Adverse Acute and Chronic Effects of Electrical Defibrillation and Cardioversion on Implanted Unipolar Cardiac Pacing Systems

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Six cases are presented in which a transient or chronic rise in the stimulation threshold of a permanently implanted unipolar pacemaker resulted in the loss of effective pacing after therapeutic defibrillation or cardioversion. Although damage to the pulse generator may still occur, leading to a loss of function as demonstrated in a seventh patient, improvements in the internal protection circuits of the present generation of pacemakers makes this less likely while possibly predisposing to endocardial burns and increased fibrosis at the electrode-endocardial interface. The theoretical explanations for this phenomenon are discussed, along with recommendations for the prospective and retrospective management of the pacemaker patient who requires defibrillation or cardioversion.

The therapeutic application of electricity to medicine continues to grow, particularly with respect to the management of electrical dysfunction of the heart. Electrical cardioversion and defibrillation are valuable tools used to convert a tachycardia to a normal rhythm while cardiac pacing is designed to manage symptomatic bradycardias. Furthermore, cardiac pacing is being used on an investigational basis to treat a multiplicity of tachyarrhythmias. Improvements in circuit design and reliability of the pulse generator, power source longevity, ease of implantation, programmability and size are some of the factors contributing to the growing acceptance of cardiac pacing as a therapeutic and, at times, a prophylactic tool. As such, there is a large and growing number of patients with an implanted pacemaker who implicitly have some form of heart disease and will possibly require elective cardioversion or emergent defibrillation in the future.

Sporadic case reports (1–4) have attested to the potentially adverse interaction between cardiac pacing and external defibrillation. Most reports cited damage to the pulse generator while one (5) showed damage to both the pulse generator and the heart from the interaction of these two modalities of electrotherapy.

To reduce the potential for damage to the pacemaker, manufacturers have incorporated protective circuits designed to shield the complex electronics from the massive amounts of energy delivered to the body by the defibrillator (6). These circuits are extremely effective in the majority of patients with the pulse generator continuing to function in accord with its specifications after defibrillation, thus protecting the patient from asystole. Some patients may be less fortunate. The protective circuit of the pacemaker shunts the excessive amounts of energy delivered by the defibrillator away from the internal electronics of the pulse generator to the pacing electrode. This may engender additional and more difficult problems to manage. The lead and electrode constitute a relatively low resistance pathway in intimate contact with the myocardium. Large amounts of energy shunted to it may induce endocardial burns with both acute and chronic rises in stimulation threshold leading to a transient or chronic loss of effective pacing. This has been demonstrated in animals by Taube et al. (7) and others (8), but alluded to in human patients in only one case (5).

In this paper, we present seven cases (Table I) demonstrating the spectrum of effects encountered in patients with a permanent unipolar pacemaker subjected to therapeutic defibrillation or cardioversion. Management of the patient, choice of the pacing system, implantation techniques and considerations for defibrillation protocols are discussed with respect to these cases.
Table 1. Clinical Summary

