The potential for interaction between pacemakers and implantable cardioverter/defibrillators (ICDs) and the medical and nonmedical environment, and between these devices and certain cardioactive drugs, has been recognized for years. Whereas a number of experimental and clinical studies have been performed to define some of these interactions, in many instances data are sparse and anecdotal clinical experiences form the basis for decision making and recommendations. Nevertheless, given the proliferation of rhythm-management devices in use in the population today, practitioners may find a guide to management of these patients helpful. This management guideline is therefore offered not as an extensive, literature-based review, but as a framework on which to understand specific types of problems that may be encountered in the daily lives of patients who have such implanted devices.

ELECTROMAGNETIC INTERFERENCE IN PATIENTS WITH IMPLANTED PACEMAKERS AND ICDs

Electromagnetic interference (EMI) occurs when electromagnetic waves emitted by one electronic source or device impede the normal function of another electronic device. Electromagnetic interference can have clinically significant effects on patients with implanted pacemakers and ICDs. Potential sources of EMI outside the operating room, where electrocautery devices have long been known to have the potential for interfering with pacemaker function, can be expected to increase in light of burgeoning telecommunications technology.

Household Appliances

The questions most often raised by patients after device implantation relate to the use of microwave ovens, passage through airport metal detectors, and use of cellular telephones. Although no recent studies that systematically test the effect of household microwave energy on implanted devices have been performed, it is widely believed and accepted that all modern pacemakers are adequately shielded from microwave energy produced by today's appliances. Pacemaker manufacturers, therefore, recommend that patients with implanted devices need not take special precautions when using microwaves or other common household appliances, such as televisions, radios (including ham systems), toasters, and electric blankets. In support of this recommendation, observations of adverse clinical interactions are extremely rare.

Airport Security Devices

The effects of airport metal-detector gates on implanted pacemakers were studied more than a decade ago. In unselected patients who were monitored as they passed through typical metal detector gates set at their highest sensitivity setting, the metal-detecting signal was, as expected, invariably activated when the patients walked through; however, in none of the patients was pacing system behavior affected. Specifically, none of the devices...
Nora Goldschlager, MD, Division of Cardiology, Department of Medicine, San Francisco General Hospital, San Francisco, and Department of Medicine, University of California, San Francisco; Andrew E. Epstein, MD, Division of Cardiovascular Disease, Department of Medicine, University of Alabama, Birmingham; Fethi M. Fouad-Tarazi, MD, PhD, Hemodynamic and Neuroregulation Laboratory and Syncope Clinic, Department of Cardiology, Cleveland Clinic Foundation, Cleveland, Ohio; Paul A. Friedman, MD, Department of Medicine, Mayo Medical School and Mayo Clinic, Rochester, Minn; Eli S. Gang, MD, Department of Medicine, University of California–Los Angeles School of Medicine, Los Angeles; Blair P. Grubb, MD, Departments of Medicine and Pediatrics and Cardiac Electrophysiology and Pacing Laboratories, Division of Cardiology, Medical College of Ohio, Toledo; Ryszard Krol, MD, PhD, Cardiovascular Institute, Arrhythmia and Pacing Service, Atlantic Health System at Passaic, Passaic, NJ; and Brian Olshansky, MD, Division of Cardiology, Department of Medicine, University of Iowa Hospitals and Clinics, Iowa City.

were reset to the “noise-protection mode” or asynchronous (fixed-rate) mode of function, nor were any of the devices’ outputs inhibited in paced patients or inappropriately delivered in patients who had spontaneous cardiac rhythm. Therefore, it is acceptable practice to advise the patient that, while airport screening gates can detect the ferrous material in the pacemaker or ICD metal case, causing an alarm to be triggered, the function of the device will likely not be affected.

In contrast to metal detector gates, hand-held metal detectors contain magnets and thus have a greater potential for interaction with the implanted device. Therefore, we recommend that patients present their device identification card to security personnel upon arrival for the purpose of obtaining security clearance by means of a hand search, an approach that should not interfere with passing of inspection.

