials done so far, testing ICD versus non-ICD therapy, it appears that device therapy is superior to alternative forms.²⁰⁻²⁶ With evolution in the understanding of cardiac disease, causes of sudden death and the most effective treatment for VT-VF related mortality, it is anticipated that ICD therapy will become an essential component of cardiovascular care. It seems timely, therefore, to reacquaint the readers with one of the most explosive implantable device developments in

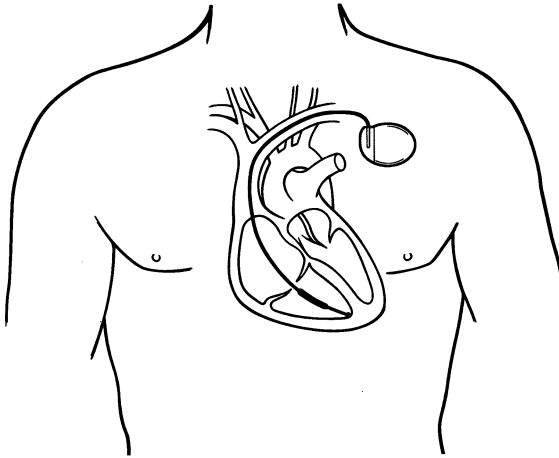


FIG 3. Endocardial lead with pectoral ICD implant. Schematic showing a single lead with a coil proximal and sensing electrode (tip) connected to a pectorally placed ICD generator. The shock vector is between the coil (in RV proximal electrode) and the casing of the generator functioning as the other electrode. The electrical field so generated is usually sufficient to defibrillate the heart.

medicine. The discussion on the topic will be presented under several separate titles for clarity, and also to allow the reader to choose an area in which he or she is most interested.



B. J. Gersh: I find the use of the word “explosive” in this context quite evocative. Undisputed, however, is that the pioneering work of Dr Michael Mirowski in the late 1960s has resulted in a therapeutic revolution.



Historical Perspective

Since the time of external DC cardioversion (CV)-DF, it has been clear that with an electrical shock delivered promptly with adequate energy, VT-VF can be terminated and a more hemodynamically stable rhythm restored.²⁷ Dr Michael Mirowski, born in Warsaw, Poland, experienced the death of a friend from VT-VF in 1966. The idea of an implantable version of the defibrillator was born and Dr Mirowski spent the rest of his professional career trying to somehow bring the idea to reality.²⁸ In the early 1970s, there was considerable doubt among his peers regarding the practicability of such an endeavor.²⁹ Implantation of a large object, surgically implanted and capable of delivering a shock that visibly jolted

TABLE 1. General characteristics of ICD

ICD Model Manufacturer*	Volume (cm³)	Weight (g)	Dimensions H×W×D (cm)
Guidant			
Vitality DS	30	82	5.9 × 6.5 × 1.1
Vitality AVT	30	82	
Contak Renewal 3	37	89	6.5 × 5.5 × 1.2
Medtronic			
Marquis, DR	36	75	
Gem III, DR, AT	39.5	78	
InSynch II MarquisI	32	77	
St. Jude			
Epic ⁺ DR	33	70	6.4 × 5.0 × 1.3

*All models listed here have dual chamber (RA-RV) pacemaker. ICD, Implantable cardioverter defibrillator; CM, centimeter; G, grams; NIEP, noninvasive EP studies.

TABLE 2. Therapy choices available with ICD

ICD Model*	Therapy Options			Other Features	
	ATP	CV	Defib	Dual Chamber Defib or Biventricular Pacing	Bradycardia Pacing, Modes
Guidant					
Vitality DS	Yes	Yes	Yes	No	Yes, all modes
Vitality Avt dual chamber defibrillator	Yes	Yes	Yes	Dual chamber defibrillator	Yes, all modes
Contak renewal 2	Yes	Yes	Yes	Biventricular pacing	Yes, all modes
Medtronic					
Marquis, DR	Yes	Yes	Yes	No	Yes, all modes
Gem III, DR, AT	Yes	Yes	Yes	Dual chamber defibrillator	Yes, all modes
In Synch II, Marquis, Biventricular	Yes	Yes	Yes	Biventricular pacing	Yes, all modes
St. Jude					
Epic ⁺ DR	Yes	Yes	Yes	No	Yes, all modes

*All models listed have dual chamber (RA-RV) pacemaker.

the body seemed barbaric to some.²⁹ Dr Mirowski's commitment to this project took impetus when a collaboration occurred between Intec, a subsidiary of Medrad, Inc and Dr Mirowski's team. The initial prototype

General Features			
Header Configuration	NIEP	Local Electrocardiogram	Longevity Range (y) (full pacing) DDDR mode
1S5-1, DF-1	Yes	Yes	5.3–6.0
1S-1, DF-1	Yes	Yes	5.5–6.5
LV-1, 1S-1, DF-1	Yes	Yes	Standard
1S-1, 3 DF-1	Yes	Yes	4.0–6.2
1S-1, 2 DF-1	Yes	Yes	3.3–4.5
1S-1, 2 DF-1	Yes	Yes	Standard
1S-1, DF-1	Yes	Yes	4.5

Other Features				
Tachycardia Zones	Program Delivered Energy Range (joules)	Waveform Programmable	Polarity	Active Can
Three	0.1-31	Mono, biphasic	Programmable	Yes
Three	0.1-31	Mono, biphasic	Programmable	Yes
Three	0.1-31	Mono, biphasic	Programmable	Yes
Three	0.2-30	Mono, biphasic	Programmable	Yes
Three	0.2-30	Mono, biphasic	Programmable	Yes
Three	0.2-30	Mono, biphasic	Programmable	Yes
Three	1.0-31	Mono, biphasic	Programmable	Yes

device was implanted in a canine and its functionality demonstrated in a video where the onset of VF, collapse of the animal, device shock, and recovery of the animal was displayed.³⁰

The first human implant occurred in 1980 and FDA approval of the

device took place in 1985.³¹ At the time of approval, the Intec device weighed close to 300 g and had a volume of around 200 cm³. It required thoracotomy to implant, which initially used a patch and a coil to patch vector. The patch was placed over the left ventricle and positioned laterally to encompass most of the ventricular mass within the electrical field generated by the shock. The generator was placed subcostally to accommodate its size. Soon after that time, Intec was acquired by Cardiac Pacemakers Incorporated, a division of Eli Lilly. Further developments included the use of two patches (Fig 2) and array of leads and decrease in size, tiered therapy, programmability of various aspects of tiered therapy, pectoral implants and shock vectors (Fig 3), sensing algorithm, and battery life, etc.

At the same time, two other manufacturers, Medtronic and Ventritex, developed their own ICD with some different features but essentially a similar purpose, ie, sensing and CV-DF of VT-VF. From the first device introduced by Medtronic, the competition has been quite fierce. A plethora of devices and lead innovation has taken place. The most visible of these, of course, has been the size of the generator. The actual dimensions and some common features of the most current devices are listed in Tables I and II for the readers to get some comparative idea. It is anticipated, however, that a further miniaturization of the ICD generator will take place. It is important to realize however that a variety of ICD models will continue to exist, some providing simpler and others more complex functions.

In addition to the weight and volume, the overall shape and thickness of the generator and implant location (Fig 3) has also changed over time. Many of these changes are in response to perceived or actual improvement in patient comfort for an implantable device in all age groups and sizes.

As will be outlined later, many other aspects of device technology have undergone improvements, which include programmable therapy options, VT-VF sensing algorithms, stored electrograms, and a variety of other data storage that can be retrieved later (Fig 4-6) (Tables I and II).

B. J. Gersh: The evolution of ICD therapy is being characterized by rapid and constant technological improvement, leading to a consistent increase in the functionality of the device. Probably the major innovations have been the ability to implant this transvenously and the addition of back-up pacing.

A**Episode Termination****Parameter Settings**

VF	On	350 ms (171 bpm)
FVT	Off	
VT	On	390 ms (154 bpm)

	NID Initial	NID Redetect
VF	18 / 24	6 / 8
VT	16	4

Dual Chamber SVT Criteria

AFib / AFlutter	On
Sinus Tach	On
Other 1:1 SVTs	Off
SVT Limit	320 ms

Sensitivity

A. Sensitivity	0.3 mV
V. Sensitivity	0.3 mV

	EGM 1	EGM 2
EGM Source	Atip / Aring	RVtip / RVcoil
EGM Range	+/- 8 mV	+/- 8 mV

Ventricular SVT Criteria

VT Stability	Off
--------------	-----

B**Episode Summary**

Type :	VF (+ SVT)
A. Median Cycle :	160 ms
V. Median Cycle :	340 ms
V. Average Cycle :	340 ms
Last Therapy :	VF Rx 1 - Defib. Successful
VT / VF Duration :	6 sec
Note :	Device is in Mode Switch prior to detection.

**SVT Criteria Triggered
Prior to VT / VF Detection**

AFib / AFlutter	
SVT Duration	6 sec.

Therapy Sequence

VF Rx 1 Defib	Energy	0.0 - 20.0 J
	Charge Time	3.48 sec
	Waveform	Biphasic
	Pathway	AX > B
	Delivered Energy	19.5 J
	Impedance	45 ohms

FIG 4. This is a sample of some of the features that can be programmed and retrieved noninvasively to assess the performance of the system. In this patient with multiple episodes of VT and who also has atrial fibrillation, this device (Medtronic 7272) shows some of the parameter settings (panel **A**). Panel **B** displays what actually happened with the episode. Within 6 seconds of arrhythmic duration VT was terminated with a 19.5 J shock. The impedance, polarity, and charge time, etc. can all be evaluated. A, Atrial; EGM, electrogram; FVT, fast ventricular tachycardia; NID, number of intervals detected; SVT, supraventricular tachycardia; V, ventricular; VF, ventricular fibrillation; VT, ventricular tachycardia. Mode Switch: conversion to VVI mode when atrium is in atrial fibrillation.

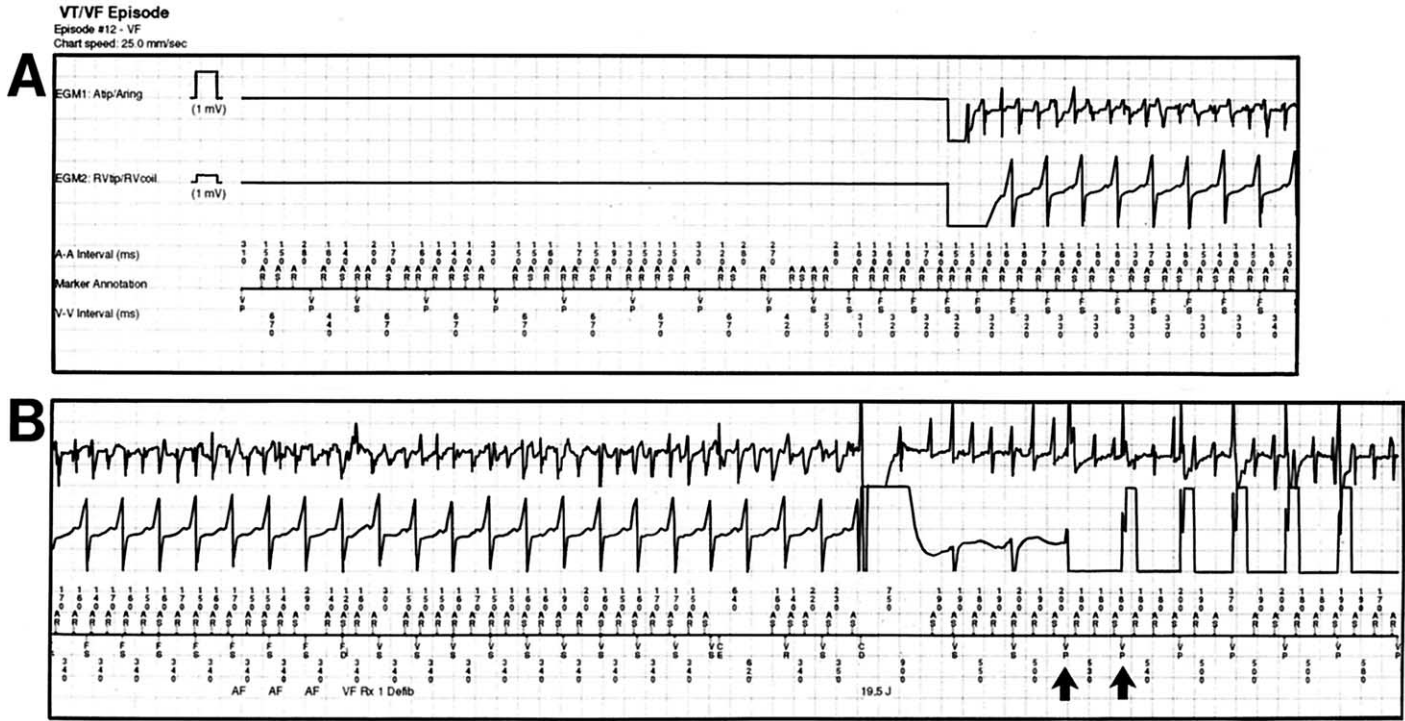


FIG 6. Episode 12 is shown in this example. The top 2 tracings are atrial and ventricular recording, respectively. Marker for size standardization is at the beginning. This tracing shows event markers with various explanations and intervals measured in msec. AR, AS has the same connotation ventricular markers initially shows VT or VF as sensing depending on the programmed rate criteria. The arrhythmia is VF (F/S) and initiated, and the capacitor starts charging at CE. With the capacitor fully charged, redetection takes place. If the same arrhythmia is ongoing, a 19.5 J shock is delivered (CD). VT terminates and spontaneous conducted complexes are seen, continue for two complexes, and then ventricular pacing is initiated. It should be noted that during ventricular pacing (VP, arrows), signal amplification can be seen in the atrial electrograms. Shock delivery interrupts the tracing momentarily due to saturation artifact.

Essentials of Defibrillation Therapy

Restoration of normal rhythm with DC shock from transthoracic electrodes is a routine medical practice.²⁷ The current required to terminate VF is approximately 200 joules and has been shown during cardiac resuscitation efforts.³² Defibrillation can be accomplished with lower energy by positioning the electrodes closer to the heart. The utilization of lower energy for DF has also gone through significant evolution. This includes use of transvenous coils, a transvenous coil and patch combination, and two patches with or without additional electrodes such as a sub-Q array of leads. The current practice is predominately to use a combination of transvenous electrodes while the casing of the generator functions as the other electrode (active or hot can) (Fig 3). With programmable shock vector combinations, one can achieve a desirable DF threshold in vast majority of the patients. In rare cases, thoracotomy may be necessary. The transvenous implantation of the lead with one or two shocking coils is done through cannulation of the cephalic, subclavian, or other veins, quite similar to permanent pacemakers. However, depending on the number and size of leads, several venous cannulations may be necessary.

