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Cardiac Rhythm Management Devices (Part I)

Indications, Device Selection, and Function

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PACEMAKER[1] and internal cardioverter–defibrillator (ICD) devices have undergone remarkable evolution since the first implantation of an asynchronous single-chamber pacemaker[1] in 1958 and of an ICD[2] in 1980. Today, more than 500,000 patients in the United States have pacemakers, and up to 115,000 new devices are implanted each year.[3] The number of ICDs implanted each year has steadily increased, reaching 50,000 new implants worldwide in 1999.[4]

Contemporary single- and dual-chamber pacemakers are sophisticated devices, with multiple programmable features, including recently introduced programmable lead configuration[5,6] and automatic mode-switching.[7–9] Many devices use adaptive-rate pacing to modify the pacing rate for changing metabolic needs. First-generation ICDs were short-lived. A formal thoracotomy was required for epicardial lead placement. Today, ICDs are multiprogrammable, are longer-lived, have transvenous leads, and may incorporate all capabilities of contemporary pacemakers.[4] Furthermore, ICDs have multiple tachycardia detection zones, with programmable detection criteria and tiered therapy (*i.e.*, antitachycardia pacing [ATP], followed by shocks if needed) for each.[4,10] ICDs also store dysrhythmia event records and treatment results. Finally, clinical experience with an internal atrial cardioverter (atrioverter) has been reported.[11–15]

In this first installment of a two-part communication, we discuss indications for implanted pacemakers or ICDs, provide an overview of how devices are selected, and describe the basics of device design and function. Only brief mention is made of temporary pacing indications and technology. In the second installment, we discuss the potential for device malfunction in the hospital environments, perioperative management for patients with implanted devices, and care of patients during device implantation or system revision.

This is the first part of a two-part article. Part II will appear in the December 2001 issue.

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Indications for a Pacemaker or an ICD

Indications for a pacing or ICD device are considered as class I, II, or III.[10] Class I indications are conditions for which there is general agreement that a device may be useful and effective (*i.e.*, is indicated). Class II indications are conditions in which a device is often used but for which there is conflicting evidence or divergence of opinion as to whether it is useful and effective (*i.e.*, may be indicated). Class II indications are subdivided as IIa if the weight of evidence or opinion is in favor of device usefulness or efficacy and IIb if usefulness or efficacy is less well established. Finally, an indication is class III if there is general agreement that a device is unnecessary and possibly even harmful (*i.e.*, not indicated).

Temporary Pacing Indications

Temporary pacing may be required for rate support in patients who experience intermittent hemodynamically disadvantageous bradydysrhythmias or for stand-by pacing in patients at increased risk for sudden high-degree atrioventricular (AV) heart block (AVHB). It is also sometimes used to overdrive or terminate atrial or ventricular tachydysrhythmias. The endpoint for temporary pacing is resolution of the indication or implantation of a permanent pacemaker for a continuing indication. Transvenous endocardial[16,17] or epicardial[16,18] leads are most commonly used for temporary pacing. Noninvasive transcutaneous and esophageal routes are also possible.[19–21] Transcutaneous pacing produces simultaneous ventricular and atrial capture and thus does not preserve optimal hemodynamics in patients with intact atrioventricular conduction. With available technology for esophageal pacing, only atrial capture is reliable; thus, the method is not suitable for patients with advanced AVHB or atrial fibrillation.

Indications for temporary pacing are not as established as for permanent pacemakers. Usual and less established indications for temporary transvenous or epicardial pacing are listed in table 1.[18,22–24] AVHB is classified as

Table 1. Usual and Less Established Indications for Temporary Cardiac Pacing[18,22–24]

Usual Indications	Less-established Indications
Sinus bradycardia or lower escape rhythms due to reversible cause and with symptoms or hemodynamic compromise	During AMI: new or age-indeterminate RBBB with LAFB, LPFB or 1° AVHB, or with LBBB; recurrent sinus pauses refractory to atropine; overdrive pacing for incessant VT
As bridge to permanent pacing with advanced 2° or 3° AVHB, regardless of etiology	During AMI: new or age-indeterminate bifascicular block or isolated RBBB
During AMI: asystole; new bifascicular block with 1° AVHB; alternating BBB; symptomatic or disadvantageous bradycardia not responsive to drugs; or type II 2° AVHB	Heart surgery:
Bradycardia-dependent tachydysrhythmias (*e.g.*, torsades de pointes with LQTS)	To overdrive hemodynamically disadvantageous atrioventricular junctional and ventricular rhythms
	To terminate reentrant SVT or VT
	To prevent pause-dependent or bradycardia-dependent tachydysrhythmias
	During the insertion of a PA catheter in patient with LBBB

AVHB = atrioventricular heart block; AMI = acute myocardial infarction; BBB = bundle branch block; LQTS = long QT interval syndrome, congenital or acquired; RBBB = right bundle branch block; LAFB = left anterior fascicular block; LPFB = left posterior fascicular block; LBBB = left bundle branch block; VT = ventricular tachycardia; SVT = supraventricular tachycardia; PA = pulmonary artery.

first-degree (1°), second-degree (2°), or third-degree (3°; complete) AVHB. Anatomically, it may occur above, within, or below the His bundle.[10] With 1° AVHB, the PR interval is greater than 0.20 s and is usually due to atrioventricular node conduction delay.[25] With 2° AVHB, there is gradual PR interval prolongation before dropped beats (type I or Wenckebach 2° AVHB) or no PR interval prolongation (type II or Mobitz 2° AVHB). Type I 2° AVHB is usually associated with a narrow QRS complex, and type II 2° AVHB with a wide QRS complex.[10] In general, type I 2° AVHB with a narrow QRS complex almost always occurs at the atrioventricular node.[10] When associated with bundle-branch block, there is infra-Hisian block in up to 30% of cases.[25] Type II 2° AVHB is most commonly encountered when the QRS is prolonged and is generally localized to within the His-Purkinje system.[25] Advanced type II 2° AVHB refers to block of two or more consecutive P waves. With 3° AVHB there is no association between atrial and ventricular beats.

Indications for a Permanent Pacemaker

Chronic Atrioventricular Heart Block in Adults. Patients with atrioventricular conduction abnormalities may be asymptomatic or have symptoms related to bradycardia, ventricular dysrhythmias, or both. The presence or absence of symptoms directly attributable to bradycardia has an important influence on the decision to implant a permanent pacemaker.[10] In addition, many indications for pacing with AVHB have evolved over 30 yr on the basis of experience rather than prospective randomized trials, in part because there is no good alternative treatment.[10]

There is little evidence that pacing improves survival with isolated 1° AVHB,[26] even though marked 1° AVHB may be symptomatic without higher-degree AVHB.[27] This may be because of the close proximity of atrial systole to the preceding ventricular systole.[28,29] With type I 2° AVHB due to atrioventricular node conduction delay, progression to more advanced AVHB is unlikely, and pacing is usually not indicated.[10] With type 2° AVHB within or below the His bundle, symptoms are frequent, prognosis is poor, and progression to 3° AVHB is common.[10] Nonrandomized studies strongly suggest that pacing improves survival for patients with 3° AVHB and symptoms.[30–35] Pacing indications for acquired AVHB are listed in table 2.[10,22,25]

Chronic Bifascicular and Trifascicular Block. Major fascicles of the conduction system below the His bundle are the right bundle branch and the left anterior and posterior fascicles of the left bundle branch. The latter activate the left ventricular free wall.[36] In addition, septal branches of the left bundle branch supply the middle third of the ventricular septum and provide the earliest ventricular activation. Isolated block of any one of these fascicles is unifascicular block. Left or right bundle-branch block with left anterior or posterior fascicular block is bifascicular block. Block involving any three fascicles is trifascicular block.