<table>
<thead>
<tr>
<th>Case</th>
<th>Age (yr)/Sex</th>
<th>Diagnosis</th>
<th>Rhythm</th>
<th>Energy Dose in Joules (J)</th>
<th>Pacing System Malfunction (Acute)</th>
<th>Pacing System Function (Chronic)</th>
<th>Pulse Generator Function</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>64M</td>
<td>CAD</td>
<td>V. Fib</td>
<td>320 (DE)</td>
<td>LOC for 90 s</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td>2</td>
<td>81F</td>
<td>CAD</td>
<td>V. Fib</td>
<td>320 (DE)</td>
<td>LOC for 12 s</td>
<td>Decreased</td>
<td>Normal</td>
</tr>
<tr>
<td>3</td>
<td>53F</td>
<td>Stokes-Adams</td>
<td>A. Flutter</td>
<td>30, 150-300 (SE)</td>
<td>LOC for 3 min</td>
<td>Normal</td>
<td>Battery depletion 1 yr later</td>
</tr>
<tr>
<td>4</td>
<td>53F</td>
<td>CHF</td>
<td>V. Tach</td>
<td>400 (SE) × 6</td>
<td>LOC for 30-90 s each time</td>
<td>Decreased</td>
<td>Normal</td>
</tr>
<tr>
<td>5</td>
<td>64M</td>
<td>CAD, CHF</td>
<td>V. Tach</td>
<td>100 (DE)</td>
<td>LOC for 2 s</td>
<td>Increase in stimulation threshold</td>
<td>Normal</td>
</tr>
<tr>
<td>6</td>
<td>41M</td>
<td>Stokes-Adams</td>
<td>A. Fib, WPW</td>
<td>160, 300 (SE)</td>
<td>LOC for 3 min</td>
<td>Normal</td>
<td>Battery depletion 2 mo later</td>
</tr>
<tr>
<td>7</td>
<td>64F</td>
<td>Stokes-Adams</td>
<td>V. Fib</td>
<td>320 (DE)</td>
<td>Persistent LOC</td>
<td>Persistent LOC</td>
<td>Damage to integrated circuits</td>
</tr>
</tbody>
</table>

A. Fib = atrial fibrillation; A. Flutter = atrial flutter; CAD = coronary artery disease; CHF = congestive heart failure; DE = delivered energy; F = female; LOC = loss of capture; M = male; NA = not available; SE = stored energy; V. Fib = ventricular fibrillation; V. Tach = ventricular tachycardia; WPW = Wolff-Parkinson-White syndrome.

Case Reports

Case 1

A 64 year old man received a Cordis Omni-Stanisor (VVI,P) unipolar pulse generator (Cordis Corporation) for intermittent complete atrioventricular (AV) block 2 years previously. During the intervening period, sensing and capture were both normal and pacing rate was stable. Noninvasive evaluation of his chronic stimulation threshold had not been performed before his admission with an acute myocardial infarction. At that time, he was pacemaker-dependent, and noninvasive manipulation of the output parameters of the pacemaker was not undertaken. His hospital course was complicated by ventricular fibrillation (Fig. 1) with cardiopulmonary resuscitation initiated promptly. Defibrillation was achieved using 320 joules delivered energy within 30 seconds of development of ventricular fibrillation. The pulse generator was located in the right pectoral fossa. The defibrillator paddles had been placed in an anterior-anterior position, one located at the upper right sternal border, near but not directly over the pulse generator, and the second placed over the cardiac apex. For a period of 90 seconds after defibrillation, external cardiopulmonary resuscitation had to be maintained before a stable rhythm could be reestablished and normal pacing function demonstrated. Subsequent evaluation showed normal pacemaker function. No attempt was made to assess the chronic stimulation threshold after the successful resuscitation. The patient survived the acute infarction and was subsequently lost to follow-up.

Case 2

An 81 year old woman had a Cordis Ectocor unipolar ventricular triggered pulse generator (VVT) (Cordis Corporation) inserted for chronic complete heart block. Ten months later, she was admitted with an acute myocardial infarction complicated by ventricular fibrillation (Fig. 2). Defibrillation was expeditiously achieved with a delivered energy of 320 joules; however, the rhythm reverted to asystole of 12 seconds’ duration before normal capture occurred. The pulse generator had been located in the right pectoral fossa and the defibrillator paddles applied in an anterior-anterior orientation. The pacemaker functioned normally for the remainder of the patient’s hospitalization; however, she developed cardiogenic shock and died from her infarction. The pulse generator could not be retrieved for subsequent analysis.

Case 3

A 53 year old woman had a bipolar epicardial VVI pace­maker inserted 7 years previously for intermittent trifascicular block. The pulse generator was located in the epigas­trium superficial to the rectus muscle. Two subsequent generator replacements were uncomplicated, the chronic stimulation thresholds being 1.5 and 1.8 mA, respectively. On the latter occasion, the system was electively converted to unipolar pacing with the unused lead capped and left in situ. The pulse generator was a Medtronic model 5945.