After leads are placed in the appropriate location, they are connected to the generator and placed in a pectoral pocket (usually the left) and testing of sensing and DF is started. In the following paragraphs, the key components of the ICD system are outlined which, would help to understand the functioning of the ICD system.

The ICD System

Encased in titanium alloy, the generator is an engineering marvel that contains the battery, capacitors, transformers, and electronics.³³ Its basic purpose is to provide reliable sensing and DF. The battery is lithium silver vanadium oxide and provides all of the low-voltage energy functions and high-voltage energy for DF through charging circuitry and transformer. Once a tachycardia is sensed, capacitor charging is initiated. The capacitor consists of an anodal and a cathodal foil with electrolyte impregnated paper separator. The amount of charge a capacitor can hold is related to its surface area. Etched aluminum has been used to increase the surface area of the anode and di-electric layer of aluminum oxide and deposited on the anode by a process known as formation. The di-electric helps to store the charge. The traditional ICD capacitor is rolled and is 17 mm in diameter, and 2 of these are necessary to create up to 750 volts. During the charge, low voltage from the battery is converted into

high-voltage energy for a brief period and stored in the capacitor for rapid discharge. Capacitors store maximum energy ranging from 1.7 to 3.6 J/con 3. The more conventional rolled capacitor has less energy density than the newer flat cap capacitor. The type of capacitor used impacts the shape, size, and thickness of the generator and also the capability of maximum delivery.

The electronic circuitry has many programmable and nonprogrammable features designed for various sensing and therapy algorithms, which will be discussed later.

The complexity of the generator depends on the ultimate purpose for its implant. The single-chamber ICD using one or two shocking coils is now less frequently used and dual-chamber pacing and ventricular DF is on the rise. Some of the basic ICD types used in clinical practice are listed here.

A. Single-chamber ventricular DF device. The current examples are Marquis VR (Medtronic) Vitality VR (Guidant) and Epic+ (St Jude). As the most basic device, it is intended to treat VT-VF only, with a single lead placed in the right ventricle using the generator can as the other electrode (active or “hot” can, Fig 3) or have a vector utilizing right ventricle, superior vena cava, or left innominate vein. This long electrode (or coil) can be part of a single lead or can be a separate lead. The details of various leads used in conjunction with an ICD system can be obtained through the physician manuals from each manufacturer. A second lead is only necessary if the desired interelectrode distance and, hence, the DF efficacy cannot be achieved with the initial lead system. For the most part, acceptable DF and safety margin is achievable with a simple vector using right ventricular (RV) coil and active can configuration where the cathode is RV and anode is the active can (Fig 3). The polarity of the shock vector can be reversed and is a programmable feature in most current devices (Table II). In some patients, the vector configuration between the right vent apex and proximal electrode is sufficient. When a three-electrode configuration is used, it is generally RV to an active can with programmable polarity, and proximal electrode to the active can, also with programmable polarity. Since the main goal of a DF shock is to terminate VF (a statistical phenomenon), the most appropriate lead location is explored at the time of implant.

B. J. Gersh: For reasons that are not clearly understood, biphasic shock wave forms have resulted in a substantial improvement in defibrillation efficacy (Glikson M, Friedman PA. The implantable cardioverter defibrillator. *Lancet* 2001;357:1107-17).

All current devices have back-up bradycardia pacing with rate responsiveness. The main disadvantage of this combination (ie, DF and bradycardia pacing) is that the constant pacing significantly reduces the longevity of the generator. On the flip side, there are several advantages of this combination. To mention a few: (1) single rather than two devices with fewer leads, (2) cost of two separate devices versus the single, and (3) possible interaction between a separate pacemaker generator (could be from a different manufacturer) with the ICD could lead to nondelivery of the ICD shock.

B. Ventricular DF with dual chamber (right atrial [RA] and RV) pacing and sensing (Figs 2-4). The ICDs with these features also have rate responsiveness. Examples are the Marquis DR (Medtronic), Vitality DS (Guidant), Epic+ (St Jude) (Table II). The ventricular DF is similar to what was described previously. An additional atrial lead, however, is necessary.

C. Dual-chamber (RA, RV) pacing and dual chamber DF. Here the additional feature is the ability to defibrillate the atrium, and can be used for conversion of atrial fibrillation (AF). Other atrial arrhythmias can also be terminated with such a depolarizing shock. However, antitachycardia pacing (ATP) and high-frequency bursts are preferable and are also incorporated in the device. Currently, the devices for dual-chamber DF are Medtronic's Gem III DR AT and Vitality AVT from Guidant (Table II). During a depolarizing shock to terminate AF, the energy utilized is in excess of 1.0 J, and is likely to be perceived as an uncomfortable sensation. Repeated use of shock for termination of AF will remain the main limitation for these devices for use in AF. The key difference unrelated to technology of AF versus VT/VF devices is the fact that AF per se is not life threatening and the recipient of atrial DF shock is fully alert. Hence the pain and discomfort sensation must be eliminated or markedly reduced. A variety of options to deal with this reality are being addressed. They include: (1) use of different vectors for energy delivery, ie, different than intracavity electrodes to hot can or CS-RA vector; (2) decrease the voltage and increase the duration or shape of the DF shock; (3) spread of energy delivery over a larger surface area of the electrode; (4) warning shock so that the patient can decide the location (home office, etc), timing of the shock; and (5) sedation before the shock.

D. Ventricular DF with atrioventricular (AV) sequential and biventricular pacing (DF plus cardiac resynchronization). The two newer devices of this are a Medtronic InSync II, Marquis, and Guidant Contak Renewal 3 (Table II). These devices require an additional electrode via the coronary sinus to be placed in one of its branches for left ventricular (LV) pacing. If one accepts that CV mortality predominately occurs in two ways, ie, VT/VF or pump failure, concept

of a device that will improve the odds of benefiting both aspects is very attractive. Ventricular defibrillators have already proven that CV survival can be improved with an ICD in patients with CAD and a low left ventricular ejection fraction (LVEF).²¹⁻²⁶ Prolongation of life in patients with overt congestive heart failure (CHF) by ICD has been recently shown.³⁴ However, the biventricular pacing component of the device improves the quality of life in NYHA Class III and IV patients.³⁵⁻³⁷ The combination of the two technologies makes sense if the clinical trials will show a value of VT-VF devices in these patients with severe CHF. It is also becoming apparent that patients with a poor LVEF, ie, $\leq 40\%$ and/or CHF, the presence of left bundle branch block leads to further deterioration of LV as a pump due to desynchronization of LV contractions. It is intriguing to postulate that the same may happen with artificially induced left bundle branch block, ie, the RV pacing. In the event the data reveals that LV or biventricular pacing (LV pacing via coronary sinus) improves the ventricular performance, such pacing may become the preferred method for ventricular pacing. Because most patients with VT due to CAD have poor LV function, a ventricular DF device with concomitant backup biventricular pacing may become a better option at the initial implant in patients with wide QRS complexes, ie, ≥ 140 msec.

B. J. Gersh: This was the conclusion of the DAVID trial, which suggested that dual-chamber pacing at the rate of 70 beats/min increased the end point of death or hospitalization for heart failure in comparison with ventricular backup pacing (Wilkoff BL, Cook JR, Epstein AE, et al. Dual-chamber pacing or ventricular backup pacing in patients with an implantable defibrillator: the Dual Chamber and VVI Implantable Defibrillator (DAVID) Trial. JAMA 2002;288:3115-23).

Accurate identification of arrhythmia is critical for success of an ICD, and has been of great concern from the birth of this device. In the ensuing pages, the various methods used, rhythm identification, and logic behind are outlined.

Sensing And Detection By ICD

Proper functioning of an ICD is dependent on reliable sensing of the ventricular depolarizations. To accomplish this, a two-part fully integrated system with sensing amplifier and detection algorithms has been

incorporated in the devices, even though sensing and detection are two different functions.

Sensing. During sensing, the sense amplifier amplifies, filters, and rectifies incoming electrogram signals. Then it compares them with sensing threshold and produces a set of R-R intervals for the detection algorithm to process the signals.

Detection. During detection, the detection algorithm classifies and counts the R-R intervals to decide whether a sustained arrhythmia is present. The ideal detection algorithm is the one that can detect low amplitude, fluctuating VF spirals, and differentiate VT and supraventricular tachycardia (SVT). It must ignore “T” waves during sinus rhythm, nonsustained arrhythmia, AF, sinus tachycardia, and myopotentials. It also should be able to switch therapies as arrhythmias change, redetect aftershocks, grapple with signal changes after therapy, and evaluate the effectiveness of therapies. At present, however, inappropriate arrhythmia detection occurs in 16% to 53% of ICD patients^{38,39} and result in unwarranted therapies, with potential pro-arrhythmic consequences.

The first generation of ICDs had excellent sensing during VF, despite the fact that they lacked data logging and electrogram shape options. These devices used the so-called “*automatic gain control*” that allows the sensing system to adapt to changes in signal amplitude with different rhythms. Signals as low as 0.2 to 0.3 mv usually associated with fine VF can be sensed with this algorithm. With automatic gain control (Guidant), the threshold for detection of a signal is a function of the amplitude of the previously sensed signal, and the gain continuously varies, so that the amplitude of the processed signal is relatively constant. Despite being the gold standard for its obvious clinical efficacy and widespread use in devices, this feature is not without limitations. There are documented reports of failure to sense VF and “T” wave oversensing, as well as ICD-pacemaker interactions, leading to inappropriate detection and shocks.⁴⁰⁻⁴² Another form of sensing called “*automatic sensitivity tracking*” (Medtronic, St Jude), allows the system to deal with rapid change in electrogram amplitude, eg, during VF. The limitation of this system is that if filtering is optimized for VF, it may oversense “T” waves or undersense very-low-amplitude VF late in the episode. “*Automatic threshold control*” sets the sensitivity to a proportion of the amplitude of the last sensed event. The sensitivity then gradually increases until the next event is sensed. Despite the use of automatic-adjusting sense amplifiers, sensing problems are still encountered. All ICDs currently in use employ cardiac rate above the programmed limit as evidence of tachyarrhythmia presence. The sensitivity of rate criterion is excellent, but rate alone is a

nonspecific indicator for VT and may be mimicked by a variety of tachycardias including sinus tachycardia. When one of the shocking electrodes is also used for bipolar sensing (integrated bipolar), postshock decrease in signal amplitude and quality can occur and may lead to delay in redetection of VF, a problem largely circumvented in some modern-day ICDs with the use of true bipolar sensing⁴³ where none of the two electrodes are used for therapeutic shock.

With the rate cut-off as the sole criterion, VF is among the least difficult arrhythmias for the ICD to recognize. Ventricular fibrillation detection is confirmed by the device once a preset number of sensed events, ie, 8/12 intervals, etc, are shorter than the VF rate cutoff. In the era of committed devices (mostly obsolete), after satisfaction of detection algorithm, and a predetermined delay period, the high-voltage capacitors were charged and committed to shock delivery. In currently available ICDs, VF must be reconfirmed at the end of capacitor charging and before delivery of the actual shock therapy. Since VT and SVT with 1:1 P-QRS relationship are more difficult to distinguish from each other, and shock therapy for SVT is undesirable, more enhanced sensing algorithms have been incorporated both in single- and dual-chamber ICDs.

VT-SVT Discrimination

Historical approaches to differentiating supraventricular from ventricular tachycardias by ICD have been rate-based. To increase the specificity for VT, certain other options like *suddenness of onset* and *rate or interval stability* have been incorporated and are independently programmable. Most ICDs today also offer some form of electrogram morphology analysis to increase discrimination specificity. Using the previously mentioned algorithm, sinus tachycardia may be differentiated by its more gradual onset,⁴⁴ and prematurity (sudden change in cycle length)⁴⁵ of the first interval of the tachycardia exceeding the rate cut-off is of value in distinguishing VT from SVT, but several SVT forms may also fulfill such criteria. Cycle length stability can be used to differentiate AF from regular SVT and VT. The criteria mentioned previously may be further refined by combining it with a prespecified arrhythmia duration (eg, 2.5 seconds, Guidant) above the rate cutoff before confirmation of the tachycardia. Another method is the "*morphology discrimination*," whereby the morphology of the detected arrhythmia, including the number, amplitude, sequence, and polarity of waveform peaks are analyzed. This algorithm is based on the principle that any rhythm originating in the ventricle exhibits a morphology fundamentally different from one having a nonventricular

origin. It compares the morphology of the arrhythmia with a template stored during the normal sinus rhythm.

Unfortunately, in spite of all these algorithms used in combination with the rate criterion, inappropriate therapy delivery remains a problem. Ventricular tachycardias do not invariably start suddenly and may be irregular as well. The problem is further compounded in patients with fixed, functional, or rate-dependent intraventricular conduction delays, where VT may get classified as SVT. In such cases, delayed detection of VT may result in other clinical sequelae like presyncope and syncope.

B. J. Gersh: In some series, inappropriate shocks primarily due to detection of supraventricular arrhythmias and sinus tachycardia were reported in approximately 30% of patients. This is a major problem that has a detrimental impact on the quality of life.

Dual-Chamber Sensing for VT/SVT Discrimination

The addition of atrial sensing information in dual-chamber ICDs promised better performance.⁴⁵ Dual-chamber ICDs' rhythm discrimination follows a stepwise analysis of rate, stability, AV relationship, and onset. Different manufacturers have different algorithms as outlined next.

To detect monomorphic VT, most ICDs *average*, or *sample* tachycardia intervals. Monomorphic VT lends itself to detection by a specific algorithm when it is hemodynamically well-tolerated and has a large stable amplitude and a monocyclic nature. "PR Logic," the detection algorithm in Medtronic devices (Gem, Gem111, Marquis), has been shown to clinically reduce the number of inappropriate VT detection, while maintaining 100% sensitivity for sustained VA.

PR Logic uses rate, pattern regularity, and AV dissociation to discriminate SVT and true VT-VF. Once detection is met by rate-only counters, PR Logic is applied. If it is able to identify an ongoing SVT, therapy is withheld. If at any time during the episode, PR Logic is unable to define an SVT, VT-VF therapy is delivered. PR Logic is programmable through four parameters, including SVT/VT limit and three "on-off switches" for Afib/Flutter, sinus tachycardia (ST), other SVTs.