Electrocardiographic criteria for fascicular block are described elsewhere.[36] Syncope is common in patients with bifascicular block but usually is not recurrent or associated with an increased incidence of sudden death.[37–39] However, bifascicular block with periodic 3° AVHB and syncope is associated with an increased incidence of sudden death.[40,41] Thus, if the cause of syncope with bifascicular or trifascicular heart block cannot be determined with certainty, or if concurrent drugs may exacerbate AVHB, prophylactic permanent pacing is indicated, especially if syncope may have been due to intermittent 3° AVHB.[10] Although 3° AVHB is most often preceded by bifascicular block, the rate of progression is slow (years). There is no evidence of acute progression to 3° AVHB during anesthesia and surgery.[42,43] Finally, no one clinical or laboratory variable, including bifascicular block, can identify patients at high risk of death from bradydysrhythmias with bundle-branch block.[10,44]

Table 2. Indications for Permanent Pacing with Acquired Atrioventricular Heart Block in Adults

Class I—Indicated	Class II—May Be Indicated	Class III—Not Indicated
3° AVHB: Symptomatic bradycardia or need for drugs causing same After catheter ablation of the arterioventricular junction Postoperative and not expected to resolve Neuromuscular diseases Escape rhythm < 40 beats/min or asystole > 3.0 s in an asymptomatic patient 2° AVHB that is permanent or intermittent, with symptomatic bradycardia	Asymptomatic 3° AVHB with average rate > 40 beats/min Type II, 2° AVHB without symptoms (permanent or intermittent) Type I, 2° AVHB at or below His bundle without symptoms 1° AVHB with symptoms of low cardiac output that are relieved by temporary pacing Marked 1° AVHB in a patient with CHF	Asymptomatic 1° AVHB Type I, 2° AVHB above His bundle without symptoms AVHB that is expected to resolve

AVHB = arterioventricular heart block; CHF = congestive heart failure.

Adapted from Gregoratos G, Cheitlin M, Conill A, Epstein A, Fellows C, Ferguson TJ, Freedman R, Hlatky M, Naccarelli G, Saksena S, Schlant R, Silka M: ACC/AHA guidelines for implantation of cardiac pacemakers and antiarrhythmia devices: A report of the ACC/AHA Task Force on Practice Guidelines (Committee on Pacemaker Implantation). J Am Coll Cardiol 1998; 31:1175–209. Copyright 1998 by the American College of Cardiology and the American Heart Association. Permission granted for one-time use. Further reproduction is not permitted without permission of the ACC/AHA.

Pacing indications for chronic bifascicular and trifascicular block are summarized in table 3.[10,25]

Atrioventricular Heart Block after Acute Myocardial Infarction. Pacing indications after acute myocardial infarction (AMI) are largely related to the presence of intraventricular conduction defects and not necessarily to symptoms.[10] The requirement for temporary pacing with AMI does not inevitably constitute an indication for permanent pacing.[23] The long-term prognosis for survivors of AMI is related primarily to the extent of myocardial injury and nature of intraventricular conduction defects rather than to AVHB itself.[10,34,45–48] With the exception of isolated left anterior fascicular block, AMI patients with intraventricular conduction disturbances have unfavorable short- and long-term prognoses, with increased risk of sudden death.[10,34,45,47] This prognosis is not necessarily due to the development of high-grade AVHB,[10] although the incidence of high-grade AVHB is higher among these patients.[45,49] Pacing indications for AVHB after AMI are listed in table 4.[10]

Sinus Node Dysfunction. Sinus node dysfunction may manifest as sinus bradycardia, sinus pause or arrest, or sinoatrial block, with or without escape rhythms. It often occurs in association with paroxysmal supraventricular tachydysrhythmias (bradycardia–tachycardia syndrome). Sinus bradycardia due to increased vagal tone is physiologic in trained athletes, who may have sleeping heart rates as low as 30 beats/min, with sinus pauses or type I 2° AVHB.[10] Patients with sinus node dysfunction may have symptoms due to bradycardia, tachycardia, or both. Correlation of symptoms with dysrhythmias is essential[10] and is established by ambulatory monitoring. Sinus node dysfunction may also present as a deficient rate response to stress or exercise (*i.e.*, chronotropic incompetence). An adaptive-rate pacemaker may benefit these patients by restoring more physiologic heart rates.[10,50,51] Although sinus node dysfunction is often the primary indication for a pacemaker,[50] pacing does not necessarily improve survival.[52,53] However, symptoms due to bradycardia may be relieved. Nonrandomized studies suggest that dual-chamber pacing improves survival more than ventricular pacing.[10] A single randomized, prospective trial of atrial *versus* ventricular pacing found significantly higher rates of survival, less atrial fibrillation, fewer thromboembolic compli-

Table 3. Indications for Permanent Pacing with Long-term Bifascicular and Trifascicular Block

Class I—Indicated	Class II—May Be Indicated	Class III—Not Indicated
Intermittent 3° AVHB associated with symptoms Type II, 2° AVHB with symptoms	BFB or TFB block with syncope not proven due to AVHB, but other causes of syncope are not identifiable (specifically, VT) HV interval > 100 ms or pacing-induced infra-Hisian block	BFB or TFB without AVHB or symptoms BFB or TFB with 1° AVHB without symptoms

AVHB = atrioventricular heart block; BFB or TFB = bifascicular or trifascicular block; VT = ventricular tachycardia; HV interval = His-Purkinje conduction time.

Adapted from Gregoratos G, Cheitlin M, Conill A, Epstein A, Fellows C, Ferguson TJ, Freedman R, Hlatky M, Naccarelli G, Saksena S, Schlant R, Silka M: ACC/AHA guidelines for implantation of cardiac pacemakers and antiarrhythmia devices: A report of the ACC/AHA Task Force on Practice Guidelines (Committee on Pacemaker Implantation). J Am Coll Cardiol 1998; 31:1175–209. Copyright 1998 by the American College of Cardiology and the American Heart Association. Permission granted for one-time use. Further reproduction is not permitted without permission of the ACC/AHA.