Cellular Telephones

With the proliferation of cellular telephones came the realization that these telephones have the potential to interfere with pacemaker function. Case reports of pacemaker malfunction, as well as an in vitro study showing possible EMI caused by cellular telephones, prompted a large multicenter trial of the effects of 5 types of cellular telephones on various permanent pacemaker models. This study confirmed that, to cause interference with proper device function, the cellular telephone needed to be closer than 10 cm from the pacemaker pocket; the highest incidence of telephone-induced EMI occurred with the telephone switched on and positioned directly over the pulse generator. In contrast, the incidence of EMI when the telephone was positioned at the patient's ear was very low. None of the interference episodes were of clinical significance (prolonged inhibition of pacemaker output causing presyncope, syncope, dizziness, or shortness of breath; provocation of spontaneous tachyarrhythmias or rapid paced ventricular rates; or changes in programmed pacemaker settings) when the cellular telephone was held at the patient's ipsilateral ear.

Analog cellular telephones are much less likely than digital devices to interfere with pacing system function. However, there is variability in interference phenomena among pacemaker manufacturers and models. The inclusion of feed-through filters, now incorporated in most modern pacemakers, lessens the likelihood of EMI. Dual-chamber pacemakers are more likely to experience EMI, likely because of the atrial channel's being programmed to be “more sensitive” to appropriately sense and respond to the low-voltage signals of spontaneous P-waves (1- to 4-mV compared with 5- to 12-mV amplitudes of spontaneous QRS complexes). Oversensing of environmental signals in the atrial channel will cause ventricular stimulus output (“tracking”) and inappropriate ventricular-paced rhythms. Dual-chamber pacemakers are also more susceptible to noise-reversion pacing, in which asynchronous (fixed-rate) pacing can occur. Whereas the highest incidence of symptoms has been reported to occur in patients considered to be pacemaker “dependent” (absence of underlying rhythm >30 per minute or symptoms of cerebral hypoperfusion at slow spontaneous rates), no clinically significant symptoms have been observed when the cellular telephone is held sufficiently distant (≥10 cm) from the pulse generator.

Recognizing that digital cellular telephones are more likely to cause EMI than are analog telephones, that the cellular telephone industry is moving toward exclusive use of digital technology, and that the majority of pacing systems implanted currently are dual-chamber, studies indicate that the use of cellular telephones (keeping the above caveats in mind) does not pose a significant health risk to patients with implanted permanent pacemakers. It is well to advise patients not to place switched-on cellular telephones in a coat pocket overlying the pulse generator.

The effects of digital cellular telephones on the function of ICDs have been studied in a relatively small number of patients with various models provided by a single manufacturer. The static magnetic field generated by the cellular telephone when placed close (<0.5 cm) to the ICD during in vitro testing has caused temporary suspension of ventricular tachycardia and defibrillation detection. However, during in vivo observations, no patients were affected by EMI oversensing. Absence of adverse effects has been found in patients with abdominal as well as pectoral ICD implant sites, and in both submuscular as well as subcutaneous pocket locations. There is a scarcity of data assessing interactions between cellular telephones and ICDs of different manufacturers, however, precluding firm recommendations for the patient. Similarly, data are sparse regarding interactions with ICDs with pacing capability, although it is likely that the pacemaker components of the ICD respond in similar fashion to pacemakers, and that the same considerations apply. It is prudent to recommend...
that a cellular telephone be carried at least 10 to 15 cm from the ICD, in a switched-off position.

**Electronic Article Surveillance Systems**

Electronic article surveillance (EAS) systems have recently been recognized as having the potential to interact with implanted rhythm devices. The commercial use of such scanning devices is widespread, and case reports have been published in which patients received “inappropriate” ICD discharges while lingering within or touching EAS gates. Because a very large number of EAS devices are extant, and because very few episodes of possible interaction between these devices and pacemakers or ICDs have been reported, it is possible that too much is being made of this issue, perhaps resulting in undue concern among patients with implanted devices.