Pattern recognition in PR Logic is accomplished by calculating the number and position of atrial events relative to ventricular events. PR Logic looks at two consecutive V-V intervals and assigns a pattern code to each beat and compares it with the predefined SVT pattern. Each V-V interval is divided into 4 zones: two junctional zones, one retrograde, and

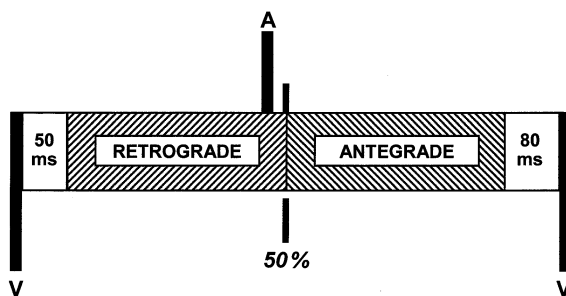


FIG 7. PR Logic divides each V-V interval into 4 zones: 2 junctional zones, 1 retrograde zone, and 1 antegrade zone. The junctional zones are defined as 50 ms post-ventricular event and 80 ms pre-ventricular event. The antegrade and retrograde zones are defined based on a percentage of each V-V interval. PR Logic classifies rhythms with a 1:1 A-V relationship as sinus tachycardia when there is a single antegrade atrial event for every ventricular event.

one antegrade zone. The antegrade and retrograde zones are defined based on a percentage of each V-V interval. A nominal pattern template is shown (Fig 7). Refinements have been made in the PR Logic in the latest models (Gem111, Marquis), including programmable 1:1VT-ST boundary, enhanced far-field R-wave criterion, and transition counter. VT-ST boundary is specifically designed to address SVTs with long PR intervals. This enhancement allows for the PR Logic's ability to withhold therapy for SVTs with 1:1 conduction and long PR interval. 1:1 VT:ST boundary describes the antegrade zone of the pattern template, and is defined as the AV interval divided by the V-V interval, and is expressed as a percentage and the nominal setting is 50%.

Reprogramming the VT-ST boundary must be done with extreme caution. In patients with 1:1 VA conduction, increasing the antegrade zone has the potential of decreasing sensitivity for VT with long and consistent 1:1 retrograde conduction. Enhanced far field R waves, requires 4/12 ventricular intervals containing evidence of far-field R waves before classifying it as sinus tachycardia. The transition counter in PR Logic holds the definition of an SVT in progress even though the SVT rules are no longer met. This allows for reclassification of arrhythmia if there is a transition between different types of SVTs. If PR Logic is unable to identify a different or ongoing SVT, VT detection is satisfied and therapy is delivered. With incorporation of the above-enhanced detection algorithm, the incremental specificity is ~85%, positive predictive value of 95%, while maintaining 100% sensitivity for sustained VT and VF in patients with SVT and long PR interval.

In ICDs with atrial sensing leads, the rhythm discrimination can also be

divided into two broad based classes depending on the relationship between atrial and ventricular activity, ie, associated or dissociated:

1. 1:1 AV association, eg, sinus tachycardia, VT with 1:1 VA conduction, atrial flutter with rapid ventricular response.
2. AV dissociation, eg, nonventricular arrhythmias occurring simultaneously with VT.

AV Rate Branch

In St Jude devices (Atlas, Photon Epic series), AV rate branch takes the form of a three-way decision tree. It is based on the concept that an atrial rate faster than or equivalent to a concurrent rapid ventricular rate generally suggests an arrhythmia other than VT or VF. When an arrhythmia is detected in the rate overlap zone (the region between the patients slowest VT and his or her maximum sinus rate), the system measures and compares the median atrial and ventricular rates within the preceding intervals. Of note, this becomes the first step in the discrimination process, other criteria like onset, stability, and morphology discrimination are not used until the AV relationship is established. Three possible scenarios for rate comparison are shown (Fig 8). VT ($V > A$) is treated immediately. An episode that falls in $V < A$ category is suggestive of an arrhythmia like atrial flutter or fibrillation, and additional discrimination features are used to distinguish VT from the former. Next, the stability criterion (recognizing AF), along with AV association algorithm assists in differentiating atrial flutter from VT. However, AV association is only applicable if stability satisfies the VT criterion.

In the third scenario, VT with 1:1 VA conduction ($V = A$) may be confused with sinus tachycardia. If the device does not use additional discriminating algorithms, SVT is diagnosed and therapy withheld. Therefore, once any episode falls into the $V = A$ branch, it automatically triggers onset and morphology discrimination into action. Also, AV relationship can be examined during AV association or if dissociated, VT is declared.

In the Prizm (Guidant) family of ICDs, two enhancements are added: ie, AF rate threshold and VF rate $>$ AF rate, to the pre-existing single-chamber algorithms. The AF rate threshold enhancement is used in conjunction with the VF rate *stability* and *onset* algorithms as therapy inhibitors. The VF rate $>$ AF rate enhancement is an inhibitor override that uses the average atrial rate to overrule all therapy inhibitors including the ventricular rate onset or stability algorithm. This algorithm again

A-V RATE BRANCH

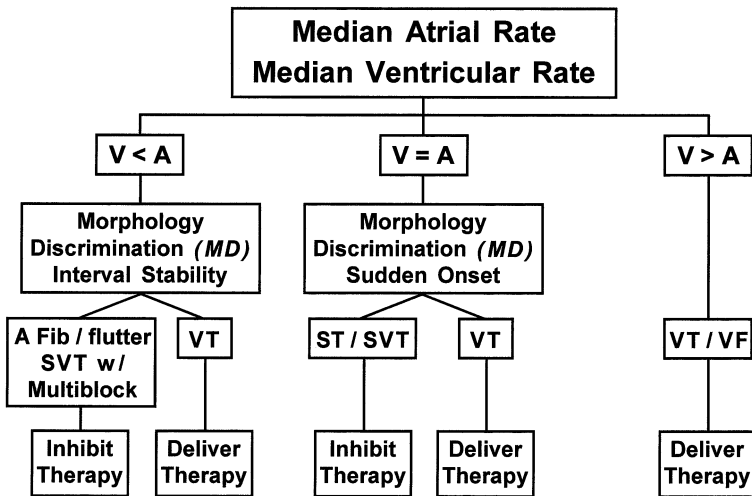


FIG 8. VT/SVT discrimination using AV relationship. The Schema explains the initial step ($V > A$ is VT/VF) a VT therapy is delivered. $A > V$ supports various forms of SVT and further discrimination primarily to exclude A fib, A Flutter, or the similar arrhythmias will take place. When $V = A$ sinus tachycardia (ST) and AV junctional SVT must be excluded. In all situations, however, if any doubt exists VT therapy overrides all other options. A, Atrial; ST, sinus tachycardia; SVT, supraventricular tachycardia; V, ventricular; VT, ventricular tachycardia.

provides for maximum sensitivity for VT-VF and is more sensitive than the ventricular rate alone algorithm.

The dual-chamber algorithms described uses different analytical methods for assessment of atrial and ventricular events that occur in patients with cardiac disease who have tachyarrhythmias. Because AF and atrial tachycardias are also common in such patient populations, these enhanced detection algorithms help to reduce inappropriate therapies. The addition of atrial sensing information in dual-chamber ICDs promised better performance. However, even these have so far not lived up to their theoretic potential for eliminating inappropriate therapy, with ventricular oversensing adding to the problem.^{46,47} In the future, newer algorithms aimed at enhancing the specificity may include atrial morphology discrimination. Better computation of signals aimed at better sensing of intracardiac events will lead to smarter, albeit more complex, devices. It should be pointed out that, regardless of the complexity of detection algorithm, ICD is designed to deliver ventricular DF shock and excessive delay in therapy delivery as the DF threshold is rising, shock delivery rather than withholding the therapy is a safer option.

M. M. Scheinman: The authors have beautifully described available techniques used for distinguishing various arrhythmia disturbances. Chronic monitoring of these patients will allow for a host of additional benefits. For example, analyses of log history will allow for appropriate estimation of AF “burden” and for assessment of rate control for patients with supraventricular arrhythmias.

The AFFIRM trial has taught us that anticoagulant therapy is required even in the group thought to have rhythm controls. The likely explanation involves periods of AF that are not appreciated by the patients (silent AF). Initial studies suggest that approximately 30% of AF episodes are truly silent.

In addition, the implanted devices allow for excellent assessment of rate control. It is well known that rapid rates produce a tachycardia-induced myopathy. Use of device therapy allows the clinicians to monitor heart rate. Control in patients who suffer from both supraventricular and ventricular arrhythmias.

Therapy Options

The modern day ICD is capable of providing both tachyarrhythmia and bradyarrhythmia treatment options.

Ventricular Tachyarrhythmia Therapy

Antitachycardia therapy can be applied in stages, so-called tiered therapy, and these are outlined below.⁴⁸⁻⁵⁰

Defibrillation and *CV* are the two primary high-voltage therapies. *Antitachycardia pacing (ATP)* is an added therapy option to terminate organized VT, whereby bursts of low-energy pacing pulses are delivered through the ventricular (pace/sense) electrodes. Although the ICD was conceived as a device meant for immediate treatment of VF, evidence that many out-of-hospital VF survivors have inducible VT, and spontaneous VTs can degenerate into VF, provided a strong rationale for development of additional therapy options, ie, to identify and terminate VT. However, in terms of preventing VT/VF related SCD, fail-safe DF remains the most important function of an ICD. For DF to be effective, a critical mass of myocardium must be depolarized to extinguish all the fibrillatory activity in the ventricles. The energy required to accomplish this is delivered between the high-energy electrodes (the generator also being an active electrode), which terminates VF and restores the non-VF rhythm.

Initially, the ICDs were the so-called “*committed devices*,” wherein,

once VF was detected, after a predetermined delay, the capacitors were charged and committed to shock delivered. The current-day ICDs are “non-committed,” wherein, once VF is detected (ie, 12/18 intervals, etc), the capacitors are charged and before shock delivery, VF is reconfirmed (6/8 intervals) and only then is the shock delivered. All through the capacitor charging period, the tachyarrhythmia is being sensed. When VF is detected, the ICD charges the high-voltage capacitors to the programmed energy. When the programmed energy is reached, the ICD attempts to synchronize the pulse to the leading edge of a ventricular sensed event. The DF pulse is timed to the first nonrefractory sensed event if possible or delivered asynchronously, if it cannot be synchronized. The time interval between onset of VF and delivery of shock is 0 to 15 seconds. The capacitor charge time usually varies at implant (0-10 sec), depending on the programmed stored energy. Charge time is the time required to charge the capacitors to the preselected voltage. The charge time lengthens as the battery voltage depletes and has a linear relationship with the programmed voltage. Short charge times <5.9 sec (Medtronic) or <5.0 sec (St Jude) are helpful clinically and may prevent syncope due to the rapid arrhythmia, by quick delivery of therapy.

M. M. Scheinman: The time from initial detection to shock delivery is extremely important in terms of whether the patient will experience syncope associated with the arrhythmia. This issue comes to the fore as in the relationship to auto driving privileges for patients fitted with defibrillators. A categorical answer to the issue of driving cannot be given since laws affecting driving vary from state to state. Nevertheless, the clinician should consult the NASPE consensus report on this issue (see the NASPE Web site). They recommend, for example, a waiting period of six months after ICD shock delivery.

The ICD therapy options have evolved from a simple nonprogrammable “shock only” device to multiprogrammable, multitiered therapy options devices. The most significant advance in this regard has been the development of ATP and low-energy CV for VT. These two antitachyarrhythmia therapy options have clearly expanded the applicability of ICDs to patients who are not ideal candidates for conventional high-energy DF therapy, eg, patients with slow VT. ICDs continue to provide fail-safe DF back-up to the preceding therapies in the event of tachycardia acceleration occurs subsequent to ATP or CV, or simply other therapies employed first to terminate VT.

ATP and CV. The development and incorporation of ATP in the ICDs followed the observations that inducible monomorphic VTs in the

electrophysiologic laboratory could often be successfully pace-terminated. The underlying mechanism in the scar-related VTs is re-entry. The presence of excitable gaps within the re-entrant circuit, allow for the successful penetration and termination of VT with critically timed paced stimuli.⁵¹ Antitachycardia pacing can be used both as a therapeutic and preventive strategy, eg, pacing to prevent pause/brady-dependent polymorphic VT-VF. Hemodynamic tolerance of the VT is a crucial prerequisite for the selection of patients for ATP. Typically, ATP is effective for VTs with cycle lengths >300 msec with a success rate of 90% to 96%.⁵² Because rate is the most important determinant of the hemodynamic outcome of VT, faster VTs are usually treated with shocks because of obvious concerns about syncope due to delay of definitive therapy. Recently, Walters et al⁵³ showed that empirically programmed ATP was effective in up to 77% of patients with fast VTs (cycle length <320 msec), with a low incidence of VT acceleration (4%). This is important information because ATP is essentially an imperceptible therapy and avoids the morbidity of painful and higher-energy shocks.

B. J. Gersh: It has been known for more than 20 years that antitachycardia pacing can terminate ventricular tachycardia, but in the minority of episodes pacing can accelerate the ventricular tachycardia rate or convert ventricular tachycardia into ventricular fibrillation (Hartzler GO. Treatment of recurrent ventricular tachycardia by patient-activated radio frequency ventricular stimulation. *Mayo Clin Proc* 1979;54:75-82). This was a major factor limiting the use of ATP in clinical practice. With defibrillation back-up, the current versions of the ICD have eliminated the problem. Obviously, the ability to terminate the majority of episodes without a shock has had a major impact on patient satisfaction.

ATP programming can be done using different stimulation protocols as follows:

- Multiple pacing impulses with fixed or adaptive coupling intervals.
- Burst pacing in which long trains of pacing impulses at a fixed cycle length are used.
- Autodecremental or ramp pacing, in which the first stimulus is delivered at percentage of the tachycardia cycle length (typical 97%) and each subsequent extrastimulus is delivered at successively shorter decrements of the first coupling interval (typically 10 msec). Relatively long initial coupling interval and gradual decrements favor capture of all the stimuli, even with relatively rapid terminal pacing rates.

- Numerous other, multicapture sequences exist, including scanning bursts, scanning ramps, and so called universal pacing. However, none are proven to be superior clinically to above-mentioned burst or auto-decremental pacing protocols.

Current ICDs are programmable to at least two or more different protocols of ATP. Overall efficacy of ATP in terminating sustained monomorphic VT in about 90% of cases, with an incidence of therapy related acceleration of <5%.