Table 4. Indications for Pacing for Atrioventricular Heart Block after Acute Myocardial Infarction

Class I—Indicated	Class II—May Be Indicated	Class III—Not Indicated
Persistent 2° or 3° AVHB in the His-Purkinje system	Persistent 2° or 3° AVHB at the atrioventricular node	Transient AVHB without intraventricular conduction defects or with isolated LAFB
Transient 2° or 3° infranodal AVHB with BBB		Acquired LAFB without AVHB
Symptomatic 2° or 3° AVHB at any level		Persistent 1° AVHB with old or age-indeterminate BBB

AVHB = atrioventricular heart block; BBB = bundle branch block; LAFB = left anterior fascicular block

Adapted from Gregoratos G, Cheitlin M, Conill A, Epstein A, Fellows C, Ferguson TJ, Freedman R, Hlatky M, Naccarelli G, Saksena S, Schlant R, Silka M: ACC/AHA guidelines for implantation of cardiac pacemakers and antiarrhythmia devices: A report of the ACC/AHA Task Force on Practice Guidelines (Committee on Pacemaker Implantation). J Am Coll Cardiol 1998; 31:1175–209. Copyright 1998 by the American College of Cardiology and the American Heart Association. Permission granted for one-time use. Further reproduction is not permitted without permission of the ACC/AHA.

cations, less heart failure, and reduced risk of AVHB with atrial pacing than with ventricular pacing after 8 yr of follow-up.[54] Pacing indications for sinus node dysfunction are summarized in table 5.[10]

Hypersensitive Carotid Sinus and Neurally Mediated Syndromes. Hypersensitive carotid sinus syndrome is an uncommon cause for syncope.[10] It is syncope or presyncope due to an exaggerated response to carotid sinus stimulation. Before a pacemaker can be prescribed, the relative contribution of cardioinhibitory components (bradycardia, asystole, and AVHB) and vasodepressor components (vasodilation with hypotension) must be determined. A hyperactive carotid sinus response is defined as asystole greater than 3 s due to sinus arrest or AVHB, an abrupt reduction in blood pressure, or both.[55] With a pure excessive cardioinhibitory response, pacing effectively relieves symptoms.[10] However, because 10–20% of patients have a mixed response, attention to both components is essential for effective therapy.[10]

Neurally mediated syncope accounts for 10–40% of patients with syncope.[10] It includes a variety of clinical scenarios in which triggering of a neural reflex results in a self-limited episode of bradycardia and hypotension.[56] Vasovagal syncope is a common presentation.[10] The use of permanent pacing in these patients is controversial,[10] since many patients have bradycardia after the onset of hypotension. Nonetheless, there was an 85% reduction in risk of recurrent syncope in patients randomized to dual-chamber pacing in one recent study.[57] Indications for pacing with hypersensitive carotid sinus and neurally mediated syndromes are summarized in table 6.[10]

Pacing in Children and Adolescents. Pacemakers are prescribed for children and adolescents with symptomatic bradycardia due to sinus node dysfunction and congenital or acquired advanced 2° or 3° AVHB. Although indications for pacing are similar in children and adults, there are additional considerations with regard to children.[10] First is heart rate. Whereas a rate of 45 beats/min may be normal for an adolescent, it is abnormal for a neonate. Second, survivors of corrective or palliative surgery for congenital heart disease with persistent ventricular dysfunction and altered circulatory physiology may have symptomatic bradycardia at heart rates that do not produce symptoms in normal children. Hence, pacing indications are based more on correlation of symptoms with bradycardia than on arbitrary rate criteria. Finally, pacing is indicated for bradycardia only after exclusion of other causes (*e.g.*, seizures, breath-holding, apnea, and neurally mediated mechanisms).

Indications for permanent pacing for congenital 3° AVHB have evolved on the basis of increased definition

Table 5. Indications for Pacing with Sinus Node Dysfunction

Class I—Indicated	Class II—May Be Indicated	Class III—Not Indicated
SND with documented symptomatic bradycardia, which may be result of drug therapy; in some patients, this will result from long-term, required drug therapy (*i.e.*, no good alternative; reduced dose not possible)	SND, occurring spontaneously or as result of necessary drug therapy, with heart rates < 40 beats/min without clear association between significant symptoms and bradycardia	SND in asymptomatic patients, including those in whom substantial sinus bradycardia (heart rate < 40 beats/min) is consequent to long-term drug treatment
Symptomatic chronotropic incompetence	SND in minimally symptomatic patients, chronic heart rate < 30 beats/min while awake	SND with symptoms of bradycardia, but that are clearly documented as not associated with bradycardia
		SND with symptomatic bradycardia due to nonessential drug therapy

SND = sinus node dysfunction.

Adapted from Gregoratos G, Cheitlin M, Conill A, Epstein A, Fellows C, Ferguson TJ, Freedman R, Hlatky M, Naccarelli G, Saksena S, Schlant R, Silka M: ACC/AHA guidelines for implantation of cardiac pacemakers and antiarrhythmia devices: A report of the ACC/AHA Task Force on Practice Guidelines (Committee on Pacemaker Implantation). J Am Coll Cardiol 1998; 31:1175–209. Copyright 1998 by the American College of Cardiology and the American Heart Association. Permission granted for one-time use. Further reproduction is not permitted without permission of the ACC/AHA.

Table 6. Indications for Pacing with Hypersensitive Carotid Sinus and Neurally Mediated Syndromes

Class I—Indicated	Class II—May Be Indicated	Class III—Not Indicated
Recurrent syncope caused by carotid sinus stimulation; minimal carotid sinus pressure induces asystole > 3 s duration in the absence of drugs that depress the sinus node or atrioventricular conduction	Recurrent syncope without clear provocative events and with a hypersensitive cardioinhibitory response Neurally mediated syncope with significant bradycardia reproduced by a head-up tilt with or without provocative maneuvers (isoproterenol)	Hyperactive cardioinhibitory response to carotid sinus stimulation, but no symptoms Hyperactive cardioinhibitory response to carotid sinus stimulation with vague symptoms such as dizziness, light-headedness, or both Recurrent syncope, light-headedness, or dizziness in the absence of a hyperactive cardioinhibitory response Situational vasovagal syncope in which avoidance behavior is effective

Adapted from Gregoratos G, Cheitlin M, Conill A, Epstein A, Fellows C, Ferguson TJ, Freedman R, Hlatky M, Naccarelli G, Saksena S, Schlant R, Silka M: ACC/AHA guidelines for implantation of cardiac pacemakers and antiarrhythmia devices: A report of the ACC/AHA Task Force on Practice Guidelines (Committee on Pacemaker Implantation). J Am Coll Cardiol 1998; 31:1175–209. Copyright 1998 by the American College of Cardiology and the American Heart Association. Permission granted for one-time use. Further reproduction is not permitted without permission of the ACC/AHA.

of the natural history of the disease, as well as advances in technology and diagnosis. For example, pacing may improve long-term survival and prevent syncope in selected patients with congenital complete AVHB.[58,59] A number of criteria, including average heart rate, QT interval duration, exercise tolerance, and associated structural heart disease, are weighed before pacemaker implantation in asymptomatic patients.[10]

For patients with chronic advanced 2° or 3° AVHB following cardiac surgery, the prognosis is poor without pacing.[60] However, the need for pacing in patients with residual bifascicular heart block and intermittent AVHB is less certain.[10] Before a device is implanted, the embolic risk of residual intracardiac defects and requirement for lifelong pacing must be considered. The bradycardia–tachycardia syndrome commonly occurs following congenital heart surgery.[61] Both antibradycardia and ATP have been used for treatment,[62,63] but the results are equivocal.[10,61,64,65] Nonetheless, symptomatic bradycardia and proarrhythmia with drugs (*i.e.*, provocation of new or worse dysrhythmias) limit their usefulness for treatment. Thus, pacing is weighed as adjunctive therapy for the bradycardia–tachycardia syndrome.[10] Finally, the use of pacing and β-blockers in patients with congenital long QT syndrome has support,[66,67] especially in cases of pause-dependent ventricular tachydysrhythmias. Pacing indications for children and adolescents are summarized in table 7.[10]

Miscellaneous Pacing Indications.