Electronic surveillance systems use principally 3 technologies to detect the presence of a metal alloy tag (and thus a potential theft) within an electromagnetic field created between 2 parallel gates: magnetic audiofrequency, swept radiofrequency, and acoustomagnetic or pulsed low frequency. The specific type of EAS device used by a particular facility is not known to the consumer. The literature suggests that significant EMI with implanted rhythm devices is most likely to occur with the acoustomagnetic surveillance systems. Pacemakers are more likely to be affected than ICDs, likely because of differences in electronic technology and stronger shielding in the latter devices. In one study of patients with ICDs who were subjected to electromagnetic fields of 6 different EAS devices, no instance of significant interference with normal ICD function was seen during positioning of the patients for 5 minutes between the EAS gates while rotating 360° and leaning against the EAS transmitter. In another study, patients with ICDs with pacing capability walked through EAS gates and underwent prolonged exposure within the gates, both during pacing from the implanted device and during spontaneous cardiac rhythm. The absence of clinically significant interaction between EAS gates and ICDs was confirmed during walking, even at a very slow pace, through the gates. Under conditions of extreme exposure, however, 4% of 169 patients did exhibit some interaction between the ICD and the EAS device, manifested by noise oversensing that resulted in prolonged or complete inhibition of device output. Such output inhibition might be clinically relevant under specific circumstances, such as pacemaker dependency, and could also have resulted in inappropriate ICD shocks had this function not been suspended during the testing protocol. Older-generation ICDs, and those implanted in the abdomen, were more likely than newer-generation subpectorally implanted ones to manifest these interactions. In general, EAS devices do not pose a threat to the tachycardia functions of ICDs under normal conditions; more prolonged exposures or closer proximity to the EAS transmitter can result in inappropriate shocks.

In contrast to ICDs, interaction between permanent pacemakers and EAS devices has been observed in up to 20% of patients with dual-chamber devices and in up to 10% of those with single-chamber devices under study conditions of prolonged standing within and rotating around the EAS gates. Asynchronous (noise-reversion mode) pacing, atrial and ventricular undersensing and oversensing, and surveillance device–induced pacing have been described during a “real-life” walk through such gates. Reprogramming has not been reported, and patients do not experience severe symptoms (such as syncope). These effects on pacemakers occur only while the patient is within the EAS device’s magnetic field; unipolar devices are more susceptible than bipolar devices, the latter being relatively immune to interference.

It is prudent to advise patients to walk normally, and not slowly, through EAS systems and to avoid both lingering within the surveillance gates and direct contact with the gates. Although data are lacking, it is likely that the same considerations and cautions apply to the pacing-function components of ICDs.

**Medical Sources of EMI**

Pacemakers and ICDs should be interrogated by a specialist before transthoracic cardioversion, if possible, to determine whether the patient is pacemaker dependent, to determine the rate and stability of the intrinsic cardiac rhythm, and to program off rate-adaptive capabilities because the latter may affect pacemaker behavior if the patient becomes agitated during sedation. Surface cardioverting electrodes should be positioned in the anteroposterior position more than 5 cm from the implanted device, and transcutaneous pacing should be available. After cardioversion, complete analysis of the implanted pacemaker or ICD should be performed to ensure that no damage to the system occurred. The same evaluation needs to be undertaken after defibrillation, recognizing that device testing before defibrillation is not possible in emergency situations.

Pacemakers and ICDs should also be evaluated before any operation in which electrocautery is used, to determine whether the patient is pacemaker dependent, to define the backup rate and mode of function of the device, and to program off rate-adaptive capabilities or other special algorithms. Since ICDs are designed to detect rapid, low-amplitude electrical activity (eg, ventricular fibrillation), tachycardia-detection functions must be turned off before any cautery (unipolar or bipolar) is used. For patients with either a pacemaker or an ICD, bipolar cautery should be used, if possible, to minimize any chance of cautery-device interaction. If unipolar cautery is used, the indifferent electrode should be positioned as close to the surgical field and as far away from the pacing system as possible. Good contact of the indifferent cautery electrode must be maintained throughout the procedure, and electrocautery times should be kept to a minimum. Electrocardiographic (EGC) monitoring is mandatory, taking care that the ECG tracings are free of noise that might obscure the P-QRS complexes of the patient’s na-
Pacemakers and ICDs should be interrogated before radiation therapy. Direct irradiation must be avoided. If this is not possible, the implanted device should be relocated to a new site. Even if outside the direct irradiation field, the pacemaker must be shielded, and cumulative dose to the generator must be monitored, with the total cumulative dose to the device less than 2 gauss. Electrocardiographic monitoring is necessary during treatment to detect any inappropriate device function. After radiation therapy is complete, the device system should be tested frequently for several months and replacement considered as indicated.