The absolute threshold for pacing versus shock therapy remains unclear and varies from patient to patient. In general, tachycardias with cycle lengths <300 msec should be strongly considered for immediate shock therapy, whereas those longer than 320 to 350 msec, can be terminated with pacing and back-up DF. Long periods of ATP should be avoided because of the clinical and hemodynamic consequences of a prolonged ongoing VT. It cannot be overemphasized that a delay in the delivery of successful therapy (shock) can also increase the DF threshold, due to ischemia generated during the ventricular arrhythmia. It should be clear that for preventing VT-VF sudden death, prompt DF receives the highest priority under any form of electrical therapy hierarchy. In summary, ATP does provide an elegant, efficient and painless means of tachycardia termination. The incorporation of ATP as part of the tiered electrical prescription has not resulted in compromise of device efficacy, size, or battery longevity.

Pacing is also clearly useful in patients in whom tachycardia occurs in the setting of clinical bradycardia, eg, long QT (idiopathic) and pause dependent torsades des pointes. With the availability of dual chamber pacing in the current ICDs, continuous pacing slightly above the patients spontaneous rates (overdrive suppression) has been effective in decreasing significantly the frequency of ventricular ectopy and nonsustained VT as well.



M. M. Scheinman: The authors have properly stressed the importance of avoidance of pauses that might trigger serious arrhythmias. This issue has been well documented for patients with the congenital Long QT syndrome. Studies have shown (Viskin S, Alla SR, Barron HV, Heller K, Saxon L, Kitzis I, et al. Mode of onset of Torsade de Pointes in congenital long QT syndrome. *J Am Coll Cardiol* 1996;28:1262-8) that most of the documented episodes of torsades in patients with the congenital Long QT syndrome are pause induced torsades short-long-short sequences, or pauses alone. It should be emphasized that ICD insertion in the patient with the congenital long QT syndrome is not a cure and every effort should be made to prevent ICD discharges, since discharges may result in recurrent bursts of torsades (ICD

Storm). Overdrive pacing has been shown to be one effective mode of therapy to prevent the pauses that might trigger torsades. (Dorostkar PC, Eldar M, Belhassen B, Scheinmann MM. Long-term follow-up of patients with long QT syndrome treated with beta blockers and continuous pacing. *Circulation* 1999;100:2431-6).

Cardioversion. Cardioversion shocks usually range between 5 and 15 J but could be lower or higher. The addition of CV as a therapy option, in the multitiered therapy algorithms, has potential advantages, including prompt, synchronized shock delivery and less battery drain. All the current ICDs are provided with an “R” wave triggered CV modality having a programmable shock energy. Several points need consideration before CV is programmed “ON” as an option. It should be used only with back-up DF and not as stand-alone therapy. The efficacy of CV in terminating very rapid VTs, ie, cycle lengths <280 msec is low, with antecedent danger of further acceleration. Low energy CV, therefore, should not be programmed into therapy zones for fast VTs. Because CV and ATP have similar efficacies, and the fact that ATP is painless and better tolerated, ATP attempts should generally precede CV attempts. It should however be noted that the response to ATP is not predictive of response to CV and vice-versa. Use of proper hierarchy with multitiered therapy devices contributes to better patient acceptability, decreases battery drain, with need for fewer shocks without compromising patient safety.

M. M. Scheinman: The authors well summarize the potential problem with low-energy shocks. It should be emphasized that low energy shocks do not obviate or lessen the chest pain felt by the patient and ICD discharge. Hence, the potential benefits are largely mitigated by need for delivery of effective therapy.

The importance of ATP has been well-emphasized by the authors for use in patients with stable somewhat slower VT. Not only does ATP frequently terminate VT in a painless fashion but also VT detection promises to teach us a good deal about efficacy of drug therapy particularly for patients with genetic disorders that lead to ventricular arrhythmias. Currently, ICD insertion is used as a default option because we do not know the efficacy of specific drug treatments. With emergence of newer drug therapy a very reasonable therapeutic approach would be to insert an ICD for patient protection while drugs are administered. Device detection of ventricular arrhythmias serves as an excellent method to learn both about the natural history of the disease as well as to assess drug efficacy in a manner that provides for maximum patient safety.

Brady Therapy

The ICD was conceived as a pure antitachyarrhythmia device and has evolved from providing postshock bradycardia backup pacing (VVI) to the current dual-chamber rate responsive pacing capability. Not only the addition of an atrial lead has helped in improving the specificity of the device detection algorithm, but also the hemodynamic benefits can be accrued in patients in whom AV synchrony is crucial. From suppression of premature beats initiating tachycardia to prevention of pause-dependent polymorphic VT, bradycardia pacing capabilities offer significant advantages to an otherwise pure antitachycardia device.

Implantation Technique

The techniques of ICD implantation have developed in parallel with technical innovations. Two decades ago, the ICD system consisted of a thoracotomy lead system requiring an open chest and an abdominally placed pulse generator (Fig 2). With the advent of nonthoracotomy lead and “active can” pectoral implants, using endocardial transvenous leads (Fig 3) have totally replaced the thoracotomy approach. Today the implant is carried out under intravenous sedation and local anesthesia compared with major surgery and general anesthesia earlier. The current generation ICDs are significantly smaller ($<40 \text{ cm}^3$) in size (Table I) compared with the first generator ICD ($>180 \text{ cm}^3$). Virtually all ICD implants in major centers are now carried out by clinical electrophysiologists in the electrophysiology/catheter laboratories.⁵⁴

Implantation of an ICD requires a strict sterile environment and technique used in the operating room. The electrophysiology laboratories have the advantage of excellent fluoroscopic and recording equipment and nurses and technicians familiar with arrhythmias and the treatment of potential complications. Moreover, an increasing number of implants are being carried out immediately postdiagnostic electrophysiology studies performed when risk stratification and transportation to another location is not advisable. Concerns about the risk of infections due to certain operating room conditions like laminar airflow and surgical lighting not available in a typical electrophysiology lab have not been confirmed.

The basic equipment required for ICD implant is listed below:

- Electrocardiogram and intracardiac electrogram monitoring systems
- Fluoroscopy capable of multiple views
- Portable lights/ceiling lights
- Pulse oximetry
- Arterial blood pressure monitoring

- Two external defibrillators with R2 patches
- Pericardiocentesis tray

Operation/Anesthesia Consideration

Critical to the pectoral implant is the pre-operative assessment of the patients' body habitus, assessment of chest wall thickness/deformities. Left pectoral implants are preferred for system DF vector efficacy and the need for current delivery to the left ventricle. If the left side cannot be used, a right-sided approach is reasonable before proceeding to thoracotomy. With the current day devices and lead system (biphasic shocks with variable tilt) the need for thoracotomy leads is extremely rare. Additional measures to reduce the DFTs, like the subcutaneous array or coil electrode can be easily placed alongside the active can in a pectoral implant. A parallel shift has occurred in the type of anesthesia used for ICD implant. Almost all implants (pectoral) are now carried out under intravenous conscious sedation and local anesthesia; the safety, efficacy of this approach has been well established. In fact in one study, ICD implants carried out with local anesthesia and intravenous sedation had higher intraoperative blood pressure and shorter hospital stay compared with patients who underwent general anesthesia.

We typically use intravenous conscious sedation using incremental doses of midazolam and fentanyl for the implant, and intravenous propofol for DF threshold testing. The choice of venous access depends upon the individual operator. However, we prefer cephalic venous approach to subclavian vein punctures for lower incidence of lead fractures and absence of pneumothorax. The current-day peri-operative mortality for an ICD implant is <1%.

B. J. Gersh: Nonetheless, the rate of procedure-related complications including lead dislodgment, pocket infection, device migration, diaphragmatic stimulation, subclavian stenosis, and vascular access problems are quite substantial. This is comprehensively discussed elsewhere in this monograph.

Choice of Lead

Both single and dual coil leads are available for implants. The choice of lead depends upon the individual patient, size of the RV and the LV. It should be noted that an effective DF will occur only when the

majority of the heart is contained within the current field. A typical system consisting of two electrode positions, one at the RV apex and the other in the left pectoral location (active can) provides adequate field current to cover both the ventricles (Fig 3). If adequate safety margin is not achieved, either a dual coil lead, ie, with one electrode in the superior vena cava/in nominate vein or a subcutaneous array can be added. It is noteworthy, however, that additional proximal electrodes in the superior vena cava/in nominate vein is not always beneficial and can be detrimental, for it may direct current delivery away from the left ventricle. Patients with large RV tend to benefit with proximal electrode as it provides better field coverage to the anterior part of the heart. It should be further noted that almost all manufacturers provide a high-energy version of their defibrillators as well.

Electrophysiologic Evaluation of the ICD and the Lead System

This requires induction of VF and currently, evaluation of the DF threshold (DFT) is performed noninvasively through the device. Given the very high success rate of DF with the current devices, use of an emulator has been mostly abandoned. A variety of techniques are available for induction of VF. We prefer to use a T-wave shock with a four-beat drive train at 400 msec followed by a 1-J monophasic shock delivered with a coupling interval of 290 to 310 msec. The coupling intervals may have to be adjusted for patients on concomitant medications that prolong repolarization.

For DFT delineation typically an initial energy level is chosen 10 joules less than the maximum energy output of the device. If the device is successful in terminating VF at this energy level, DFT can be deemed to be completed. The number of VF inductions depends upon the precision with which the DFT needs to be measured and often limited by patients cardiovascular status. The measured DFT is defined as the minimum energy producing DF success and might be significantly lower than the lowest energy required for consistent DF. The relationship between DF energy and success is a probability curve, with success increasing steadily with each increase in energy unit, a 100% success plateau is reached. To allow an adequate safety margin for therapy, the first programmed therapy for DF should be at least 10 J greater than the lowest energy found to successfully terminate VF.

Indications for ICDs

The basic logic to the development of the ICD was to save individuals from VT-VF-initiated SCD. Although the device was clumsy, it delivered a depolarizing shock through ventricular myocardium to terminate VT-VF. In this regard, the ICD has proven its efficacy in all situations where it was tested, for example in the operating room, spontaneous VT-VF, inducible VT-VF, and even device-triggered episodes. Although the success of DF is a statistical phenomenon and depends upon the energy delivered, the success rate can exceed 99%. Higher energy does require more time to charge the capacitor, which may delay the shock and this somewhat reduces the success rate. Four to five shocks of maximum energy are applied by the device, and if the arrhythmia does not terminate, further therapy is aborted; the purpose being that long delays lead to brain damage and survival at that point may not be without undesirable sequelae. If, however the VT-VF terminate for a period of time, a series of new full-energy shocks will be applied.

With the evolution of ATP and low-energy CVs, patients with organized VT have entered the pool of ICD recipients. Painless termination of VT with ATP has increased the acceptance of device therapy for both patients and health professionals and hence the indications for ICDs have grown and will continue to grow.

The initial efforts were mostly focused on prevention of SCD due to VT/VF. However it should be realized that already these devices do more than just shock against VT/VF. For example the current devices already have dual-chamber pacing and sensing. The addition of biventricular pacing technology to the devices and further expansion with dual-chamber DF will make the choice of devices and lead more complex. Also it should not be ignored that there are only limited access points along the greater veins, so the initial choice of device should be well thought out; otherwise in the near future another device and more leads may be required and create a real problem both for implanter and implantee due to limited access sites.

Currently, the recommendations from AHA, ACC, and ACP are in three categories:

1. Indicated - Class I
2. May be indicated - Class IIa and IIb
3. Not indicated - Class III

Each one of these classes is then supported by published supporting data in that particular category in a descending scale of data strength, ie, A

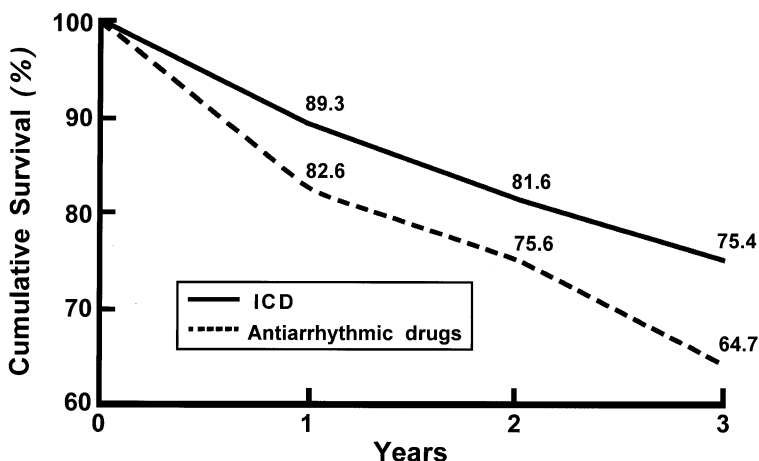


FIG 9. Anti-arrhythmic versus ICD (AVID) (secondary prevention trial). Survival curves are shown in patients randomized to ICD versus amiodarone or Sotalol. As can be appreciated, the survival was better in the ICD recipients in patients who previously experienced life threatening ventricular tachycardia/ventricular fibrillation. Reproduced with permission from The AVID Investigators. A comparison of antiarrhythmic drug therapy with implantable defibrillators in patients resuscitated from near fatal ventricular arrhythmias. *N Engl J Med* 1997;337:1576-83. Copyright © 1997 Massachusetts Medical Society. All rights reserved.

through C, respectively. In the future, the indications will continue to evolve with technology.

It is clear from the foregoing that the implant, the testing of the device and lead system, the appropriate programming, and close follow-up by someone familiar with these issues, is important for proper care of these patients.

Recommendations for ICD Therapy

Class I. ^{20-25,53,55-76} (See Figs 9-12.)