Hypertrophic Obstructive Cardiomyopathy. A dual-chamber pacemaker with a short atrioventricular delay reduces the magnitude of left-ventricular outflow tract obstruction and alleviates symptoms in patients with severely symptomatic obstructive hypertrophic cardiomyopathy.[68–70] Recent trials confirm this and also demonstrate improvement in functional status.[71,72] However, the perceived symptomatic improvement may be little more than a placebo effect.[73,74] Mechanisms by which pacing might improve the LV outflow obstruction are unclear but possibly involve changes in the ventricular contraction pattern.[10] Selection of optimal atrioventricular delay appears critical to achieving a beneficial hemodynamic result.[70,75]

Dilated Cardiomyopathy. Several observational studies show hemodynamic improvement after institution of dual-chamber pacing with short atrioventricular delay for dilated cardiomyopathy.[76–79] Possibly, well-timed atrial contractions prime the ventricles and decrease mitral regurgitation, thereby augmenting stroke volume and arterial pressure.[10] Greater improvement may be obtained with atrioventricular synchronous biventricular pacing than with single-site right ventricular pacing in patients with intraventricular conduction block and end-stage heart failure.[80]

Cardiac Transplantation. The incidence of bradydysrhythmias after cardiac transplantation ranges from 8 to 23%, with the majority of occurrences due to sinus node dysfunction.[10] Because of symptoms and delayed rehabilitation, some centers are more aggressive with pacing for persistent postoperative bradycardia. However, because one half of patients with bradydysrhythmias after cardiac transplantation show improvement by 1 yr, long-term pacing may be unnecessary.[10,81,82]

Termination and Prevention of Tachydysrhythmias by Pacing. Pacing can terminate a variety of tachydysrhythmias, including atrial flutter, paroxysmal reentrant supraventricular tachycardia (SVT), and ventricular tachycardia (VT).[10] A number of pacing patterns are used, including programmed extrastimulation and short bursts of rapid pacing. Although use of dedicated antitachycardia pacemakers has been reported,[83] today this capability is more likely to be incorporated in an ICD device as part of a tiered antidysrhythmia therapy (below). Pacing and β-blockers are used to prevent dysrhythmias with congenital long QT syndrome[66,67] and to

Table 7. Indications for Pacing in Children and Adolescents

Class I—Indicated	Class II—May Be Indicated	Class III—Not Indicated
Advanced 2° or 3° AVHB with symptomatic bradycardia, low cardiac output, or CHF	BTS with the need for long-term antiarrhythmic drug treatment (except digitalis)	Transient postoperative AVHB: return of normal atrioventricular conduction within 7 days
SND with correlation of symptoms during age-inappropriate bradycardia	Congenital 3° AVHB after age 1 yr; average rate < 50 beats/min or pauses 2–3× basic cycle length	Postoperative bifascicular block, with or without 1° AVHB, and no symptoms
Postoperative 2° or 3° AVHB not expected to resolve or that persists at least 7 days	LQTS with type II, 2° or 3° AVHB	Asymptomatic type I, 2° AVHB
Congenital 3° AVHB with wide QRS escape rhythm or ventricular dysfunction	Complex CHD: asymptomatic sinus bradycardia with resting rate < 35 beats/min or pauses > 3 s	Sinus bradycardia without symptoms in adolescents with CHD, when longest R-R interval is < 3 s and minimum rate > 40 beats/min
Congenital 3° AVHB in an infant with rates < 50–55 beats/min or CHD and rates < 70 beats/min	Transient postoperative 3° AVHB; return of normal atrioventricular conduction by 7 days	
Sustained, pause-dependent VT, with or without long QT, in which the efficacy of pacing is thoroughly documented	Asymptomatic postoperative bifascicular block, with or without 1° AVHB	
	Asymptomatic type I, 2° AVHB	
	Adolescents: asymptomatic sinus bradycardia (longest R-R interval < 3 s; minimum rate > 40 beats/min)	

AVHB = atrioventricular heart block; CHF = congestive heart failure; SND = sinus node dysfunction; CHD = congenital heart disease; VT = ventricular tachycardia; BTS = bradycardia-tachycardia syndrome; LQTS = long QT syndrome.

Adapted from Gregoratos G, Cheitlin M, Conill A, Epstein A, Fellows C, Ferguson TJ, Freedman R, Hlatky M, Naccarelli G, Saksena S, Schlant R, Silka M: ACC/AHA guidelines for implantation of cardiac pacemakers and antiarrhythmia devices: A report of the ACC/AHA Task Force on Practice Guidelines (Committee on Pacemaker Implantation). J Am Coll Cardiol 1998; 31:1175–209. Copyright 1998 by the American College of Cardiology and the American Heart Association. Permission granted for one-time use. Further reproduction is not permitted without permission of the ACC/AHA.

prevent recurrences of paroxysmal SVT[84] and bradycardia-dependent atrial fibrillation.[85–88]

Indications for ICDs

An ICD can be used for the prevention of sudden death in a patient with life-threatening ventricular tachydysrhythmias.[10] An implanted atrial ICD[89] or combined atrial and ventricular ICD[90] may be prescribed for patients with paroxysmal atrial tachydysrhythmias or susceptibility to both atrial and ventricular tachydysrhythmias. However, there is no consensus with regard to indications for use of these devices.

It has been clearly shown in prospective clinical trials that ICDs revert sustained VT and ventricular fibrillation (VF). ICDs terminate VF successfully in more than 98% of episodes.[91,92] When an ICD is used with tiered therapy, VT is converted with ATP in 89%[91] to 96%[93] of episodes. Inappropriate ICD therapy, namely, high-energy shocks delivered for misdiagnosed dysrhythmias, is administered to 5–11% of patients. Availability of stored events has made it possible to estimate the benefit of ICDs in the absence of placebo-controlled studies.[94–97] In these studies, ICDs have achieved greater than 98% conversion of VF or VT with circulatory collapse, with a significant projected survival benefit in comparison with that in untreated populations.[94] This benefit is incremental and continues to increase up to 4 yr. A similar benefit exists for patients with sustained VT.[95] In addition, survival of patients with ICDs is influenced by left ventricular function. Survival among patients with a left ventricular ejection fraction greater than or equal to 30% is lower at 3 yr than among those with higher ejection fractions.[98,99] However, both groups derive a significant survival benefit with ICDs in comparison with the benefit of drug treatment alone.[100]

Drugs and surgical or catheter ablation are other options to reduce or prevent VT or VF in at-risk patients, although drugs and ICDs together may improve quality of life by reducing the need for shocks.[10] Whereas serial electrophysiologic testing or Holter monitoring is used to guide drug therapy, maintaining effective therapy may be difficult because of intolerance and prodysrhythmia or adverse effects with prolonged use.[101,102] Although β-blockers do reduce mortality after acute infarction,[103,104] there are no data to support the use of β-blockers as single therapy for ventricular tachydysrhythmias.[10,100] Class III drugs, especially amiodarone, are associated with significantly lower rates of tachydysrhythmia recurrence, sudden death, and total mortality.[10,100] AVID, a large, prospective, randomized trial, compared long-term therapy with ICDs and class III drugs for survivors of cardiac arrest and patients with unstable VT.[100] For ICDs and drugs, unadjusted survival estimates at 1 yr were 89% and 82%; at 2 yr, 82% and 75%; and at 3 yr, 75% and 64%, respectively. With ICDs, the estimated relative risk reduction was 39% at 1 yr and 31% at 3 yr.