Three years after this generator change, the patient was admitted with atrial flutter requiring cardioversion. An initial 30 joule synchronized shock did not convert the flutter (Fig. 3A). A second attempt using 150 joules proved unsuccessful in altering the flutter, but was followed by pacing system malfunction characterized by loss of capture with no change in the interval between consecutive pacing stimuli. High grade AV block was present with a junctional escape rhythm of 47 beats/min (Fig. 3B). A third attempt
Figure 1. Case 1. Serial electrocardiographic tracings. When charged to 400 watt-seconds, the defibrillator delivered 320 joules (arrow). In addition to loss of capture, failure to sense the single native ventricular beat occurring in rhythm strip B is shown. Rhythm strips are not continuous, but recorded sequentially over a period of 90 seconds before effective and normal pacing resumed.

Figure 2. Case 2. Continuous pacing with loss of capture lasting for 12 seconds is shown in this pacemaker-dependent patient after emergent defibrillation. The delivered energy was 320 joules (arrow).

at cardioversion with 300 joules was also ineffective with persistent pacing system failure. Within 1 minute of the last attempt at cardioversion, the rhythm spontaneously converted to sinus but with persistent high grade AV block. Loss of capture persisted for 3 minutes before normal pacing function resumed.

One year later, the pulse generator was replaced because of battery depletion, which was appropriate for this device that had been implanted for approximately 4 years. At this time, the chronic stimulation threshold measured 4.0 mA, indicating that a major rise in the chronic stimulation
Case 4

A 63 year old woman had an implanted nonprogrammable VVI pacemaker for several years for the treatment of supraventricular tachycardias due to the Wolff-Parkinson-White (type B) syndrome, with the tachycardia interrupted by magnet application to the pacemaker. When admitted to the hospital for treatment of congestive heart failure, she was noted to have pacemaker failure evidence by a prolongation of the automatic interval, and the pulse generator was replaced with a Siemens 668 (VVI,M) unipolar pacemaker (Siemens-Elema Corporation). The pacemaker was programmed to the hysteresis mode (escape interval longer than automatic interval) and the refractory period was programmed to 437 ms to enable it to function in a dual demand mode (9). When programmed in this manner, the pacemaker reverts to its asynchronous rate in the presence of a tachycardia of 137 beats/min or faster.

The patient later developed recurrent ventricular tachycardia unrelated to pacing or delivery of pacing stimuli in the vulnerable zone during either sinus rhythm or an episode of tachyarrhythmia. Ventricular tachycardia was poorly tolerated and immediate defibrillation was performed on six separate occasions, each time within 20 to 30 seconds after the onset of the tachycardia. After each episode, there was a transient loss of pacemaker capture for 30 to 90 seconds (Fig. 4). With use of the pacemaker’s Vario function, the threshold was assessed noninvasively when the patient was in stable condition, and found to be greater than 2.45 V and less than 2.8 V at 2 minutes after defibrillation. The patient subsequently died from congestive heart failure and the pulse generator was retrieved. Electrical analysis showed the unit to be functioning within specifications.

Case 5

A 64 year old man with chronic bifascicular block, intermittent AV block, complex ventricular arrhythmias and congestive cardiomyopathy had a Pacesetter Programalith (VVI,P) unipolar pulse generator (Pacesetter Systems, Inc.) implanted in the right pectoral fossa. Noninvasive threshold measurements demonstrated the expected acute rise and then fall in the stimulation threshold within 2 weeks after implantation (Fig. 5). Shortly after discharge, the patient was readmitted to the hospital with sustained ventricular tachycardia from which he was successfully cardioverted with...
100 joules delivered energy using an anterior-anterior position for the cardioversion paddles. Stimulation threshold measurements were not obtained immediately but in the early postconversion period, the few pacemaker stimuli present before the native rate accelerated were ineffective. Assessment of the chronic stimulation threshold 3 days later demonstrated that it had almost doubled compared with the predischarge measurements before cardioversion. It remained stable at that level for the next few weeks (Fig. 5) and for months thereafter.