1. Cardiac arrest due to VF or VT not due to a transient or reversible cause. (Level of Evidence: A)
2. Spontaneous sustained VT in association with structural heart disease. (Level of Evidence: B)
3. Syncope of undetermined origin with clinically relevant, hemodynamically significant sustained VT or VF induced at electrophysiology study when drug therapy is ineffective, not tolerated, or not preferred. (Level of Evidence: B) on sustained VT in patients with coronary disease, prior MI, LV dysfunction, and inducible VF or

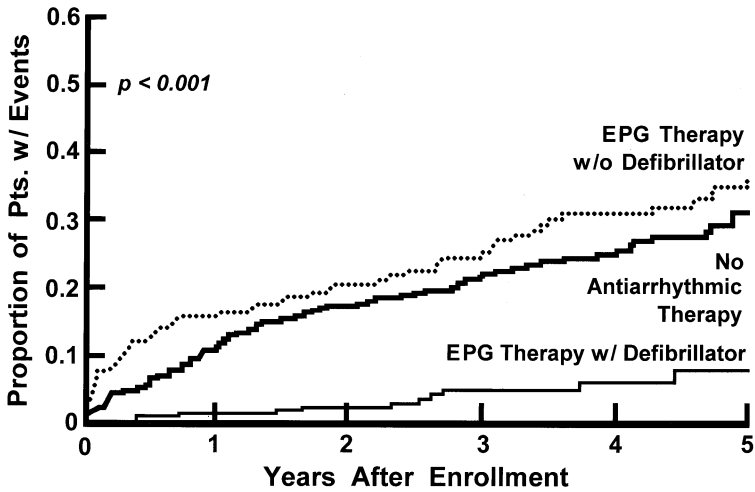


FIG 10. MUSTT Outcome (primary prevention trial). Patients with event are plotted against the years after enrollment. The best outcome in this patient group is with EP guided therapy plus defibrillator. The same management without concomitant ICD (*dotted line*) had the worst outcome, indirectly suggesting that ICD therapy was the critical difference. Reproduced with permission from Buxton AE, Lee KI, Fisher JD, Josephson ME, Prystowsky EN, Hafley G, for the Multicenter Unstained Tachycardia Trial Investigators. A randomized study of the prevention of sudden death in patients with coronary artery disease. *N Engl J Med* 1999;341:1882-90. Copyright © 1999 Massachusetts Medical Society. All rights reserved.

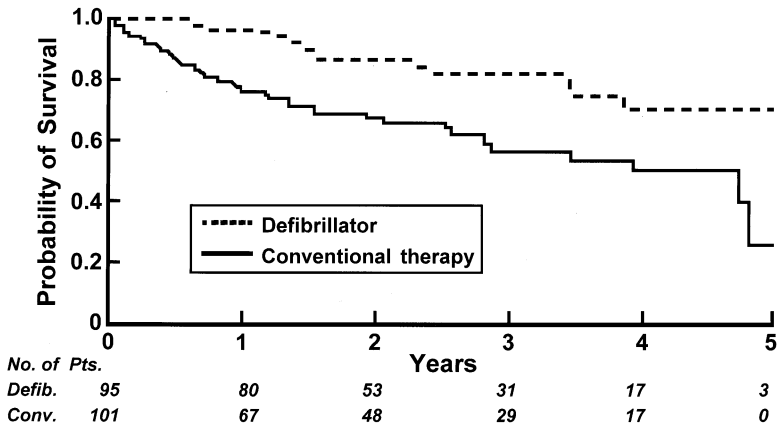
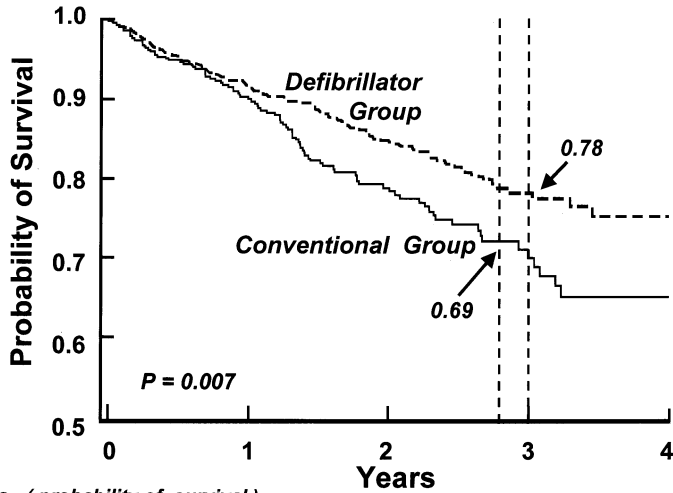


FIG 11. MADIT (primary prevention trial). Soon after the beginning of the trial it becomes clear that the ICD provides a better chance of survival compared to conventional therapy, which turns out to be amiodarone in most cases. This study was prematurely terminated. Reproduced with permission from Moss AJ, Hall WJ, Cannom DS, Daubert JP, Higgins SL, Klein H, et al. Improved survival with an implanted defibrillator in patients with coronary disease at high risk for ventricular arrhythmia. Multicenter Automatic Defibrillator Implantation Trial Investigators. *N Engl J Med* 1996;335:1933-40. Copyright © 1999 Massachusetts Medical Society. All rights reserved.



No. of Pts. (probability of survival)					
Defibrillator	742	503 (0.91)	274 (0.84)	110 (0.78)	9
Conventional	490	329 (0.90)	170 (0.78)	65 (0.69)	3

FIG 12. MADIT II (primary prevention trial). The graph again displays the superiority of ICD for prevention of mortality. The death rate with conventional therapy is statistically lower ($P = .007$) in patients with a left ventricular ejection fraction of $<30\%$ due to CAD. Reproduced with permission from Moss AJ, Zareba W, Hall WJ, Klein H, Wilber DJ, Cannom DS, et al. Prophylactic implantation of a defibrillator in patients with myocardial infarction and reduced ejection fraction. *N Engl J Med* 2002;346:877-83. Copyright © 2002 Massachusetts Medical Society. All rights reserved.

sustained VT at electrophysiology study that is not suppressible by a Class I anti-arrhythmic drug. (Level of Evidence: A)

- Spontaneous sustained VT in patient without structural heart disease not amenable to other treatments, (Level of Evidence: C)

B. J. Gersh: To my mind, the high-risk patients with hypertrophic cardiomyopathy (among which a strong family history of sudden cardiac death is a powerful risk factor) constitutes one of the strongest indications for an ICD implant. Sudden cardiac death and hypertrophic cardiomyopathy are usually “both from the blue” and the majority of patients are asymptomatic or mildly symptomatic before the episodes. Moreover, the intervals between recurrences may be lengthy. ICD in the primary and secondary prevention of sudden cardiac death and hypertrophic cardiomyopathy has revolutionized the clinical management of patients and their families with this disease (Maron BJ, Estesna III, Maron MS, et al. Primary prevention of sudden death as a novel treatment strategy in hypertrophic cardiomyopathy. *Circulation* 2003;107:2872-5).

Class IIIa. ²⁵ Patients with LVEF of $\leq 30\%$ at least 1 month after myocardial infarction and 3 months post coronary artery revascularization surgery, (Level of Evidence: B).

Class IIIb. ⁷⁷⁻⁹³

1. Cardiac arrest presumed to be due to VF when electrophysiologic testing is precluded by other medical conditions. (Level of Evidence: C)
2. Severe symptoms (eg, syncope) attributable to ventricular tachyarrhythmias in patients awaiting cardiac transplantation. (Level of Evidence: C)
3. Familial or inherited conditions with a high risk for life-threatening ventricular tachyarrhythmias such as long-QT synchrony or hypertrophic cardiomyopathy. (Level of Evidence: B)
4. Nonsustained VT with CAD, prior MI, LV dysfunction, and inducible sustained VT or VF at electrophysiologic study. (Level of Evidence: B)
5. Recurrent syncope of undetermined origin in the presence of ventricular dysfunction and inducible ventricular arrhythmias at electrophysiologic study when other causes of syncope have been excluded. (Level of Evidence: C)
6. Syncope of unexplained origin or family history of unexplained SCD in association with typical or atypical right bundle-branch block and ST-segment elevations (Brugada syndrome). (Level of Evidence: C)
7. Syncope in patients with advanced structural heart disease in whom thorough invasive and noninvasive investigations have failed to define a cause (Level of Evidence: C)

B. J. Gersh: One has to be very careful before establishing that the arrhythmia was due to a reversible cause. Hypokalemia, for example, may occur as a secondary phenomenon and many patients with apparently “acute ischemic” episode recurrent arrhythmias occur after coronary revascularization (Kliegel A, Eisenburger P, Steurz F. Survivors of ventricular tachyarrhythmias due to transient or reversible disorder have a higher recurrence rate of lethal cardiac events. *Resuscitation* 2002;54:237-43) (Baoudeg Niebauer M, Kluwahal. Incidents of implantable defibrillator discharges after coronary revascularization in survivors of ischemic sudden cardiac death. *Am Heart J* 1995;130:277-80).

Class III. ⁹⁴⁻¹⁰³

1. Syncope of undetermined cause in a patient without inducible ventricular tachyarrhythmias and without structural heart disease. (Level of Evidence: C)
2. Incessant VT or VF. (Level of Evidence: C)
3. VF or VT resulting from arrhythmias amenable to surgical or catheter ablation; for example, atrial arrhythmias associated with the Wolff-Parkinson-White syndrome, right ventricular outflow tract VT, idiopathic left VT, or fascicular VT. (Level of Evidence: C)
4. Ventricular tachyarrhythmias due to a transient or reversible disorder (eg, acute myocardial infarct, electrolyte imbalance, drugs, or trauma) when correction of the disorder is considered feasible and likely to substantially reduce the risk of recurrent arrhythmia. (Level of Evidence: B)
5. Significant psychiatric illnesses that may be aggravated by device implantation or may preclude systematic follow-up. (Level of Evidence: C)
6. Terminal illnesses with projected life expectancy less than 6 months (Level of Evidence: C)
7. Patients with CAD with LV dysfunction and prolonged QRS duration in the absence of spontaneous or inducible sustained or nonsustained VT who are undergoing coronary bypass surgery. (Level of Evidence: B)
8. NYHA Class IV drug-refractory CHF in patients who are not candidates for cardiac transplantation. (Level of Evidence: C)

B. J. Gersh: Within the next year or two, we should have the results of several randomized trials that we hope will clarify the indications for ICD implantation in patients with and without underlying ischemic heart disease. There are old and new methods of risk stratification, including T-wave alternans in patients with left ventricular dysfunction as currently under prospective evaluation.

Complications Associated with ICD

A variety of untoward problems can be associated with the implantation of these devices. These can be generally divided into those due to (1) procedure-related, (2) lead-related, and (3) generator-related.

Implantation Procedure-related Complications. This depends a great deal on the overall expertise of the operator. However, as with any invasive procedure, certain statistical odds are that over a number of

procedures some complications will occur. These include bleeding, clotting, tissue perforation, hematoma, pericardial effusion, hemo- and pneumothorax, air embolism, infection, seroma around the generator, and coronary venous occlusion where the coronary sinus is cannulated for LV pacing. Perhaps pericardial effusion leading to tamponade, and death are the most serious of procedure-related complications, but they are fortunately rare. In our own experience, the overall mortality related to ICD is less than 1 in 1000 going back to the beginning of our experience. In patients receiving devices for CHF, a higher morbidity and mortality is anticipated because of the relative advanced cardiac disease in these patients.

Lead-related Problems. These are somewhat similar to pacemaker leads including, but not limited to, the insulation break, lead fracture, perforation, tissue reaction, occlusion of the cannulated vessel, dislodgment, nerve or muscle stimulation.

Generator-related Problems. These include random component failure, sensing and detection malfunction, and therapy or lack thereof. The main reason for ICD implantation is to terminate VT-VF and therefore is geared towards acceptance of other malfunctions over lack of appropriate delivery of shock in the event potentially fatal VT or VF occurs. A battery failure is hence carefully monitored by periodic testing and capacitor reformation with possible internally diverting high voltage discharge. Despite that, an occasional failure to perform this initial function does occur, resulting in SCD. It is important to point out that ICD however has a remarkable performance record. In the presence of a functioning, appropriately-placed ICD, arrhythmic death incidence is <2% per year. When SCD death occurs in a rare situation and data analysis is retrieved from the device, one or more of the following are often the reason: (1) inadvertent or deliberate (due to intolerance to repeated shocks) turning off of the device; (2) hemodynamic collapse with associated asystolic death; (3) inadvertent depletion of battery when patient is lost to follow-up; and (4) massive infarct associated with VT, which cannot be converted to a more stable rhythm despite the maximum number of full voltage shocks.

Perhaps one of the most common problems with the ICD is the inappropriate shocks in response to a variety of supraventricular arrhythmias as pointed out earlier, and despite a score of sensing algorithm including dual chamber sensing, it still occurs. Often it can be treated with medication such as digitalis, beta-blockers or calcium channel blockers. When recurrent AF is the reason and is difficult to control, sotalol,

amiodarone, and even AV junctional ablation may be necessary to prevent inappropriate ICD discharges.

B. J. Gersh: Atrial fibrillation is a frequent accompaniment of severe heart failure and is one of the most common causes of inappropriate discharge.

Another somewhat less appreciated aspect of ICD systems is the psychological problems some of these patients experience. These include the fear of shock, fear of dependence on the ICD, depression, and even suicide. Pre-implant and ongoing psychological support if necessary must be considered as a critical part of care in patients with ICDs.

B. J. Gersh: Perhaps as many as 15% to 20% of patients will suffer severe psychologic distress, particularly soon after the implantation and among those receiving frequent shocks. Caregivers should be alerted too the need to address issues such as anxiety,, depression, and fear of physical and sexual activity directly and up front with patients and their families because the latter may be reluctant to raise these concerns.

Chronic infection of the pocket and leads is another troubling complication sometimes necessitating explant of the system. Local tissue necrosis, extrusion, and chronic discomfort from the generator are seen on occasion.

Further synopsis of routine ICD follow-up, the search for common problems, and the reasons for there concerns are listed as follows.

ICD Follow-up

System Evaluation

Routine follow up of the ICD includes:

- Battery status: Evaluation of the battery voltage and charge times, comparing them with recommended replacement indicators
- Arrhythmia episode chronology and electrograms, looking for appropriate, inappropriate, or ineffective therapy
- Lead impedance, comparing with previous values (very low suggests insulation break; very high suggests wire fracture)
- Sensing measurements of intrinsic P waves and QRS complexes

- Pacing threshold(s)

A multitude of factors contribute to system malfunction and include lead type, pulse generator location, and a concomitant pacing system, to mention a few. Patients with biphasic, active can, pectoral systems with mutilunial leads, are at low risk for system complications. Electrogram review, radiography, pacing parameter, and painless high-voltage lead assessment allow for quick detection of any complications without the need for arrhythmia induction. Lead failures increase over time so frequency of follow-up increases over time depending on the risk of system complications.

Recommendations have been developed by the NASPE to assist in the assessment of physician competence in the implantation and follow-up of ICDs. There are two levels of expertise that may be achieved by physicians who care for patients with ICDs. The first level is expertise in the indications for ICDs, follow-up, and troubleshooting for these devices. A more advanced level of training is required for implantation of ICDs.

Battery Status/Charge Time

Elective replacement indicator (ERI) and longevity of each device varies and are available through Guidant, Medtronic, and St Jude and other manufacturers. A rough estimate of longevity (provided in [Table I](#)). The two main determinants of longevity are the amount of pacing and the number of high-voltage shocks delivered.