Radio-frequency current ablation is most effective for sustained monomorphic VT induced during electrophysiologic study or cardiac surgery and mapped to specific ventricular sites.[10] Surgical experience is more extensive and favorable for patients with coronary disease, and low recurrence rates (< 10% at 2 yr) and minimal sudden death rates have been reported.[105–107]

Table 8. Indications for ICD Therapy for Primary or Secondary Prevention

Class I—Indicated	Class II—May Be Indicated	Class III—Not Indicated
Cardiac arrest due to VT/VF not due to a transient or reversible cause	Cardiac arrest presumed due to VT/VF: other medical conditions preclude EPS	Syncope of undetermined cause; no inducible VT/VF
Spontaneous sustained VT	Severely symptomatic VT before heart transplantation	Incessant VT/VF
Syncope of undetermined origin with clinically relevant, hemodynamically significant sustained VT or VF induced at EPS when drug therapy is ineffective, not tolerated, or not preferred	LQTS, HCM, and other familial conditions with a high risk for life-threatening ventricular dysrhythmias	VT/VF consequent to SVT or VT amenable to surgical or catheter ablation (WPW; specific types of VT*)
NSVT with CAD, previous MI, LV dysfunction, and inducible VF or sustained VT at EPS not suppressed by a class I antidysrhythmia	Inducible sustained VT/VF in patient with NSVT, CAD, old MI, and LV dysfunction	Ventricular VT/VF due to a transient or reversible cause
	Recurrent syncope of undetermined etiology in the presence of ventricular dysfunction and inducible ventricular dysrhythmias at EPS if other causes of syncope have been excluded	Psychiatric illnesses that may be aggravated by ICD implantation or precludes systematic follow-up
		Terminal illness with ≤ 6 months life expectancy
		After CABG: prolonged QRS; LV dysfunction; no spontaneous/inducible VT
		Drug-refractory, Class IV (NYHA) CHF: not candidate for heart transplantation

* Specific VT includes idiopathic left ventricular, right ventricular outflow tract, and bundle branch or fascicular VT.

VT = ventricular tachycardia; VF = ventricular fibrillation; EPS = electrophysiological study; NSVT = nonsustained VT; CAD = coronary artery disease; MI = myocardial infarction; LV = left ventricular; LQTS = long QT syndrome; HCM = hypertrophic cardiomyopathy; SVT = supraventricular tachydysrhythmias; WPW = Wolff-Parkinson-White syndrome; ICD = internal cardioverter–defibrillator; CABG = coronary artery bypass surgery; NYHA = New York Heart Association.

Catheter ablation is most effective with right ventricular outflow tract VT, idiopathic left septal VT, and bundle branch reentrant VT.[108–110] Multiple VT morphologies and polymorphic VT, along with progressive cardiomyopathy, are less amenable to a favorable result with catheter ablation.[10]

Use of ICDs is prescribed for secondary prevention in patients who have coronary artery disease and a history of sudden death or who have documented or inducible sustained ventricular tachydysrhythmias.[10] Such patients account for the majority of those receiving ICDs.[10] ICDs are widely accepted for improving outcomes for these patients. ICDs are also indicated for patients with long QT syndrome and recurrent syncope, sustained ventricular dysrhythmias, or sudden cardiac death despite drug therapy.[67,111,112] ICDs are prescribed along with class IA antidysrhythmic drugs (mostly quinidine) for patients with idiopathic VF or the Brugada syndrome.[113] The latter is the association of right bundle-branch block and ST-segment elevation (electrocardiographic leads V1–V3) with sudden death in patients without confirmed heart disease.[114,115] Sudden death survivors with hypertrophic cardiomyopathy are considered for ICD therapy in preference to or with drugs.[10,116] ICDs are used as prophylaxis for syncope and sudden death with drug-refractory dysrhythmias and dysrhythmogenic right ventricular dysplasia.[117] Fewer than 1% of ICD implants are for primary prevention in pediatric patients.[118] However, the need for lifelong drug therapy, with possible noncompliance and adverse effects, makes ICDs an important treatment option for young patients with congenital heart disease, cardiomyopathies, or primary electrical disease (*e.g.*, long QT syndrome), patients with malignant dysrhythmias, and sudden death survivors.[10] A family history of sudden death may also influence the decision to implant an ICD.[67,111,119]

Finally, ICDs are used for primary prevention in patients with asymptomatic coronary artery disease and nonsustained ventricular tachydysrhythmias.[112,120] Other circumstances in which ICDs have been used for primary prevention include following coronary artery bypass surgery in patients with severe left ventricular dysfunction (ejection fraction < 35%) and after abnormal findings of signal-averaged electrocardiography,[121] as well as in some patients awaiting heart transplantation.[10, 122, 123] However, with the latter, the benefit is diluted by some patients' death due to heart failure. Indications for ICDs are summarized in table 8.[10]

Device Selection

Temporary Pacing. Transvenous (endocardial), epicardial, transesophageal, and transcutaneous routes are used for temporary pacing. The first two routes are considered invasive (*e.g.*, risk of sepsis, direct myocardial damage, or cardiac perforation with tamponade), and the latter two pacing routes are considered noninvasive. Discussion of the pros and cons of each, as well as methods and equipment, is beyond the scope of this article. The interested reader is referred to previous publications.[16,18,19,24]

Selection of a Permanent Pacemaker. Single- and dual-chamber pulse generators vary in size, battery capacity, cost, and unipolar or bipolar electrode configuration (below). They may incorporate sensor-modulated adaptive-rate pacing, programmable polarity, and/or automatic mode-switching. Pacing leads vary in electrode configuration, insulation material, methods for fixation, stimulation impedance, and presence of steroid elution. Other factors that influence pacemaker selection are the pacemaker programming device capabilities and access to technical support. For all devices, pacing mode, pulse amplitude and width, sensitivity, lower rate, and refractory periods are programmable. For dual-chamber devices, the atrioventricular interval and maximum tracking rate are also programmable. With adaptive-rate pacemakers, several rate-modulation parameters are programmable. Implanting physicians must also anticipate the progression of cardiac rhythm abnormalities when selecting and programming a device.[10] For example, patients with sinus node dysfunction and susceptibility to paroxysmal atrial tachydysrhythmias might develop AVHB due to needed drug therapy, disease progression, or catheter ablation for modification of atrioventricular conduction. If so, a dual-chamber pacemaker with automatic mode-switching might be indicated. Finally, the patient with an indication for pacing and at risk for VT or VF will receive a single- or dual-chamber ICD, since all ICDs today have a single- or dual-chamber pacing capability, and many have adaptive-rate pacing as well.