As with the above procedures, pacemakers and ICDs should be interrogated before lithotripsy. Pacemaker dependence should be determined. Rate-adaptive capabilities should be turned off, because vibration from the lithotriptor can affect the rate response of either pacemakers or ICDs that have rate-responsive capabilities. To decrease the possibility of damage to the pacemaker or ICD, the distance between the lithotriptor focal point and pulse generator should be maximized. This is especially important for ICDs implanted in the abdomen because of their proximity to the lithotripsy field. During the procedure, continuous ECG monitoring is advisable. Complete analysis of the implanted device should be performed after the procedure is completed.

Magnetic Resonance Imaging

Magnetic resonance imaging is performed by producing a static magnetic field, followed by application of rapidly varying magnetic and electromagnetic radiofrequency fields. Pacing system and ICD behavior are affected by all 3 components.

Static magnetic fields affect the reed switch of pacemakers, closing them in most pacemakers, resulting in pacemaker function in the magnet (asynchronous) mode and rate, which varies among pacemaker manufacturers. Some pacemakers allow the physician to turn off the magnet response to reed switch closure. Asynchronous pacing poses little problem in otherwise stable patients. The static magnetic field may also exert a torquing effect on the pacemaker generator, imposing a significant rotational force on the generator. However, no significant physical pulse generator movement has been reported in newer pacemakers that do not use large amounts of ferromagnetic material in their battery construction.

Inappropriate pacemaker function may be induced by the alternating magnetic field and rapid radiofrequency pulses emitted during the scan; for example, rapid pacing has occurred in unipolar systems exposed to the pulsing radiofrequency field due to the “antenna” effect of the electrode system.

The safety of magnetic resonance imaging in patients with implanted pacemakers and ICDs has been debated for years. In general, the presence of these devices has been considered to be an absolute contraindication to the performance of magnetic resonance imaging, because rapid cardiac pacing and total inhibition of output can occur during magnetic resonance exposure. It has recently been suggested that if patients are positioned so that the thorax does not enter the magnet bore, no significant interaction occurs; however, these data must be confirmed before magnetic resonance imaging, even of the extremities, can be allowed in patients with implanted devices.

All patients with implanted rhythm devices who are scheduled to undergo any medical procedures in which EMI is recognized, or likely to exist, should be examined before and after the procedure by an electrophysiologist or pacemaker specialist.

### METABOLIC AND DRUG EFFECTS ON CARDIAC PACEMAKERS

Myocardial capture thresholds (the minimal output energy of the pulse generator stimulus that is needed to depolarize myocardial tissue) are affected by metabolic factors (Table 1); by pharmacologic agents, notably the antiarrhythmic drugs (Table 2); by variables related to the pacing lead itself; and by the interface between the lead and the myocardium.

---

**Table 1. Metabolic Factors and Clinical States That Can Cause an Increase in Myocardial Capture Threshold**

<table>
<thead>
<tr>
<th>Factor</th>
<th>Effect on Threshold</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acidosis (by up to 80%)</td>
<td>Increase threshold</td>
</tr>
<tr>
<td>Alkalosis (by up to 70%-80%)</td>
<td>Increase threshold</td>
</tr>
<tr>
<td>Hypercalcemia (≥7 mmol/L)</td>
<td>Decrease threshold</td>
</tr>
<tr>
<td>Severe hyperglycemia (&gt;33.3 mmol/L) (≥600 mg/dL)</td>
<td>Possibly increase threshold</td>
</tr>
<tr>
<td>Hypoaemia</td>
<td>Decrease threshold</td>
</tr>
<tr>
<td>Myocardial infarction</td>
<td>Decrease threshold</td>
</tr>
<tr>
<td>Myocardial ischemia</td>
<td>Decrease threshold</td>
</tr>
<tr>
<td>Myxedema (by up to 30%-40%)</td>
<td>Decrease threshold</td>
</tr>
<tr>
<td>Eating (by up to 40%)</td>
<td>Decrease threshold</td>
</tr>
</tbody>
</table>