Lead Integrity

ICD lead failure is not uncommon. It occurs in 12% to 40% of systems over 4 to 5 years of follow-up. Clinical factors that increase the incidence of lead failure include epicardial lead systems, abdominal generator placements, and lead body construction. Nearly 60% of epicardial lead malfunctions are asymptomatic, diagnosed by DF failure, during spontaneous or induced VF, abnormalities found at generator change procedures, or on radiograph showing evidence of lead fracture. The high incidence of epicardial lead failure occurs because these are mechanically separate DF and pace/sense leads ([Fig 2](#)). Unlike transvenous lead systems, an epicardial DF lead fracture does not create “noise” at the sensing leads or stored electrograms. Epicardial DF patch integrity can only be assessed by shock delivery. In abdominal ICD generator placements, the length of the leads require tunneling and increases the risk of lead failure. Therefore, periodic VF induction is critical to assess stable lead integrity in epicardial lead and abdominal generator placements (with transvenous or epicardial leads).

Lead design affects the risk of failure in transvenous systems. Mechanical failures due to conductor fracture or insulation defect are not uncommon, and occurred almost exclusively in coaxial leads. Compared with multilumen leads, there is a much higher rate of failures in coaxial leads. Lead fractures in transvenous systems are usually accompanied by clinical symptoms (ie, shocks), a change in pace/sense thresholds, abnormal real-time or stored electrograms, or radiographic defects. One study showed that 60% of lead failures presented with inappropriate shocks, and noise was detected by electrograms (real-time or stored) in 70% of the cases. In transvenous systems with multilumen lead construction, all lead failures diagnosed during DF testing were also detectable by another diagnostic test (radiograph, electrograms, threshold assessment). However, systems using coaxial or epicardial leads only demonstrated lead failure at times of failed DF, a difference worth remembering.

The risk of lead failure in pectorally-implanted multilumen leads is low. With the recent ability to deliver low-energy painless test pulses of high voltage to evaluate the lead circuit, routine arrhythmia induction to screen for lead malfunction is not necessary (Fig 3).

Abnormal pacing parameters may suggest lead conductor fracture, insulation breach, microdislodgment or macrodislodgment, ineffective ATP, or impaired sensing. A significant elevation of the pacing impedance suggests a discontinuity in the lead or its connection to the pulse generator, and a low impedance suggests insulation defect or electric short within the lead. Lead malfunctions can result in ineffective termination of VT or inappropriate shocks. Impedance values are available noninvasively by device telemetry for this type of troubleshooting. Normal pacing impedance may vary among lead types, but the usual range is 200 to 1000 ohms. After implantation, lead impedance may change, but a value 50% greater than the measurement at implantation suggests fracture. In a mature lead, impedance changes $>30\%$ suggests lead malfunction. The development of "high pacing impedance" leads for ICDs will result in different absolute values indicative of fracture.

The trend of lead impedance is useful in diagnosing lead fracture. A decrease in impedance $>30\%$ raises the possibility of an insulation defect that permits current to escape to the surrounding tissue (which can result in local muscle stimulation). A sudden increase in lead impedance usually suggests lead fracture.

Abnormalities of shocking lead impedance are clearly of utmost importance. Early-generation ICDs used a test shock to assess shocking lead impedance. Values outside the normal range suggest a

conductor defect (high impedance) or insulation breach or short circuit (low impedance). Late-generation devices assess shocking lead impedance imperceptibly with a small test pulse, and some can be programmed to generate an audible tone if impedance measurements are abnormal.

For sensing of ventricular signals measurement of the intrinsic R wave, preferably during sinus rhythm with an amplitude of at least 5 mV should be sought. Medications, myocardial infarction, failed shock, lead tip fibrosis, microdislodgment, and macrodislodgment can result in electrogram diminution. If the peak-to-peak R-wave is <5 mV, electrophysiologic testing to confirm adequate detection during VF is recommended.

B. J. Gersh: The importance of device/drug interactions, particularly with membrane-active anti-arrhythmic agents, must be emphasized. These include pro-arrhythmic effects, elevated pacing, and defibrillation threshold, slowing of the ventricular rate during VT below programmed cut-off values, and alterations of slow rates, which can affect detection. As a general rule, the addition of anti-arrhythmic agents and some other drugs warrants consideration of ventricular defibrillation threshold testing. Amiodarone is particularly prone to increase defibrillation thresholds.

Pacing thresholds may increase due to medications (such as Flecainide), during bradycardia, and with a lead malfunction. Elevated pacing thresholds are acceptable if stable and as long as the patient infrequently requires pacing. However, appropriate pacing function and the ability to reliably capture during ATP must be confirmed. Pacing output settings for ATP, bradycardia pacing, and postshock pacing are independent.

ICDs should be examined radiographically when lead failure is suspected or after significant trauma. Macrodislodgments can be seen when the lead tip has moved compared with the previous or postimplant radiograph. A lead compressed in the narrowly confined space (ie, the first rib and the clavicle) can fracture. Loose pin connection in the header, an abandoned or incompletely removed lead, causing contact noise, may be identified radiographically. Patients with Twiddler's syndrome manipulate their systems, frequently rotating the pulse generator or twisting the leads causing dislodgment and/or fracture. This can be observed on the radiograph. Radiographic manifestations of lead failure may be seen in many patients.

Storage of Information

ICDs are currently capable of storing electrograms of arrhythmias before and after therapy, and intervals preceding the arrhythmia episodes (Fig 4). Heart rate histograms, event logs of atrial arrhythmias, and nonsustained tachycardia episodes are also stored. This information is helpful in determining the cause of a shock and assists in determining the reason for any suspected malfunction.

Furthermore, dual-chamber ICDs provide atrial electrograms, which makes rhythm diagnosis quick and accurate. Far-field electrograms are recorded between widely spaced electrodes and include a large amount of electrical information including QRS morphology and (frequently) P waves, closely resembling the surface ECG. Near-field electrograms are recorded between the lead tip and the adjacent ring or coil to sense the heart rate, representing the signal seen by the device. They provide little diagnostic information regarding VT or SVT configuration because many of these filtered electrograms are alike and their main value is in timing and appearance relative to the reference electrograms.

ICD generator recalls and safety alerts are increasing and affect many patients. Advances in technology have accompanied the changing patterns of device advisory type. Weekly FDA Enforcement Reports from January 1991 to December 2000 have shown that ICD advisories have become more frequent, and a three-fold increase in the number of devices affected per advisory has been observed. The number of devices affected by hardware advisories increased three-fold, due primarily to a 700-fold increase in electrical/circuitry abnormalities. A 20-fold increase in potential battery/capacitor malfunctions has occurred. The number of devices recalled due to firmware (computer programming) abnormalities more than doubled.

Predischarge Evaluation

Initial postoperative evaluation of the ICD begins before patient discharge from the hospital. Most commonly, the patient and ICD are evaluated one day post-ICD implant. The incision should be carefully examined for any evidence of hematoma or infection. The presence of incisional pain should be discussed with the patient. Most commonly, pain management can be achieved through oral analgesics. During the initial 24-hour postoperative period, an arm immobilizer may be helpful to the patient to prevent discomfort from inadvertent above-the-shoulder movement of the affected arm. ICD evaluation can take place at the bedside or during noninvasive electrophysiologic testing

in the laboratory. Currently there is a divergence of opinion among electrophysiologists whether routine predischarge ICD testing in the electrophysiologic laboratory is necessary in all patients, or in just a selected few.

Bedside Evaluation

Bedside evaluation should include review of battery voltage, lead impedance, pacing thresholds, and lead sensing. Significant changes in sensing and capture thresholds from the time of implant may suggest potential lead dislodgment or change in lead position. Although chest radiographs are helpful in evaluation for lead migration, lead fracture insulation breaks are less easily identifiable.¹⁰⁴ Changes in lead impedance may indicate problems with lead integrity. Real-time ICD electrograms should also be reviewed for appropriate sensing of P and R waves. Erratic sensing on the electrogram channel in postoperative period can indicate a connection problem at the header of the pulse generator such as loose set screw. Occasionally, gentle manipulation of the pulse generator within the pocket may reproduce electrical chatter on the electrogram channel, further suggesting a connection problem. Lead migration can also produce erratic sensing and the CS lead location should always be evaluated. Issues related to lead dislodgment or loose connections require surgical intervention.

Noninvasive EP Testing

Noninvasive EP testing of the ICD in the laboratory may be performed in place of the predischarge bedside evaluation. The rationale behind predischarge EP testing is to verify proper functioning of the device and to fine-tune device programming.¹⁰⁵ Patients with slower monomorphic VTs who would benefit from ATP are candidates for predischarge ICD testing in the lab. Timing constraints, as well as repeated inductions of VT-VF in the operating room, may not allow ATP to be programmed at the time of ICD implant. In addition, patients with higher DFTs may benefit from repeat-testing predischarge, to assure there is adequate safety margin for DF.

B. J. Gersh: The role of electrophysiologic testing to determine antitachycardia pacing parameters is controversial since the characteristics of the arrhythmia that is induced in the laboratory may differ from that noted in the clinical situation.

During the predischARGE electrophysiologic testing procedure, the patient is brought to the electrophysiologic lab in the fasting state. Conscious sedation is most commonly administered; however, varying degrees of sedation may be required. Evaluation of battery voltage, lead impedances, pacing thresholds, and sensing are measured similar to the bedside evaluation of the ICD. Then induction of VT can be accomplished noninvasively by programmed electrical stimulation. Detection criteria and therapy for VF, and VT if applicable, should be programmed to the values being tested. Options for VT termination therapies include antitachycardia pacing, low-energy CV, and high- or maximum-energy DF. The patient's hemodynamic status needs to be carefully monitored during repeated VT inductions. Allowing the patient to remain in VT during long unsuccessful ATP attempts could result in prolonged hypotension and increased DFTs.¹⁰⁵

Induction of VF should also be tested at least once during predischARGE electrophysiologic testing and is often a part of intraoperative testing for assuring adequate safety margin and final VF therapy programming. VF induction can be achieved noninvasively via ventricular burst pacing, or T-wave shock or 50-Hz induction. Marker channels need to be reviewed carefully to evaluate for appropriate sensing during VF. Stored electrograms should also be printed and reviewed. The stored electrograms for VT and VF inductions should be saved and can be utilized for comparison during clinical therapies. The patient can be discharged once fully awake and stable post-EP testing.

PredischARGE Teaching

PredischARGE teaching should include the patient and family members if possible. Patients are frequently overwhelmed after undergoing a recent invasive surgery for a potentially life-threatening arrhythmia and family members can assist in retention of information given. The patient and family need to be instructed regarding ICD function and rate cut-off. A model of the ICD can be shown to the patient and family to better comprehend the device and implant procedure. Patients and families benefit from knowing the programmed rate cut-off or having documentation of the ICD settings for potential emergency department visits or possible follow-up stress tests. A better understanding of how the device functions may help reduce patient and family anxiety. The patient and family should be instructed regarding the signs of potential infection. If erythema, swelling, or drainage from the incision develops, the electro-

physiology team should be notified. Pocket fluctuation early after surgery is not always a sign of infection but should be monitored closely.

The patient will receive an ICD identification card from the manufacturer within about 4 to 6 weeks postimplant. The patient should be instructed to carry this card with them at all times in case they need medical care. Duplicate or replacement cards can also be ordered by calling the ICD manufacturers patient services department. Medic Alert bracelets should be encouraged since they indicate to emergency medical personnel that the patient has an ICD. The phone numbers for the electrophysiology team should also be given to the patient for any follow-up questions, concerns, or to report receiving a shock.

The importance of regular follow-up evaluation of the patient and ICD should be emphasized. Initially, patients may be anxious about follow-up visits because of fear of pain or receiving a shock. A good understanding of the procedure during a follow-up visit helps reduce these fears. In addition, the procedure to follow in the event of a shock should be discussed with the patient and family. If the patient receives a single shock and feels well, the electrophysiology team should be notified. However, patients or families should contact emergency medical services for multiple shocks or prolonged symptoms of syncope. This information should be reviewed frequently at follow-up visits. The patient and family should be educated about the sensation of being shocked. Shocks generated by the ICD are a unique aspect of this treatment and have potential to cause psychological distress.¹⁰⁶ The shock can be described as a feeling of being kicked in the chest, however, some patients do report a lighter sensation. Family members and significant others should know that touching a person with an ICD at the time of a shock will not cause injury to themselves. A realistic expectation for the ICD shock may help patients to deal with the associated anxiety and stress.

B. J. Gersh: Frequent, repetitive shocks often indicate lead or device malfunction or the entity of "VT storm" and warrant hospital administration.

Patients may have concerns about the resumption of exercise and activity. Heavy lifting (>10-15 lbs) should be avoided for 4-6 weeks. Patients should be instructed not to raise the affected arm above shoulder height for approximately 4 weeks. In addition, rough, physical sports should be avoided. Patients and family members need to be cognizant and avoid situations that could put them at increased risk in the event of loss

of consciousness (ie, swimming alone). Patients may resume sexual activity once their incisions heal.

B. J. Gersh: The rules about driving vary between countries and also between states within the United States. In general, driving should be prohibited within six months of implantation in patients presenting with syncope or after ICD discharge with clinically significant symptoms. Guidelines have been published (Epstein AE, Miles WM, Benditt DG. Personal and public safety issues related to arrhythmias that may affect consciousness: implications for regulation and position recommendations. A medical/scientific statement from the American Heart Association and the North American Society of Pacing and Electrophysiology. *Circulation* 1996;94:1147-66) (Petch MC. Driving and heart disease. *Eur Heart J* 1998;19:1165-77). It appears that a high proportion of patients do not adhere to advice about driving, but fortunately the accident rate is low (0.4% per year)—lower than among the general driving population (Akiyama T, Powell JL, et al. Resumption of driving after life-threatening ventricular arrhythmias. *N Engl J Med* 2001;345:391).

Patient education should include the information that strong electromagnetic fields can interfere with ICD function. Microwave ovens, cordless telephones, personal computers and most household equipment can be utilized safely. However, keep the motor of any hand-held appliance or tool at least 6 to 12 inches away from the ICD. Most cellular phones are safe to utilize however cellular technology is constantly changing. To avoid potential interference, patients need to keep the cell phone at least 6 inches away from the ICD. The antenna of a citizen band, amateur radios, walkie-talkies or other radio transmitters can produce interference. The distance to maintain from the antenna depends on the transmitter power, frequency, and type of antenna. The ICD manufacturer can be contacted for more specific instructions. The patient should be instructed to avoid heavy electrical or industrial equipment. More importantly, the patient and family should be instructed to contact the electrophysiology team or ICD manufacturer for any questions regarding potential electromagnetic interference.