Adaptive-rate Pacemakers. A 1996 industry-wide survey in the United States indicated that adaptive-rate pacing was a programmable option in 83% of all implanted pulse generators.[10] In patients with chronotropic incompetence, adaptive-rate pacing improves exercise capacity and quality of life.[10] Most sensors are piezoelectric crystals or accelerometers that detect motion, acceleration, vibration, or pressure.[10,124] Nevertheless, minute ventilation[125] or stimulus-to-T interval[126] sensors may provide a rate response more proportional to exercise.[10]

Single-pass Lead Systems. Commonly, dual-chamber devices have a separate atrial lead to detect atrial depolarization in patients with sinus node dysfunction. Single-pass leads have both atrial and ventricular electrodes, negating the need for separate leads.[6] However, it was found that the amplitude of sensed signals with separate, floating atrial leads was inconsistent and varied significantly with changes in posture.[127,128] In addition, atrial pacing was not possible. With newer, single-pass leads, the atrial signal amplitude is higher and dual-chamber pacing is possible.[129,130]

Programmable Lead Configuration and Automatic Mode-switching. Most contemporary pacemakers offer separately programmable lead configurations for both pacing and sensing in the atrium and ventricle.[6] Thus, if the pacing system uses bipolar leads, it is possible to noninvasively switch back and forth between unipolar and bipolar lead configurations. With the former, all or part all of the pulse generator metal housing (can) serves as the anode (+) and the distal electrode of the bipolar lead as cathode (−). With the bipolar configuration, proximal and distal lead electrodes serve as anode and cathode, respectively. The ability to program unipolar pacing is necessary if lead insulation or conductor failure occurs in a bipolar lead system.[6] In addition, the ability to program separate lead configurations for sensing and pacing permits exploitation of either while minimizing disadvantages (*e.g.*, oversensing with unipolar leads).[5,6] Dual-chamber pacemakers with automatic mode-switching are used for patients with AVHB and susceptibility to paroxysmal atrial tachydysrhythmias. Algorithms detect rapid, nonphysiologic atrial rates and automatically switch the pacing mode to one that excludes atrial tracking and the associated risk of ventricular pacing at or near the programmed maximal rate.[7–9,131]

Pacemaker Leads. Most contemporary pacemakers use transvenous (endocardial) leads. Bipolar leads are being used increasingly worldwide.[6] Bipolar sensing reduces risk of inappropriate pacing inhibition or stimulation due to oversensing. However, with some bipolar leads, there has been an unacceptably high failure rate due to lead insulation degradation,[10] although newer lead designs have improved on this.[132,133] An important advance has been development of steroid-eluting leads.[6,10] These have a small reservoir of corticosteroid that is slowly released into the electrode–tissue interface, reducing inflammation, fibrosis, and chronic capture thresholds.

Selection of an ICD. Many of the above considerations apply to ICD selection, since they feature antibradycardia pacing as well as ATP and shocks for tachydysrhythmias. A primary feature that distinguishes contemporary ICDs from earlier models is the availability of ATP as a programmable option. Although ATP increases pulse generator cost, it is useful in a majority of patients receiving ICDs, since it converts up to 96% of episodes of VT without the need for shocks.[93] Nonetheless, ATP may accelerate VT in 2–6% of episodes,[93,134,135] although this may be influenced by whether the pacing algorithm to terminate VT is used empirically or on the basis of results of electrophysiologic testing.[135] Patients with only VF before ICD implantation are less likely to subsequently have VT detected by their ICDs.[136] However, the incidence of VT in these patients (18%) is significant[136]; thus, it is desirable to have ATP as a programmable feature of ICDs, even without a history of VT.[4,10] Finally, ICDs with dual-chamber pacing and sensing are appropriate for patients who require dual-chamber pacing and therapy for VT or VF or who have atrial dysrhythmias that might trigger inappropriate ICD therapies.[10]

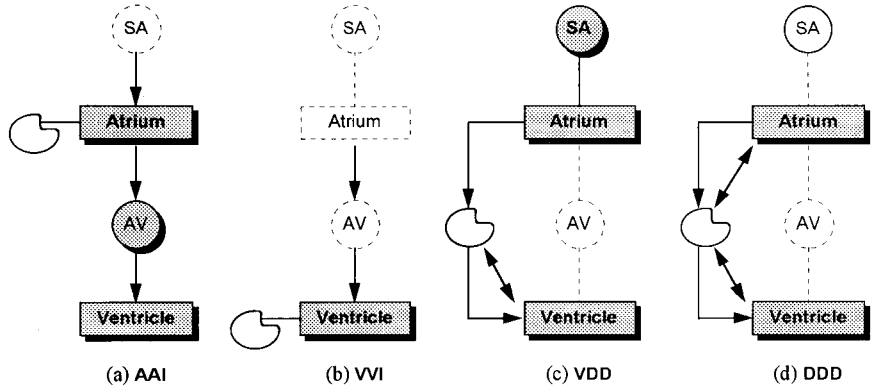

Fig. 1. Examples of antibradycardia pacing modes. (*A*) Atrial-inhibited (AAI) pacing for sinus arrest or bradycardia. The pulse generator is shown with atrial leads only. The atrium is paced, unless pacing is inhibited by sensed spontaneous atrial depolarizations. (*B*) Ventricular-inhibited (VVI) pacing for atrioventricular (AV) heart block (AVHB) with atrial fibrillation. The pulse generator is shown with ventricular leads only. The ventricle is paced, unless pacing is inhibited by sensed spontaneous ventricular depolarizations. (*C*) Ventricular-inhibited, atrial-triggered (VDD) pacing for AVHB with normal sinoatrial (SA) node and atrial function. The pulse generator is attached to atrial leads for sensing only and to ventricular leads for pacing and sensing. If a spontaneous atrial depolarization is sensed, the ventricle is paced after an appropriate atrioventricular interval to permit ventricular filling. This is the atrial-triggered ventricular pacing (VAT) component of the VDD mode, which also includes capabilities of the VVI mode. (*D*) Dual-chamber sequential or atrioventricular universal (DDD) pacing for sinus bradycardia and AVHB. The pulse generator is shown attached to atrial and ventricular leads for dual-chamber sensing and pacing. This mode incorporates AAI, VVI, and VAT pacing capabilities. Reprinted with permission from Bernstein AD, Parsonnet V: Fundamentals of antibradycardia-pacemaker timing. ACC Educational Highlights 1995; 11:5–9; copyright American College of Cardiology.