**Table 2. Effect of Some Cardioactive Drugs on Myocardial Capture Thresholds**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Effect on Threshold</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increase threshold</td>
<td></td>
</tr>
<tr>
<td>Flecainide acetate</td>
<td></td>
</tr>
<tr>
<td>Propafenone hydrochloride</td>
<td></td>
</tr>
<tr>
<td>Bretylium tosylate</td>
<td></td>
</tr>
<tr>
<td>Amiodarone hydrochloride</td>
<td></td>
</tr>
<tr>
<td>Possibly increase threshold</td>
<td></td>
</tr>
<tr>
<td>Lidocaine hydrochloride</td>
<td></td>
</tr>
<tr>
<td>Proacainamide hydrochloride</td>
<td></td>
</tr>
<tr>
<td>Quindine sulfate and gluconate</td>
<td></td>
</tr>
<tr>
<td>Decrease threshold</td>
<td></td>
</tr>
<tr>
<td>Atropine sulfate</td>
<td></td>
</tr>
<tr>
<td>Epinephrine hydrochloride</td>
<td></td>
</tr>
<tr>
<td>Isoproterenol hydrochloride</td>
<td></td>
</tr>
<tr>
<td>Corticosteroids</td>
<td></td>
</tr>
<tr>
<td>No significant effect on threshold</td>
<td></td>
</tr>
<tr>
<td>Digitalis glycosides</td>
<td></td>
</tr>
<tr>
<td>β-Blockers</td>
<td></td>
</tr>
<tr>
<td>Angiotensin-converting enzyme inhibitors</td>
<td></td>
</tr>
</tbody>
</table>

*Based on observations and reports before the 1990s.*
Most observations of changes in capture threshold caused by metabolic and drug effects were made in the 1960s to 1980s, and their relevance to today’s lead and electrode designs (especially corticosteroid-eluting and high-impedance leads) is not confirmed. However, because a goal of pacing system design is to provide low-threshold leads, it remains possible that under certain clinical circumstances a programmed low voltage will be insufficient to produce consistent pacing.

**Metabolic Effects on Myocardial Stimulation**

Capture thresholds can increase during sleep or eating, when sympathetic tone is low and vagal tone is increased. In contrast, an increase in sympathetic tone, such as during exercise, is associated with a decrease in capture threshold. Metabolic abnormalities that can increase capture threshold include acidosis, alkalosis, hypoxemia, and hypercarbia. Consistent capture is often not achieved until oxygen saturation and acid-base balance have been restored toward normal. Whereas acute myocardial infarction (especially involving the right ventricle, in pacing systems in which the ventricular lead is placed in the right ventricle) can increase capture thresholds significantly, myocardial ischemia produces variable effects on stimulation threshold, depending on the location of the pacing electrode relative to the ischemic myocardium. Loss of capture during acute ischemia is rare.

Hyperkalemia increases myocardial capture threshold when the serum potassium concentration equals or exceeds 7.0 mmol/L. Loss of atrial capture often precedes loss of ventricular capture because of the differential effects of hyperkalemia on atrial and ventricular myocardium. The reduced muscle excitability caused by the hyperkalemia can be corrected by the intravenous administration of calcium gluconate. Hyperglycemia in the range of 33.3 mmol/L (600 mg/dL) can increase capture thresholds by as much as 60%. Thus, patients with renal failure or diabetes, in whom there is a continuing risk for electrolyte abnormalities or abnormal glucose metabolism, should be provided a larger safety margin of pacemaker output voltage than usually recommended (twice capture threshold). Hypothyroidism can increase pacing thresholds, an effect that is reversible with thyroxine replacement.

Glucocorticosteroids can decrease capture thresholds and have even been used to treat failure to capture, although their effects are variable and unstable over time. Endogenous and synthetic catecholamines can lower pacing thresholds. Intravenous and sublingual isoproterenol can reverse high capture thresholds caused by antiarrhythmic drugs. These therapies have been used in the critical-care setting only and are insufficiently reliable to be used in the outpatient setting.