Before undergoing any medical or dental procedures, patients or family members should notify the medical and dental personnel about the ICD. Most procedures such as diagnostic radiographs, CT scans can be performed without interference to the ICD. Magnetic resonance imaging is not recommended for a patient with an ICD. In addition, the ICD will most likely need to be deactivated before electrocautery, diathermy, or lithotripsy to prevent interference.

Patient education should also include information about traveling. Patients who travel should be encouraged to obtain the names of an electrophysiology team in the state or country they are visiting. This information can be utilized in case of an emergency while traveling. Patients need to be advised that the ICD is not affected when passing through the security gates in airports or by electronic antitheft gates in stores. However, in an airport, the ICD will most likely trigger the metal detection system. The patient should notify airport security of the ICD and show their ICD identification card. The hand-held screening wands should be avoided if possible because these may have strong magnets. A hand search should be requested to clear them through the security system.

B. J. Gersh: Drs Bhatia and colleagues have written a comprehensive and very useful monograph on the technology, indications, and implications of the ICD. The ICD has within a relatively short space of time transformed the management of malignant ventricular arrhythmias and SCD. The device came along at the perfect time as we realized the limitations of currently available anti-arrhythmic drugs and the ability of programmed electrical stimulation to assess therapeutic efficacy.

From the perspective of the secondary prevention of SCD in survivors of malignant ventricular arrhythmias or out-of-hospital cardiac arrests, randomized trials have settled the issues. This is not the case, however, in regards to the indications for the ICD. The primary prevention of patients at high risk, although several trials have made major contributions to our understanding of the efficacy of the device. Nonetheless, issues regarding cost and reimbursement are paramount and we must also remember the complications of the device itself (eg, inappropriate shocks and their effects on the quality of life). Ongoing trials will, we hope, clarify the indications and perhaps identify new approaches to risk stratification of high-risk individuals other than categorization on the basis of ejection fraction alone.

The ICD has provided us with a powerful new weapon in our therapeutic armamentarium. If the patients in which the device is currently utilized represent but a tip of the iceberg of those who are vulnerable to sudden cardiac death, the ICD will not address the societal problem of sudden cardiac death that would hinge on the primary prevention of atherosclerotic cardiovascular disease.

M. M. Scheinman: The authors are to be commended for a first-rate effort in summarizing rather complex data using an orderly, rational, and logical approach. This chapter will be of immense help for clinicians as well as nurses and technical personnel who care for patients with defibrillators. Especially appreciated is their approach to patient care, giving detailed and practical instructions for both ICD indications as well as follow-up.

The chapter also enforces the need for continued re-evaluation of published ICD guidelines. For example, recent studies have emphasized the benefits of ICDs for those with syncope in the presence of severe cardiac

disease, even with negative (noninducible) invasive electrophysiologic studies. In addition, the proviso that ICD is not indicated in the face of remedial conditions (electrolyte abnormalities) needs to be re-examined. It is now well appreciated that such patients have a poor prognosis. For example, hypokalemia often accompanies cardiac arrest because the intense adrenergic stimulus drives glucose and K⁺ into cells. Hence, hypokalemia alone should not be used to deny ICD insertion.^{100,101} Very important problems arise with respect to asymptomatic individuals who are affected by genetic abnormalities that predispose to ventricular arrhythmia (ie, long QT syndrome⁸⁴ and Brugada^{93,94}). Since we lack appropriate risk factors to stratify affected individuals who may be at risk, ICD insertion is at times required to allay psychosocial pressures for asymptomatic affected individuals and this indication is not discussed in the guidelines.

REFERENCES

1. Doyle JT, Kannel WB, McNamara PM, Quickenton P, Gordon T. Factors related to suddenness of coronary death; Combined Albany-Framingham studies. *Am J Cardiol* 1976;37:1073-8.
2. Kuller LH, Perper JA, Dai WS, Rutan G, Traven N. Sudden death and the decline in coronary heart disease mortality. *J Chronic Dis* 1986;39:1001-19.
3. Kannel WB, Cupples LA, D'Agostino RG. Sudden death risk in overt coronary heart disease: The Framingham study. *Am Heart J* 1987;113:799-804.
4. Eisenberg M, Bergner L, Hallstrom A. Paramedic programs and out-of-hospital cardiac arrests: I. Factors associated with successful resuscitation. *Am J Public Health* 1979;69:30-38.
5. Cobb LA, Werner JA, Trobaugh GB. Sudden cardiac death: 1. A decade's experience with out-of-hospital resuscitation. *Mod Concepts Cardiovasc Dis* 1980;49:31.
6. Myerburg RJ, Kessler KM, Bassett AL, Castellanos A. A biological approach to sudden cardiac death: structure, function and cause. *Am J Cardiol* 1989;63:1512-6.
7. Kannel WB, Thomas HE Jr. Sudden coronary death: the Framingham Study. *Ann NY Acad Sci* 1982;382:3.
8. Ornato JP, Om A. Community experience in treating out-of-hospital cardiac arrest. In: Akhtar M, Myerburg RJ, Ruskin JN, editors. Sudden cardiac death: prevalence, mechanisms and approaches to diagnosis and management. Philadelphia (PA): Williams & Wilkins; 1994. p. 450-62.
9. Ornato JP, McNeill SE, Craren EJ, Nelson NM. Limitations on effectiveness of rapid defibrillation by emergency medical technicians in a rural setting. *Ann Emerg Med* 1984;13:1096-9.
10. Weaver WD, Sutherland K, Wirkus MJ, Bachman R. Emergency medical care requirements for large public assemblies and a new strategy for managing cardiac arrest in this setting. *Ann Emerg Med* 1989;18:155-60.
11. O'Rourke RA. Coronary artery surgery for the prevention and treatment of sudden cardiac death. In: Akhtar M, Myerburg RJ, Ruskin JN, editors. Sudden cardiac death: prevalence, mechanisms and approaches to diagnosis and management. Philadelphia (PA): Williams & Wilkins; 1994. p. 531-40.
12. Widerhorn J, Rahimtoola, SH. Results of large-scale studies with β -adrenergic-

- blocking drugs and other non-antiarrhythmic agents for the prevention of sudden cardiac death. In: Akhtar M, Myerburg RJ, Ruskin JN, editors. Sudden cardiac death: prevalence, mechanisms and approaches to diagnosis and management. Philadelphia (PA): Williams & Wilkins; 1994. p. 419-38.
13. Hjalmarson A, Elmfeldt D, Herlitz J, Holmberg S, Malek I, Nyberg G, et al. Effect on mortality of metoprolol in acute myocardial infarction: a double-blind randomised trial. *Lancet* 1981;2:823-7.
 14. CAST Investigators. Preliminary report: effect of encainide and flecainide on mortality in a randomized trial of arrhythmia suppression after myocardial infarction. *N Engl J Med* 1989;321:406-12.
 15. CAST II Investigators. Effect of the antiarrhythmic agent moricizine on survival after myocardial infarction. *N Engl J Med* 1992;327:227-33.
 16. Heger JJ, Prystowsky EN, Jackman WM, Naccarelli GV, Warfel KA, Rinkenberger RL, et al. Clinical efficacy and electrophysiology during long-term therapy for recurrent ventricular tachycardia or ventricular fibrillation. *N Engl J Med* 1981; 305:539-45.
 17. Mason JW, for the Electrophysiologic Study versus Electrocardiographic Monitoring (ESVEM) Investigators. A comparison of electrophysiologic testing with Holter monitoring to predict antiarrhythmic-drug efficacy for ventricular tachyarrhythmias. *N Engl J Med* 1993;329:445-51.
 18. Winkle RA, Mead RH, Ruder MA, Gaudiani VA, Smith NA, Buch WS, et al. Long-term outcome with the automatic implantable cardioverter defibrillator. *J Am Coll Cardiol* 1989;13:1353-61.
 19. Tchou PJ, Kadri N, Anderson J, Caceres JA, Jazayeri M, Akhtar M. Automatic implantable cardioverter defibrillators and survival of patients with left ventricular dysfunction and malignant ventricular arrhythmias. *Ann Intern Med* 1988;109:529.
 20. Kuck KH, Cappato R, Siebels J, Ruppel R. Randomized comparison of antiarrhythmic drug therapy with implantable defibrillators in patients resuscitated from cardiac arrest: the Cardiac Arrest Study Hamburg (CASH). *Circulation* 2000;102: 748.
 21. The AVID Investigators. A comparison of antiarrhythmic drug therapy with implantable defibrillators in patients resuscitated from near fatal ventricular arrhythmias. *N Engl J Med* 1997;337:1576-83.
 22. Connolly SJ, Gent M, Roberts RS, Dorian P, Roy D, Sheldon RS, et al. Canadian Implantable Defibrillator Study (CIDS): a randomized trial of the implantable cardioverter defibrillator against amiodarone. *Circulation* 2000;101:1297-302.
 23. Moss AJ, Hall WJ, Cannom DS, Daubert JP, Higgins SL, Klein H, et al. Improved survival with an implanted defibrillator in patients with coronary disease at high risk for ventricular arrhythmia. Multicenter Automatic Defibrillator Implantation Trial Investigators. *N Engl J Med* 1996;335:1933-40.
 24. Buxton AE, Lee KL, Fisher JD, Josephson ME, Prystowsky EN, Hafley G, for the Multicenter Unstained Tachycardia Trial Investigators. A randomized study of the prevention of sudden death in patients with coronary artery disease. *N Engl J Med* 1999;341:1882-90.
 25. Moss AJ, Zareba W, Hall WJ, Klein H, Wilber DJ, Cannom DS, et al. Prophylactic implantation of a defibrillator in patients with myocardial infarction and reduced ejection fraction. *N Engl J Med* 2002;346:877-83.
 26. Bigger JT Jr, for the CABG-Patch Investigators. Prophylactic use of implanted

- cardiac defibrillators in patients at high risk for ventricular arrhythmias after coronary artery bypass graft surgery. *N Engl J Med* 1997;337:1569-75.
27. Lown B, Newman J, Amarasingham R, Berkovits BV. Comparison of alternating current with direct current electroshock across the closed chest. *Am J Cardiol* 1962;10:223-233.
 28. Mirowski M, Mower MM, Staewen WS, Tabatznik B, Mendeloff AI. Standby automatic defibrillator: an approach to prevention of sudden coronary death. *Arch Intern Med* 1970;126:158-161.
 29. Lown B, Axelrod P. Implanted standby defibrillators. *Circulation* 1972;46:637-9.
 30. Mirowski M, Mower MM. The automatic implantable defibrillator: Some historical notes. Brugada P, Willens H, eds. *Cardiac arrhythmias: where to go from here?* Mount Kisco (NY): Futura Publishing Company, Inc; 1987.
 31. FDA Approval of ICD (1985).
 32. Troup PJ. Implantable cardioverters and defibrillators. *Curr Prob Cardiol* 1989;14: 673-843.
 33. Kroll M, Lehmann M, editors. *Implantable cardioverter defibrillator: the engineering-clinical interface*. Boston (MA): Kluwer Academic Publishers; 1996.
 34. Bardy GH. Abstract presented at the American College of Cardiology Annual Scientific Session; March 7-10, 2004; New Orleans, La. See: Bardy GH, Lee KL, Mauk DB, and the SCD-HeFT Pilot Investigators. The Sudden Cardiac Death in Heart Failure Trial: pilot study [abstract]. *Pacing Clin Electrophysiol* 1997;20: 1148.
 35. Daubert J, Linde C, Cazeau S, Kappenberger L, Sutton R, Bailleur C. Clinical effects of biventricular pacing in patients with severe heart failure and chronic atrial fibrillation: results from the multisite stimulation in cardiomyopathy (MUSTIC Study) Group II [abstract]. *Circulation* 2000;102(II):693. Abstract 106459.
 36. Cazeau S, Leclercz C, Lavergne T, Walker S, Varma C, Linde C, et al. Effects of multisite biventricular pacing in patients with heart failure and intraventricular conduction delay. *N Engl J Med* 2001;344:873-80.
 37. Leclercq C, Alonso C, Revault F, et al. Is the long-term benefit of biventricular pacing in patients with advanced heart failure influenced by the baseline QRS duration? [abstract]. *Pacing Clin Electrophysiol* 2001;24(4 Pt 2):540.
 38. Schaumann A, Muhlen F, Crouska B, et al. Enhanced detection criteria in implantable cardioverter-defibrillators to avoid inappropriate therapy. *Am Heart J* 1996;78:42.
 39. Swerdlow CD, Chen PS, Kass RM, Allard JR, Peter CT. Discrimination of ventricular tachycardia from sinus tachycardia and atrial fibrillation in a tiered therapy cardioverter-defibrillator. *J Am Coll Cardiol* 1994;23:1342-55.
 40. Micro Jewel II system reference guide. Publication number UC 960158laen1968-03-002, Medtronic Inc., Minneapolis, MN, July, 1996.
 41. Duetz L, Bardy G, Mitchell L, et al. Clinical evaluation of electrogram width measurements for automatic detection of ventricular tachycardia [abstract]. *Pacing Clin Electrophysiol* 1996;19(4 Pt 2):582.
 42. Barold M, Newby K, Tomassoni G, Kearney M, Brandon J, Natale A. Prospective evaluation of new and old criteria to discriminate between supraventricular and ventricular tachycardia in implantable defibrillators. *Pacing Clin Electrophysiol* 1998;21:1347-55.
 43. Wilkoff B, Cook J, Epstein A, Greene H, Halstrom A, Hsia H, et al. Dual-chamber