Device Design and Function

Pacemaker Design and Function. Pacemakers are powered by lithium-iodide batteries, with an expected service life of 5–12 yr, depending on device capabilities. Actual service life will depend on the need for pacing and the programmed stimulus parameters. Most systems use bipolar transvenous leads. These are positioned under fluoroscopic guidance, with the lead configuration programmable (above). A single-chamber pacemaker stimulates the atria or ventricles on the basis of programmed timing intervals. In addition, by sensing intrinsic atrial and/or ventricular depolarizations, it can be inhibited from providing unnecessary or inappropriate stimuli. Dual-chamber devices also time delivery of ventricular stimuli relative to sensed atrial depolarizations to maintain proper atrioventricular synchrony. In figure 1, we illustrate how a pacemaker might be configured to pace in patients with sinus node dysfunction or AVHB. Throughout the remainder of the current article and in the sequel, the North American Society for Pacing and Electrophysiology–British Pacing and Electrophysiology Group (NASPE/BPEG) pacemaker code (sometimes called NBG code; table 9) is used as shorthand to describe pacing modes.[137]

Timing Design: Single-chamber Pacemakers. Today, most pacemakers in the United States are conventional or adaptive-rate, dual-chamber devices.[10] However, with normal atrioventricular conduction and sinus node function, they may operate as single-chamber devices, in the AAI/AAIR or VVI/VVIR modes, depicted in figure 1. They have a single timing interval, the interval between stimuli in the absence of sensed depolarization. For single-chamber pacing modes, this interval is the atrial or ventricular escape interval. It is inversely proportional to the pacing rate in paced pulses per minute (ppm):

$$\text{Escape interval (ms)} = 60{,}000/\text{rate (ppm)}$$

In the AAI mode (fig. 2), pacing will occur at the end of the programmed atrial escape interval, unless a spon-

Table 9. The NASPE-BPEG Generic (NBG) Pacemaker Code

I	II	III	IV	V
Chamber Paced	Chamber Sensed	Response to Sensed Event	Programmability/Rate Response*	Antitachycardia Functions†
O (none)	O (none)	O (none)	O (none)	O (none
A (atrium)	A (atrium)	I (inhibit)	R (adaptive rate)	P (ATP)
V (ventricle)	V (ventricle)	T (triggered)	P (simple programmable)	S (shock)
D (dual: A + V)	D (dual: A + V)	D (I and T)	M (multiprogrammable)	D (dual: P + S)
S (single)‡	S (single)‡		C (communicating)	

* In current terminology, only the adaptive rate response (R) is indicated by the fourth position; all current pacemakers have full programming and communicating capability. Therefore, the letters P, M, and C are no longer used.

† ICD with antibradycardia and antitachycardia pacing capabilities.

‡ Single-chamber device that paces either the atrium or ventricle.

ATP = antitachycardia pacing.

From Bernstein AD, Camm AJ, Fletcher RD, Gold RD, Rickards AF, Smyth NP, Spielman SR, Sutton R: The NASPE/BPEG generic pacemaker code for antibradyarrhythmia and adaptive-rate pacing and antitachyarrhythmia devices. Pacing Clin Electrophysiol 1987; 10:794–9. Used with permission.

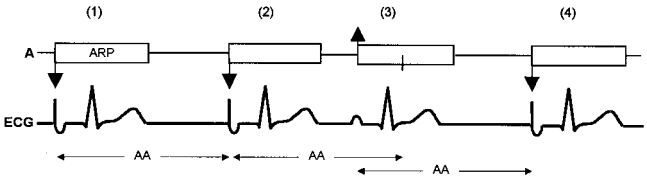

Fig. 2. Depiction of atrial-inhibited pacing, as for sinus bradycardia with intact atrioventricular conduction. In the first beat, the atrium (A) is paced (by convention, an arrow pointing toward the electrocardiogram [ECG] in the atrial timing diagram). The atrial refractory period (ARP) prevents conducted R and ensuing T waves from being interpreted by the device as a P wave and inappropriately resetting the atrial escape interval (AA). After the programmed AA interval, another atrial stimulus occurs and resets the interval. In the third beat, a spontaneous P wave is sensed (by convention, an arrow pointing away from the electrocardiogram in the atrial timing diagram) before the AA interval times out. This resets the AA interval without pacing (the short vertical line in the atrial timing diagram shows where the stimulus would have occurred). In the absence of further sensing, the atrium is paced in the fourth beat when the AA interval times out. This example illustrates a principle that is useful for interpreting a single-chamber pacemaker electrocardiogram. Once the escape interval is known (from the clinical records, device telemetry, or measurement between consecutive paced beats), electrocardiographic interpretation is facilitated by working backward from the last stimulus to identify the sensed event that reset the pacemaker's escape timing as a P wave (or R wave, as the case may be), and not a spurious signal. Reprinted with permission from Bernstein AD, Parsonnet V: Fundamentals of antibradycardia-pacemaker timing. ACC Educational Highlights 1995; 11:5–9; copyright American College of Cardiology.

taneous atrial depolarization is sensed first and resets the interval. Stimulus timing is identical for the VVI mode (fig. 3). Ventricular pacing will occur at the end of the ventricular escape interval, unless a spontaneous ventricular depolarization is sensed first and resets the interval. Because of this timing similarity, some single-chamber pacemakers can be used with pacing leads in either the

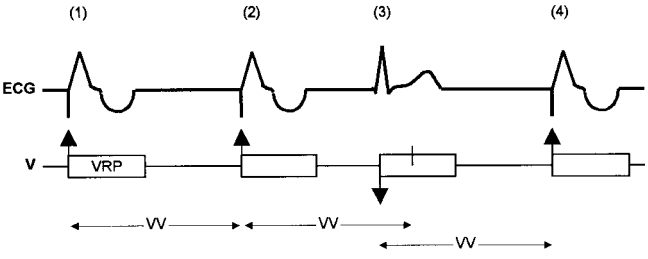

Fig. 3. Depiction of ventricular-inhibited pacing, as for atrioventricular heart block with atrial fibrillation. In the first beat, the ventricle (V) is paced. The pacemaker's ventricular refractory period (VRP) prevents the ensuing T wave from being interpreted as an R wave and inappropriately resetting the ventricular escape interval (VV). The programmed VV interval times out with delivery of a ventricular stimulus and resets the VV interval. However, a spontaneous R wave (third beat) is sensed before this times out. It inhibits the ventricular stimulus that would have occurred (short vertical line in the ventricular-channel timing diagram) and resets the VV interval. With no further sensing, pacing occurs when the VV interval times out (fourth beat). ECG = electrocardiogram. Reprinted with permission from Bernstein AD, Parsonnet V: Fundamentals of antibradycardia-pacemaker timing. ACC Educational Highlights 1995; 11:5–9; copyright American College of Cardiology.