Case 6

A 41 year old man was treated with a Cordis Omni Stanicor (VVI,P) unipolar pulse generator for intermittent complete heart block. The pulse generator was located in the right pectoral fossa and periodic stimulation threshold measurements were made by programming both the rate and output. The stimulation threshold was consistently below the lowest output of the pulse generator as capture was maintained at the test setting, which is approximately 1.5 mA, and at a rate of 100 pulses/min. The pacemaker was set at the low setting (approximately 3 mA) and was functioning normally 4.5 years after implantation. The patient subsequently developed atrial fibrillation and cardioversion was accomplished with an anterior-anterior paddle orientation using two shocks, the first of 160 joules and the second of 300 joules delivered energy. Transient loss of capture persisted for 3 minutes (Fig. 6) after the second delivered shock. After the episode of loss of capture, the output was programmed to the high setting of approximately 8 mA.

Figure 4. Case 4. Serial tracings showing transient loss of capture after defibrillation (DEFIB.) with intermittent capture first demonstrated in the supernormal zone of the native beats (rhythm strip D). Sensing function is normal as the refractory period of this pacemaker was programmed to 437 ms. E. Demonstration of the stimulation threshold testing using the Vario function of this pacemaker. This is initiated by placing a test magnet over the implanted pulse generator. This starts a test cycle with two phases repeating themselves as long as the magnet remains in place. The first part of the test cycle consists of 16 impulses at the magnetic rate of 100 pulses/min delivered at the full output (5 V) of the pulse generator. At the end of the 16 impulses, the Vario mechanism comes into play at a rate slightly faster than the preceding magnetic rate. In a conventional 5 V pulse generator, the output voltage is decreased successively by approximately 0.35 V/step during the 16 impulses down to 0 amplitude (shown at 0). The threshold can then be calculated by counting the number of ineffective pacing stimuli from right to left. At the end of the test cycle, the pacemaker automatically returns to its programmed voltage output.
Cardioversion 100 joules

Figure 5. Case 5. A graph of the serial stimulation threshold measurements made with the Pacesetter Programalith system. After cardioversion, there is a significant rise in stimulation threshold, which remained stable at the higher level during the follow-up period. Immediate postcardioversion measurements were not obtained. (Reprinted with permission from Levine PA. Why Programmability; Indications for and Clinical Utility of Multiparameter Programmability. Sylmar, California: Pacesetter Systems, Inc., 1981.)

Figure 6. Case 6. Surface leads 1 and 2 and intracardiac leads from the high right atrium (HRA) and His bundle (HBE). The upper and lower tracings are recorded at different paper speeds. The upper tracing shows the successful cardioversion (arrow) of atrial fibrillation, but the resultant atrial mechanism is atrial standstill. The pacing stimuli designated by the open arrows on the bottom tracing were ineffective for a total period of approximately 3 minutes. The patient developed an idioventricular escape focus after cardioversion and had to be managed pharmacologically until effective pacing resumed.

Three months after discharge, the patient presented to another hospital with syncope and the pacemaker was shown to have both an increase in the pacing interval characteristic of battery depletion and intermittent failure to capture. A chest X-ray film showed the pacing electrode to be stable in the area of the right ventricular apex. The patient had the pulse generator replaced, at which time the capture threshold was 3.5 mA. This pulse generator was returned to the man-
manufacturer and, other than battery depletion, was functioning within specification. It is suspected that the increase in programmed output after loss of capture that resulted from the defibrillation induced the battery depletion of the pulse generator, which had been implanted for almost 5 years. The chronic stimulation threshold measurement made at the time of pulse generator replacement was significantly above that made noninvasively at the time of routine follow-up before the cardioversion intervention.