Although the effects of acid-base and electrolyte abnormalities on myocardial capture thresholds are potentially life threatening, their effects on the quality and integrity of the atrial and ventricular intracardiac signal can also be substantial, leading to undersensing and inappropriate delivery of pacing stimuli. Ventricular undersensing may be especially problematic during acute myocardial infarction, where inappropriate stimulus delivery on the T-wave of a spontaneous QRS complex can cause ventricular tachycardia or fibrillation. Fortunately, with current pacing system design features aimed at sensing poor intracardiac signals, and in today’s medical environment of early aggressive therapy in acute coronary syndromes including myocardial infarction, this problem appears to be rare.

**Drug Effects on Cardiac Pacemakers: Antiarrhythmic Drugs**

Several antiarrhythmic drugs have been demonstrated to increase myocardial stimulation thresholds, although most reports are clinical observations rather than systematically performed studies (Table 2). Class I antiarrhythmic drugs, which decrease sodium conductance and the rate of rise of the action potential, can increase the pacing threshold, especially when administered in high doses or when associated with high plasma levels. Class 1c drugs (particularly flecainide acetate) have been associated with increased capture thresholds, which correlate with the change in QRS duration. Flecainide has been reported to increase the myocardial capture threshold both in the short term after intravenous administration and in the long term during oral administration. The most significant rise in pacing threshold occurs during long-term oral therapy and appears to be dose related. The effects of long-term oral flecainide on the pacing threshold dissipate about 10 days after discontinuation of the medication. Flecainide is probably best avoided in patients with cardiac pacemakers, particularly if they are pacemaker dependent. Clinical experience suggests that oral amiodarone hydrochloride can be a common cause of increase in myocardial stimulation threshold, leading to non-capture even at high pacemaker output settings, in up to 10% of patients. This effect may be dose related and may not respond immediately to reduction in dose or drug discontinuation because of the long (weeks or months) half-life of this agent. Intravenously administered propranolol has been shown to increase pacing threshold, although not to clinically important degrees. Verapamil, digoxin, and lidocaine hydrochloride have minimal effects on both short- and long-term pacing thresholds. However, when lidocaine has been used in patients receiving other antiarrhythmic therapy, failure to capture has been reported to occur. Increase in capture thresholds with other cardioactive agents, such as angiotensin-converting enzyme inhibitors and angiotensin II receptor blockers, has not been observed, but clinical studies are unavailable.

Management of failure to sense or capture is based on programming of appropriate voltage outputs and sensing parameters by a pacemaker specialist. Consultation with a pacemaker specialist before initiating antiarrhythmic drug therapy may be warranted, depending on the therapy chosen, presence or absence of structural heart disease, and baseline and frequent threshold testing scheduled, especially in pacemaker-dependent patients.

Some currently available pacing systems use a myocardial cap-
macologic agents, especially antiar-
tients with ICDs and prescribing 
these devices will be caring for pa-
sicians with limited experience with 
growth in the use of ICDs, more phy-
ascertained. 
clinical significance, can thereby be 
changes, and thus their potential 
magnitude of threshold 
ture thresholds, which can then be 
turing changes in myocardial cap-
cmatic feature can be of use in docu-
old measurements over time are 
equate” safety margin. These thresh-
daily and voltage outputs automati-
cally measured once or several times 
stimulation thresholds are automati-
ture verification feature in which 
should be followed by arrhythmia in-
dia, therefore, in many instances 
in patients with ventricular tachycar-
tion of these membrane-active agents 
untreated by device therapy. Initia-
tion of these membrane-active agents 
in patients with ventricular tachycar-
dia, therefore, in many instances 
be cloned by arrhythmia in-
duction in the electrophysiology labo-
ary to assess detection by the im-
ated device.18-20 
Drug Effects on Defibrillators 
Because of the recent dramatic 
growth in the use of ICDs, more phy-
sicians with limited experience with 
these devices will be caring for pa-
tients with ICDs and prescribing 
medical therapy. Since some phar-
mologic agents, especially antiar-
rhythmic drugs,18-30 can signifi-
cantly influence device function and 
efficacy, awareness of drug-device in-
tractions is important. In most 
cases, consultation with an electro-
physiologist is indicated, because 
ICD testing and even arrhythmia in-
duction studies are often required. 
The purpose of the arrhythmia in-
duction study is to test the rates of 
duced arrhythmias and the reli-
ability and efficacy of arrhythmia 
detection and termination (antitachy-
cardia pacing or defibrillation). 
Potentially life-threatening drug-
device interactions are associated 
predominantly with the use of mem-
brane-active antiarrhythmic agents 
(class I or III drugs [Table 3]). These 
agents are often used in patients 
with ICDs to treat supraventricular 
arrhythmias that might otherwise, 
because of their rapid rates, lead to 
inappropriate shock therapy; to 
suppress ventricular tachyarrhyth-
ias, thereby preventing excessive, al-
beight appropriate, shocks; and to slow 
the rate of ventricular tachyarrrhyth-
ias to mitigate their hemodynamic 
effects and to increase responsive-
ness to antitachycardia pacing, 
thereby avoiding the necessity for 
shocks.