- pacing or ventricular backup pacing in patients with an implantable defibrillator: the Dual Chamber and VVI Implantable Defibrillator (DAVID) Trial. *JAMA* 2002;288:3115-23.
44. Natale AN, Sra J, Axtell K, Akhtar M, Newby K, Kent V, et al. Undetected ventricular fibrillation in transvenous implantable cardioverter-defibrillators: prospective comparison of different lead system-device combinations. *Circulation* 1996;93:91-8.
 45. Kopp DE, Burke MC, Lin A, et al. Overusing in dual chamber implantable cardioventricular-defibrillators [abstract]. *Pacing Clin Electrophysiol* 2000;23(4 Pt 2):591.
 46. Schallmann A, von zur Muhlen F, Herse B, Gonska BD, Kreuzer H. Empirical versus tested antitachycardia pacing in implantable cardioverter defibrillators: a prospective study including 200 patients. *Circulation* 1998;97:66-74.
 47. Peinado R, Almendral JM, Rius T, Moya A, Merino JL, Martinez-Alday J, et al. Randomized, prospective comparison of four burst pacing algorithms for spontaneous ventricular tachycardia. *Am J Cardiol* 1998;82:1422-5.
 48. Calkins H, el-Atassi R, Kalbfleisch S, Langberg J, Morady F. Comparison of fixed burst versus decremental burst pacing for termination of ventricular tachycardia. *Pacing Clin Electrophysiol* 1993;16:26-32.
 49. Restivo M, Gough WB, El Sherrif N. Reentrant ventricular rhythms in the late myocardial infarction period: prevention of reentry by dual stimulation during basic rhythm. *Circulation* 1988;77:429-44.
 50. Bardy GH, Poole UE, Kudenchuck PJ, Dolack GL, Kelso D, Mitchell R. A prospective randomized repeat crossover comparison of antitachycardia pacing with low-energy cardioversion. *Circulation* 1993;87:1889-96.
 51. Almendral J, Arenal A, Villacastin JP, San Roman D, Bueno H, Alday JM, et al. The importance of antitachycardia pacing for patients presenting with ventricular tachycardia. *Pacing Clin Electrophysiol* 1993;16:535-9.
 52. Saksena S, Krol RB, Makija V. Comparison of clinical benefits and outcome in patients with programmable and nonprogrammable implantable cardioverter defibrillators. *Pacing Clin Eletrophysiol* 1992;15:1279-90.
 53. Wathen MS, Sweeney MO, DeGroot PJ, Stark AJ, Koehler JL, Chisner MB, et al. Shock reduction using antitachycardia pacing for spontaneous rapid ventricular tachycardia in patients with coronary artery disease. *Circulation* 2001;104:796-801.
 54. Schmitt C, Alt E, Plewan A, Schomig A. Initial experience with implantation of cardioverter defibrillator under local anesthesia by electrophysiologists. *Eur Heart J* 1996;17:1710-6.
 55. Saksena S, Poczobutt-Johanos M, Castle LV, Fogoros RN, Alpert BL, Kron J, et al, for the Guardian Multicenter Investigators Group. Long-term multicenter experience with second-generation implantable pacemaker-defibrillator in patients with malignant ventricular tachyarrhythmias. *J Am Coll Cardiol* 1992;19:490-9.
 56. Bardy GH, Troutman C, Poole JE, Kudenchuk PJ, Dolack GL, Johnson G, et al. Clinical experience with a tiered-therapy, multiprogrammable antiarrhythmia device. *Circulation* 1992;85:1689-98.
 57. Mirowski M, Reid PR, Mower MM, Watkins L, Gott VL, Schauble JF, et al. Termination of malignant ventricular arrhythmias with an implanted automatic defibrillator in human beings. *N Engl J Med* 1980;303:322-4.
 58. Lehmann MH, Steinman RT, Schuger CD, Jackson K. The automatic implantable

- cardioverter defibrillator as antiarrhythmic treatment modality of choice for survivors of cardiac arrest unrelated to acute myocardial infarction. *Am J Cardiol* 1988;63:803-5.
59. Fogoros RN, Fiedler SB, Elson JJ. The automatic implantable cardioverter-defibrillator in drug-refractory ventricular tachyarrhythmias. *Ann Intern Med* 1997;107:635-41.
 60. Fogoros RN, Elson JJ, Bonnet CA, Fiedler SB, Burkholder JA. Efficacy of the automatic implantable cardioverter-defibrillator in prolonging survival in patients with severe underlying cardiac disease. *J Am Coll Cardiol* 1990;16:381-6.
 61. Newman D, Sauve MJ, Herre J, Langberg JJ, Lee MA, Titus C, et al. Survival after implantation of the cardioverter-defibrillators. *Am J Cardiol* 1992;69:899-903.
 62. Powell AC, Fuchs T, Finkelstein DM, Garan H, Cannom DS, McGovern BA, et al. Influence of implantable cardioverter-defibrillators on the long-term prognosis of survivors of out-of-hospital cardiac arrest. *Circulation* 1993;88:1083-92.
 63. Crandall BF, Morris CD, Cutler JE, Kudenchuk PJ, Peterson JL, Liem LB, et al. Implantable cardioverter-defibrillator therapy in survivors of out-of-hospital sudden cardiac death without inducible arrhythmias. *J Am Coll Cardiol* 1993;21:1186-92.
 64. Saksena S, for the PCD Investigators. Clinical outcome of patients with malignant ventricular tachyarrhythmias and a multiprogrammable implantable cardioverter-defibrillator implanted with or without thoracotomy: an international multicenter study. *J Am Coll Cardiol* 1994;23:1521-30.
 65. Zipes DP, Roberts D, for the Pacemaker-Cardioverter-Defibrillator Investigators. Results of the international study of the implantable pacemaker cardioverter-defibrillator: a comparison of epicardial and endocardial lead systems. *Circulation* 1995;92:59-65.
 66. Wever EF, Hauer RN, Schrijvers G, van Capelle FJ, Tijssen JG, Crijns HJ, et al. Cost-effectiveness of implantable defibrillator as first-choice therapy versus electrophysiologically guided, tiered strategy in postinfarct sudden death survivors: a randomized study. *Circulation* 1996;93:489-96.
 67. Borggreve M, Chen X, Martinez-Rubio A, Hindricks G, Haverkamp W, Block M, et al. The role of implantable cardioverter defibrillators in dilated cardiomyopathy. *Am Heart J* 1994;127:1145-50.
 68. Morady F, Harvey M, Kalbfleisch SJ, el-Atassi R, Calkins H, Langberg JJ. Radiofrequency catheter ablation of ventricular tachycardia in patients with coronary artery disease. *Circulation* 1993;87:363-72.
 69. Wever EF, Hauer RN, van Capelle FL, Tijssen JG, Crijns HK, Algra A, et al. Randomized study of implantable defibrillator as first-choice therapy versus conventional strategy in postinfarct sudden death survivors. *Circulation* 1995;91:2195-203.
 70. Krol RB, Saksena S. Clinical trials of antiarrhythmic drugs in recipients of implantable cardioverter-defibrillators. In: Saksena S, Luderitz B, editors. *Interventional electrophysiology*, 2nd ed. Armonk (NY): Futura Publishing Co, 1996:365-75.
 71. Saksena S, Briethardt G, Dorian P, Greene HL, Madan N, Block M. Nonpharmacological therapy for malignant ventricular arrhythmias: implantable defibrillator trials. *Prog Cardiovasc Dis* 1996;38:429-44.
 72. Nisam S, Kaye SA, Mower MM, Hull M. AICD automatic cardioverter defibrillator

- clinical update: 14 years' experience in over 34,000 patients. *Pacing Clin Electrophysiol* 1995;18:142-7.
73. Axtell K, Tchou P, Akhtar M. Survival in patients with depressed left ventricular function treated by implantable cardioverter defibrillator. *Pacing Clin Electrophysiol* 1991;14:291-6.
 74. Hook BG, Marchlinski FE. Value of ventricular electrogram recordings in the diagnosis of arrhythmias precipitating electrical device shock therapy. *J Am Coll Cardiol* 1991;17:985-90.
 75. Leitch JW, Gillis AM, Wyse DG, Yee R, Klein GJ, Guiraudon G, et al. Reduction in defibrillator shocks with an implantable device combining antitachycardia pacing and shock therapy. *J Am Coll Cardiol* 1991;18:145-51.
 76. Bocker D, Haverkamp W, Block M, Borggrefe M, Hammel D, Breithardt G. Comparison of D, L-sotalol and implantable defibrillators for treatment of sustained ventricular tachycardia or fibrillation in patients with coronary artery disease. *Circulation* 1996;94:151-7.
 77. Saksena S, Moss AJ, Gorgeberidze I, et al. Factors associated with shock delivery in the Multicenter Automatic Defibrillator Implantation Trial (MADIT) [abstract]. *J Am Coll Cardiol* 1997;29(Suppl A):79.
 78. Bardy GH, Yee R, Jung W, for the Active Can Investigators. Multicenter experience with a pectoral unipolar implantable cardioverter-defibrillator. *J Am Coll Cardiol* 1996;28:400-10.
 79. Groh WJ, Silka MJ, Oliver RP, Halperin BD, McAnulty JH, Kron J. Use of implantable cardioverter-defibrillators in the congenital long QT syndrome. *Am J Cardiol* 1996;78:703-6.
 80. Grimm M, Wieselthaler G, Avanesian R, Grimm G, Schmidinger H, Schreiner W, et al. The impact of implantable cardioverter-defibrillators on mortality among patients on the waiting list for heart transplantation. *J Thorac Cardiovasc Surg* 1995;110:532-9.
 81. Sweeney MO, Ruskin JN, Garan H, McGovern BA, Guy ML, Torchiana DF, et al. Influence of the implantable cardioverter/defibrillator on sudden death and total mortality in patients evaluated for cardiac transplantation. *Circulation* 1995;92:3273-81.
 82. Freidberg CK, Donoso E, Stein WG. Nonsurgical acquired heart block. *Ann N Y Acad Sci* 1964;111:835-47.
 83. DePasquale NP, Bruno MS. Natural history of combined right bundle branch block and left anterior hemiblock (bilateral bundle branch block). *Am J Med* 1973;54:297-303.
 84. Garson AJ, Dick MI, Fournier A, Gilette PC, Hamilton R, Kugler JD, et al. The long QT syndrome in children: an international study of 287 patients. *Circulation* 1993;87:1866-72.
 85. McKenna WJ, Franklin RC, Nihoyannopoulos P, Robinson KC, Deanfield JE. Arrhythmia and prognosis in infants, children and adolescents with hypertrophic cardiomyopathy. *J Am Coll Cardiol* 1988;11:147-53.
 86. Fananapazir L, Epstein SE. Hemodynamic and electrophysiologic evaluation of patients with hypertrophic cardiomyopathy surviving cardiac arrest. *Am J Cardiol* 1991;67:280-7.
 87. Wichter T, Block M, Bocker D, Dorggrefe G, Briethardt G. Cardioverter-

- defibrillator therapy in a high-risk subgroup of patients with arrhythmogenic right ventricular disease [abstract]. *J Am Coll Cardiol* 1993;21:127.
88. Evans RW, Manninen DL, Dong FB, Frist WH, Kirklin JK. The medical and surgical determinants of heart transplantation outcomes: the results of a consensus survey in the United States. *J Heart Lung Transplant* 1993;12:42-5.
 89. Maron BJ, Fananapazir L. Sudden cardiac death in hypertrophic cardiomyopathy. *Circulation* 1992;85(Suppl I):I-57-63.
 90. Kaminer SJ, Pickoff AS, Dunnigan A, Sterba R, Wolff GS. Cardiomyopathy and the use of implanted cardio-defibrillators in children. *PACE Pacing Clin Electrophysiol* 1990;13:593-7.
 91. Mehta D, Saksena S, Krol RB, John T, Saxena A, Raju R, et al. Device use patterns and clinical outcome of implantable cardioverter defibrillator patients with moderate and severe impairment of left ventricular function. *Pacing Clin Electrophysiol* 1993;16:179-85.
 92. Wilber DJ, Olshansky B, Moran JF, Scanlon PJ. Electrophysiological testing and nonsustained ventricular tachycardia: use and limitations in patients with coronary artery disease and impaired ventricular function. *Circulation* 1990;82:350.
 93. Priori SG, Napolitano C, Gasparini M, Pappone C, DellaBella P, Giordano U, et al. Natural history of Brugada syndrome: insights for risk stratification and management. *Circulation* 2002;105:1342-7.
 94. Brugada J, Brugada R, Brugada P. Pharmacological and device approach to therapy of inherited cardiac diseases associated with cardiac arrhythmias and sudden death. *J Electrocardiol* 2000;33(suppl):41-7.
 95. Stevenson WF, Khan H, Sager P, Saxon LA, Middlekauff HR, Natterson PD, et al. Identification of reentry circuit sites during catheter mapping and radiofrequency ablation of ventricular tachycardia late after myocardial infarction. *Circulation* 1993;88:1647-70.
 96. Gonska BD, Cao K, Schaumann A, Dorsezewski A, von zur Muhlen F, Kreuzer H. Catheter ablation of ventricular tachycardia in 136 patients with coronary artery disease: results and long-term follow-up. *J Am Coll Cardiol* 1994;24:1506-14.
 97. Hindricks G, for the Multicentre European Radiofrequency Survey (MERFS) Investigators of the Working Group on Arrhythmias of the European Society of Cardiology. The Multicentre European Radiofrequency Survey (MERFS): complications of radiofrequency catheter ablation of arrhythmias. *Eur Heart J* 1993;14:1644-53.
 98. Klein LS, Shih HT, Hackett FK, Zipes DP, Miles WM. Radiofrequency catheter ablation of ventricular tachycardia in patients without structural heart disease. *Circulation* 1992;85:1666-74.
 99. Anderson JL, Hallstrom AP, Epstein AE, Pinski SL, Rosenberg Y, Nora MO, et al, for the AVID Investigators. Design and results of the antiarrhythmics vs implantable defibrillators (AVID) registry. *Circulation* 1999;99:1692-9.
 100. Michaud GF, Sticherling C, Tada H, Oral H, Pelosi F Jr, Knight BP, et al. Relationship between serum potassium concentration and risk of recurrent ventricular tachycardia or ventricular fibrillation. *J Cardiovasc Electrophysiol* 2001;12:1109-12.
 101. Michaud GF, Strickberger SA. Should an abnormal serum potassium concentration be considered a correctable cause of cardiac arrest? *J Am Coll Cardiol* 2001;38:1224-5.
 102. Vlay SC, Olson LC, Friehehione GL, Friedman R. Anxiety and anger in patients

- with ventricular tachyarrhythmias: responses after internal cardioverter defibrillator implantation. *PACE Pacing Clin Electrophysiol* 1989;12:366-73.
103. Luderitz B, Jung W, Deister A, Marneros A, Manz M. Patient acceptance of the implantable cardioverter defibrillator in ventricular tachyarrhythmias. *Pacing Clin Electrophysiol* 1993;16:1815-21.
 104. Gupta A, Zegel HG, Dravid VS, Nierenberg SJ, Freiman DB. Value of radiography in diagnosing complications of cardioverter defibrillators implanted without thoracotomy in 437 patients. *AJR Am J Roentgenol* 1997;168:105-8.
 105. Wolbrette DL, Naccarelli GV. Management of implantable cardioverter defibrillator patients: Role of pre-discharge electrophysiologic testing and proper patient instruction before hospital discharge. *Curr Opin Cardiol* 2001;16:72-5.
 106. Thomas SA, Friedmann E, Kelly FJ. Living with an implantable cardioverter-defibrillator: a review of the current literature related to psychosocial factors. *AACN Clin Issues* 2001;12:156-63.