**Case 7**

A 64 year old woman was treated for intermittent complete heart block with an Arco Li3D (VVI) unipolar pulse generator (Arco Medical Products) placed in the right pectoral fossa. Thirty months after implantation, she was admitted to the hospital with diverticulitis complicated by a ruptured viscus and peritonitis. Persistent infection, metabolic derangement and poor wound healing were noted after an exploratory laparotomy. After 3 weeks in the hospital, she developed ventricular fibrillation, which was successfully converted after a single 320 joule delivered energy shock. In the immediate postdefibrillation period, ineffective pacing stimuli (Fig. 7) were noted with a measured pulse duration of 0.14 ms as compared with the predi­brillation measurement of 0.9 ms. The pacing rate, which was originally 72 pulses/min, also became unstable. It was 100 pulses/min immediately after defibrillation, finally slowing to the range of 80/min. When the patient died, the pulse generator was retrieved and returned to the manufacturer for analysis. Damage to two integrated circuits in the output oscillator and sensing amplifier was identified and attributed to an inadequate protective circuit.

**Discussion**

Seven patients are presented in whom transient acute loss of capture or sensing, or both, and a chronic increase in stimulation threshold occurred after electrical cardioversion or defibrillation. In only one case could the loss of capture be attributed to direct damage to the pacemaker itself. In the remaining six patients, transient loss of capture occurred immediately after the procedure. These observations underscore the importance of being ready for the treatment of asystole in this situation. In addition, three patients exhibited a late rise in threshold as assessed both noninvasively in one case and invasively in two others.

All the pulse generators, with the exception of that in Case 3, were located in the right pectoral fossa and all were unipolar. In each patient, cardioversion or defibrillation was accomplished by application of an anterior-anterior paddle orientation. This requires that one paddle be placed over the cardiac apex and the second at the upper right sternal border. Although not directly over the pulse generator, this placed one of the paddles in close proximity to the pacemaker in six of the patients, and also resulted in a dipole of current flow parallel to a portion of the endocardial lead in all patients.

**Figure 7.** Case 7. Serial tracings for ventricular fibrillation after defibrillation (arrow). Unlike the previous cases, there is an apparent change in the pacing interval in addition to the loss of capture after defibrillation, signifying concomitant damage to the pulse generator in addition to any rise in the stimulation threshold that might have occurred. Stimulation thresholds were never able to be measured in this patient antemortem. The pacemaker was explanted and pulse generator damage confirmed by the manufacturer.
Mechanisms for Post-Defibrillation Pacemaker Malfunction

A number of mechanisms exist that may explain the rise in stimulation threshold and transient loss of capture after electrical defibrillation or cardioversion. In one experimental study (10) evaluating defibrillation utilizing an endocardial pacing electrode as one of the electrodes for defibrillation, it was shown that pacing thresholds rose from a level of 1 to 3 mA before defibrillation to greater than 20 mA for the first few minutes after defibrillation. This would result in at least transient loss of capture in a permanent pacing system that delivers approximately 10 mA at the nominal output settings. Similar results may occur clinically in implanted pacing systems during cardioversion or defibrillation by either shunting current to the lead by way of a zener diode or capacitive coupling of energy directly to the lead and, hence, to the heart at the electrode-myocardial interface.

Activation of zener diode (shunting of energy to the endocardial pacing lead). Zener diodes within the pulse generator are designed to protect the unit during defibrillation so that it will continue to function normally after a large energy surge, thus protecting the patient who needs continuous pacing. The zener diode is a voltage regulator that allows large amounts of current flow through it while keeping the voltage across it fairly constant. In normal use, it may have a regulation voltage of 12 V. Because the pulse generator never emits a pulse that approaches 12 V, the diode is virtually inactive. When presented with a defibrillation pulse, the zener diode is activated to allow high currents to pass through it to limit the voltage across it. In unipolar pulse generators, the zener diode connects the lead and the housing of the pulse generator, as these are the active parts of the pacing circuit other than the internal electronics.