Sensing and Detection 
All currently available ICDs use heart 
rates as the primary discriminator be-
tween arrhythmias that require 
therapy and rhythms such as sinus 
tachycardia and supraventricular 
tachycardias that do not. Membrane-
active antiarrhythmic drugs slow the 
rate of ventricular tachycardia. If the 
ventricular tachycardia rate is slowed 
below the programmed detection cut-
off rate of the device, the arrhythmia 
will not be detected and will remain 
antent by device therapy. Initia-
tion of these membrane-active agents 
in patients with ventricular tachycar-
dia, therefore, in many instances 
should be followed by arrhythmia in-
duction in the electrophysiology labo-
atory to assess detection by the im-
anted device.18-20

To improve the specificity of ar-
rhythmia detection and avoid inap-
propriate delivery of shocks during 
sinus tachycardia, atrial fibrilla-
tion, or other supraventricular ar-
rhythmias, many ICDs use program-
mable therapy “inhibitors.” When 
activated, these inhibitors prevent 
delivery of shock therapy despite a 
heart rate in the tachycardia zone, 
if other factors suggest a supraven-
tricular mechanism. Some devices 
analyze the width or morphologic 
characteristics of the intracardiac 
electrical signal (electrogram) to dis-
tinguish wide-complex (ventricular) 
from narrow-complex (supra-
ventricular) tachycardia. Sodium 
channel–blocking drugs (espe-
cially class Ic agents; Table 3) in-
crease the QRS width, which may 
lead to rhythm misclassification by 
the device and inappropriate deliv-
er of therapy. Rarely, pharma-
logic effects on the caliber (voltage 
and rate of change of voltage [slew]) 
of the electrogram may adversely 
affection of the intracardiac sig-
als and impede arrhythmia detec-
tion. Also rarely, drug-induced QT-
interval prolongation (especially by 
class Ia or class III drugs) may lead 
to “double counting” (sensing by the 
ICD of both QRS complexes and T 
waves), resulting in the device’s de-
tecting a rate that is twice the ac-
tual heart rate, with consequent in-
appropriate delivery of therapy.

Pacing Function 
Since all current ICDs also provide 
pacing capability to treat brady-
cardia, the effects of drugs on pacemak-
ers (discussed above) also apply to 
these devices. Drug-device interac-
tions unique to the pacing function 
of the ICD also exist. In the anti-
tachycardia pacing function of the 
ICD, short pacing bursts at rates 
slightly greater than the tachycar-
dia rate can terminate up to 95% of 
episodes of ventricular tachycar-
dia, avoiding the need for delivery 
of a shock. Class Ic agents exhibit 
“use dependency,” in which their ef-
fects are amplified at higher heart 
rates; thus, any elevation in pacing 
threshold caused by these agents 
may be more pronounced during an-
titachycardia pacing than during 
standard pacing for bradycardia, po-
tentially reducing the efficacy of the 
antitachycardia pacing therapy.