The housing of the pacemaker which comprises the indifferent electrode is in close proximity to one of the defibrillator paddles. Energy delivered during defibrillation will then be shunted by the zener diode through the lead to the electrode. Although this protects the pacemaker circuitry, the lead, being a low impedance system connected directly to the heart, shunts and concentrates the energy at the electrode-myocardial interface, resulting in burns and local electrical trauma which can induce an acute rise in the stimulation threshold.

This was demonstrated experimentally by Taube et al. (7) in dogs with chronically implanted unipolar pacing systems. The animals were subjected to either single low dose, single high dose or five low dose defibrillations. During these studies, one paddle was placed directly over the cardiac apex and the second was placed directly over the pulse generator. An acute rise in stimulation threshold, often above the output of the implanted pacemaker, occurred such that capture would be transiently lost after defibrillation. A chronic elevation of stimulation threshold was demonstrated 1 to 7 days postdefibrillation. All the animals were sacrificed and the heart subjected to both gross and histologic examination. Increased areas of fibrosis and apparent endocardial burns were demonstrated at the electrode-myocardial interface. On histologic sectioning, fibrosis beginning at the endocardial contact site of the electrode was shown to extend through the full thickness of the myocardium in many animals. The only correlation with this laboratory finding and the published clinical data is a report (11) of exit block due to low levels of continuous current drain in a series of malfunctioning pulse generators. In that report (11), 10% of the patients with the defective units experienced exit block, which was out of proportion to the authors' prior experience.

While external defibrillation has been reported to cause myocardial necrosis (12–15), the anatomic location of the endocardial and myocardial fibrosis in the animals with an implanted pacing system supports Taube's conclusion that the damaging energy was delivered to the heart through the pacing lead and not more diffusely through the thorax.

Capacitive coupling of energy to pacing lead and heart. A second explanation for the rise in threshold is capacitive coupling of the energy delivered by the defibrillator directly to the pacing lead and, thence, to the heart. The amount of energy delivered during defibrillation is large. The orientation of the paddles in an anterior-anterior position is generally parallel to a portion of the lead system whether the pulse generator is in the right or left pectoral fossa. Shepard et al. (16) noted an unacceptably high incidence of exit block in patients with a permanent pacemaker who underwent open heart surgery during which electrocautery was employed. Subsequent in vitro studies (16) were performed using a standard pacing lead consisting of a conductor coil surrounded by an outer insulating sheath of silastic rubber immersed in a saline solution with both the electrode and terminal pin outside this bath when electrocautery was delivered to the saline reservoir. By recording the energy at the electrode tip, the authors demonstrated induced currents of 160 mA and potentials of 140 V without contact between the electrocautery device and the pacing lead, the silastic insulation serving as a dielectric between the two. The ability to induce currents in the insulated conductor coil was attributed to capacitive coupling, thus showing that sufficient energy could be delivered to the heart at the electrode-myocardial interface. In addition, this amount of energy could result in microelectrocautery burns providing an explanation for the observed increases in stimulation threshold in their patients. Capacitive coupling occurred independent of the pulse generator and would be expected to occur with equal frequency in unipolar and bipolar pacing systems.

Displacement of pacing lead by the electrical shock. A third potential mechanism for a threshold increase is micro- or macrodisplacement of the lead as a direct result of the vigorous cardiac contraction engendered by the electrical shock. In an unpublished study, Taube (personal communication) found that lead displacement occurred after de-
fibrillation only if the tip was not adequately secured within the surrounding trabeculae during the acute implantation. In defibrillation tests with both myocardial and chronic transvenous leads, displacement did not occur. Microdislocation has been postulated clinically in only one case (17), but in that patient there was also damage to the pulse generator itself, raising questions as to the true mechanism for the loss of capture.