Defibrillation 
The most potentially dangerous ef-
effect of drugs on ICD function is an 
alteration in defibrillation shock ef-
ficacy, which could result in failure 
of defibrillation. Although it is well 
known that pharmacologic agents 
can modulate defibrillation effec-
tiveness, drug-defibrillation inter-
actions are complex. Moreover, rec-
ommendations based on review of 
the literature on drug influence on 
defibrillation are confounded by the 
effects of anesthetic agents; hetero-
ogeneity in defibrillator waveform and 
lead type, and variability in the mod-
els in which the interactions are 
tested (eg, human vs canine vs 

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Table 3. Membrane-Active Drugs That May Significantly Affect Defibrillator Function

<table>
<thead>
<tr>
<th>Class</th>
<th>Drugs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ia</td>
<td>Quinidine sulfate and gluconate, procainamide hydrochloride, disopyramide phosphate</td>
</tr>
<tr>
<td>Ib</td>
<td>Lidocaine hydrochloride, phenytoin sodium, mexiletine hydrochloride</td>
</tr>
<tr>
<td>Ic</td>
<td>Flecaidine acetate, propafenone hydrochloride, moricizine hydrochloride</td>
</tr>
<tr>
<td>III</td>
<td>Sotalol hydrochloride, ibutilide, fumarate, dofetilide, amiodarone hydrochloride</td>
</tr>
</tbody>
</table>

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Table 4. Important Interactions Between Medications and Implanted Defibrillators

<table>
<thead>
<tr>
<th>ICD Function</th>
<th>Potential Medication Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arrhythmia detection</td>
<td>Diminished slew (rate of change of electrogram voltage)</td>
</tr>
<tr>
<td></td>
<td>Slowing of VT rate below cutoff rate for detection</td>
</tr>
<tr>
<td></td>
<td>QRS widening, altering the criteria for ventricular arrhythmia</td>
</tr>
<tr>
<td></td>
<td>recognition</td>
</tr>
<tr>
<td>Pacing</td>
<td>Increase in capture threshold</td>
</tr>
<tr>
<td></td>
<td>Increase in capture threshold at rapid pacing rates (“use</td>
</tr>
<tr>
<td></td>
<td>dependency”)</td>
</tr>
<tr>
<td>Defibrillation</td>
<td>Induction of atrial bradycardia and/or AV block, necessitating</td>
</tr>
<tr>
<td></td>
<td>antiarrhythmic pacing</td>
</tr>
<tr>
<td></td>
<td>Production of proarrhythmia, leading to increased shock</td>
</tr>
<tr>
<td></td>
<td>frequency</td>
</tr>
<tr>
<td></td>
<td>Increase in defibrillation threshold</td>
</tr>
<tr>
<td></td>
<td>Decrease in defibrillation threshold</td>
</tr>
</tbody>
</table>

*ICD indicates implantable cardioverter-defibrillator; VT, ventricular tachycardia; and AV, atrioventricular.

In general, agents that impede the fast inward sodium current (such as lidocaine and possibly mexiletine hydrochloride21) tend to increase the defibrillation threshold, whereas agents that block repolarizing potassium currents (such as sotalol hydrochloride and ibutilide fumarate) lower the defibrillation threshold and are attractive drug choices when antiarrhythmic agents are needed in patients with ICDs. Recent advances in defibrillator technology, including the use of biphasic waveforms and incorporation of the pulse generator can as an active lead, have improved defibrillation efficacy, mitigating these adverse pharmacologic effects. However, oral amiodarone, a frequently used antiarrhythmic agent, warrants special mention, since its use may result in an increase in defibrillation thresholds to a clinically relevant degree, even in modern systems.

In view of these important device-drug interactions (Table 4), when agents associated with them are initiated or their doses changed, referral to an electrophysiologist for defibrillator testing and subsequent follow-up is indicated.

Beneficial Medications

Given the prevalence of coronary artery disease and left ventricular dysfunction in patients with ICDs, it is important to note that medications used to treat these conditions (including β-adrenergic blocking agents, angiotensin-converting enzyme inhibitors, angiotensin-receptor antagonists, diuretics, digitalis, aspirin, and warfarin sodium) do not pose a risk to these patients. Indeed, since the rationale for ICD use is to prolong survival, all patients with ICDs and concomitant cardiovascular disease should be treated with these drugs that have independently been shown to reduce mortality.