**Location of the pacemaker generator.** Seventy percent of pacemakers implanted in the United States are unipolar devices (18). Most implanted units are placed in the right pectoral fossa, as was the case in six of our seven patients. This sets the stage for transient acute and possibly late loss of capture as a direct consequence of cardioversion or defibrillation based on the shunting of energy acquired by the housing during defibrillation when an anterior-anterior paddle orientation is employed. In addition, the patient with an epicardial unipolar pulse generator had the apex defibrillator paddle placed in close proximity to that pulse generator. A similar problem, if due to capacitive coupling as described by Shepard et al. (16), would be independent of the polarity of the pacing system.

**Postcardioversion rise in stimulation threshold.** Three of our patients whose hearts were defibrillated in an emergency setting experienced a transient loss of capture. Except in Case 4, stimulation thresholds were not measured immediately postdefibrillation; and in this case, when measured, they were unchanged from predefibrillation levels. While all three patients were defibrillated before sufficient time elapsed to result in generalized metabolic, acid-base or ventilatory derangements, local changes in the myocardium with developing acidosis, hyperkalemia and other abnormalities (20,21) could have accounted for a transient threshold elevation independent of the proposed endocardial burn induced by either of these mechanisms. If this were the case, loss of capture would be expected to be transient without causing any chronic problems. However, while this explanation cannot be totally excluded in our patients, three of our six patients were cardioverted electively, two from atrial arrhythmias and one from ventricular tachycardia. In none of these patients were major acid-base or electrolyte derangements noted that might account for either the immediate or late rises in threshold that were demonstrated. These three cases (Cases 3, 5 and 6) are also consistent with Taube’s experience (7) showing a chronic threshold elevation after cardioversion, presumably due to increased fibrosis at the electrode tissue interface which could, theoretically, progress to the point of “exit block” with loss of capture.

**Guidelines for Management of the Pacemaker Patient**

**Location of defibrillation paddles.** With respect to unipolar pulse generators, the manufacturers recommend that defibrillator paddles be placed at least 5 inches (12.7 cm) from the pulse generator (22,23), a virtual impossibility with a pacemaker in the right pectoral fossa when using an anterior-anterior paddle orientation. Still, keeping the paddle as far from the pulse generator as possible and titrating the energy to permit use of the lowest effective energy level will minimize the chance of problems. If available, an anterior-posterior orientation for the defibrillator or cardioversion paddles should be employed. This latter system will also be effective in minimizing the chance of capacitive coupling as the electrical field will be perpendicular and not parallel to the course of the lead.

**Placement and type of pacemaker system.** In planning a primary implantation, particularly if a patient is prone to arrhythmias that might warrant cardioversion or defibrillation, placement of the pulse generator in the left pectoral fossa will serve to keep it as far from the defibrillator paddles as possible when only anterior-anterior cardioversion systems are available. Intentional choice of a bipolar system will further reduce the likelihood of problems if the major explanation was energy shunting from the housing of the pulse generator to the electrode by way of the zener diode, but not if capacitive coupling by means of the lead itself was the major site for energy entry (23).

**Assessment of stimulation threshold.** The advent of output programmable pacemakers permits the noninvasive assessment of the chronic stimulation threshold. As a direct result of this capability, rises of stimulation threshold after cardioversion and defibrillation can now be recognized. Serial measurements of stimulation threshold beginning hours after electrical defibrillation or cardioversion in patients with these units is recommended and should be made on a frequent basis for the next 2 months to screen for a significant rise in stimulation threshold. As demonstrated in Case 6, an increase in the delivered energy from the pulse generator to compensate for the rise in stimulation threshold may result in an abrupt abbreviation of the functional life of the system. Thus, if changes are made in the parameters of the pacemaker based on these considerations, careful follow-up is mandated to be sure that the system continues to function in accord with its design specifications.

**References**