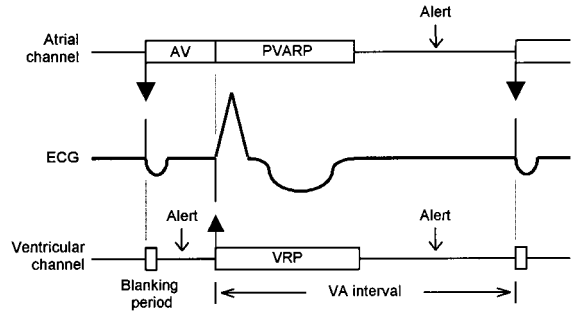

Fig. 4. Basic timing of a dual-chamber pacemaker, with pacing and sensing in both chambers. Atrial and ventricular stimuli are shown in the timing diagrams by arrows pointing toward the electrocardiogram (ECG) from above and below, respectively. The programmed atrioventricular (AV) interval provides time for ventricular filling. The atrial channel is refractory during the atrioventricular interval and from delivery of the ventricular stimulus until the end of the programmed postventricular atrial refractory period (PVARP). This prevents atrial sensing from resetting the escape timing. The blanking period (ventricular channel) prevents sensing of the atrial stimulus. However, sensing in the alert period after the blanking period would enable a spontaneous R wave to reset the interval between the ventricular stimulus or sensed spontaneous R wave and the subsequent atrial stimulus (the VA interval), thereby inhibiting ventricular stimulation. As shown, this does not occur, so the atrioventricular interval times out with delivery of a ventricular stimulus. The ventricular refractory period (VRP) prevents sensed T waves from inappropriately resetting the VA interval. Sensing during the alert periods after the PVARP and VRP will reset basic timing, initiating new atrioventricular and VA intervals, respectively. Reprinted with permission from Bernstein AD, Parsonnet V: Fundamentals of antibradycardia-pacemaker timing. ACC Educational Highlights 1995; 11:5–9; copyright American College of Cardiology.

atrium or the ventricle. In addition, some single-chamber pacemakers offer rate hysteresis as a programmable option. With this, the atrial or ventricular escape interval after a sensed depolarization is longer than that after a paced depolarization. Rate hysteresis encourages the emergence of an intrinsic rhythm, thereby reducing the likelihood of competition between paced and spontaneous rhythm and prolonging battery life.

Timing Design: Dual-chamber Pacemakers. Figure 4 illustrates the basic timing design of a dual-chamber pacemaker that can pace and sense in both the atrium and the ventricle. Dual-chamber pacemakers have two basic timing intervals, whose sum is the pacing-cycle duration. The first is the atrioventricular interval, which is the programmed interval from a paced or sensed atrial depolarization to the subsequent ventricular stimulus. Some dual-chamber pacemakers offer the option of programmable atrioventricular interval hysteresis. If so, the atrioventricular interval after an atrial stimulus is longer than that following a sensed spontaneous P wave to maintain a uniform interval between atrial and ventricular contractions. The second basic timing interval is the VA interval, the interval between a ventricular stimulus or sensed spontaneous R wave and the subsequent atrial stimulus. During the pacemaker's atrial and ventricular refractory periods (fig. 4), sensed

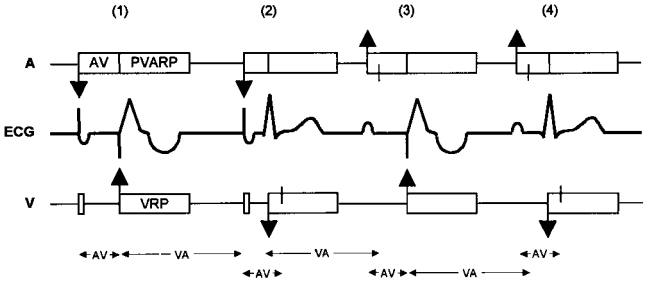

Fig. 5. Basic patterns in dual-chamber pacing. The first beat is fully paced and is an example of atrioventricular (AV) sequential pacing. In the second beat, a spontaneous R wave is sensed in the ventricular (V) channel before the atrioventricular interval times out, initiating a new VA interval (the interval between the ventricular stimulus or sensed spontaneous R wave and the subsequent atrial stimulus). Thus, it inhibits the ventricular stimulus that would have occurred (short vertical line in the ventricular-channel timing diagram). In the third beat, a P wave is sensed in the atrial (A) channel before the VA interval times out, initiating a new atrioventricular interval. It also inhibits the stimulus that would have occurred (short vertical line in the atrial-channel timing diagram). This is an example of atrial synchronous ventricular pacing, which is equivalent to the VAT mode (fig. 1). In the last beat, spontaneous P and R waves are sensed before the respective VA and atrioventricular intervals time out. ECG = electrocardiogram; PVARP = postventricular atrial refractory period; VRP = ventricular refractory period. Reprinted with permission from Bernstein AD, Parsonnet V: Fundamentals of antibradycardia-pacemaker timing. ACC Educational Highlights 1995; 11:5–9; copyright American College of Cardiology.

events do not reset the device's escape timing. During the ventricular channel blanking period (fig. 4), ventricular sensing is disabled to avoid overloading of the ventricular sense amplifier by voltage generated by the atrial stimulus. It also prevents the atrial stimulus from inappropriately resetting the VA interval without delivery of a ventricular stimulus. Sensing during alert periods after the post-ventricular atrial and ventricular refractory periods (fig. 4) resets basic pacemaker timing and initiates new atrioventricular or VA intervals, respectively.

A dual-chamber pacemaker provides atrioventricular sequential, atrial, ventricular, or no pacing, depending on the sensing patterns (fig. 5). Whenever sensing occurs outside the atrial or ventricular refractory periods or the blanking period, the current atrioventricular or VA interval is terminated without stimulation (fig. 5). The next timing interval begins at once. In addition, sensed R and P waves reset the atrial and ventricular timing intervals, respectively, without stimulus delivery (fig. 5).

Internal Cardioverter–Defibrillator Design and Function. An ICD system consists of a pulse generator and leads for tachydysrhythmia detection and therapy. ICDs provide antitachycardia and antibradycardia pacing, synchronized (cardioversion) or nonsynchronized (defibrillation) shocks, telemetry, and diagnostics, including stored event electrograms and history logs.[4,138] Essentially, the pulse generator is a self-powered computer within a hermetically sealed titanium casing (can). One or two (in series) 3.2-V lithium–silver vanadium oxide (SVO) batteries with high power density are used to power the pulse generator, circuitry, and aluminum electrolytic storage capacitors.[138] Most ICD designs use two capacitors in series to achieve a maximum voltage for defibrillation.[138] A major challenge in ICD design is the large range of voltages that must be controlled in a very small package. While monitored intracardiac signals may be as small as 100 μV, therapeutic defibrillatory shocks approach 750 V, with a leading edge of 15 amperes (A) and a pulse termination spike of 210 A.[138] Furthermore, because ICD batteries contain up to 20,000 joules (J), a potential hazard exists if the charging and firing circuits were to unload all this energy either electrically or thermally into the patient in a brief period.[138] Indeed, an ICD might reach a temperature of 85°C during a high-current state (*e.g.*, a battery short or component failure within the high-voltage circuit).[138] Therefore, manufacturers reduce this hazard by use of current and thermal fuses in the power supplies.[138] In addition, the number of shocks delivered during treatment is usually limited to five or six per dysrhythmia.

Modern ICDs use transvenous lead systems for sensing, pacing, and shocks. Epicardial leads are still used in infants and small children. The expected service life is 5–8 years.[53,138] Aside from the leads and battery, major subsystems of dual-chamber, adaptive-rate ICD pulse generators include (1) up to 100 kilobytes of ROM for system start-up tasks and some program space; (2) up to 512 kilobytes of RAM for additional program space and storage of operating parameters and lead electrogram data; (3) low-voltage supplies (3–15 V) for pacing and digital circuits and to control charging circuits; and (4) a high-voltage supply and output switching to generate and control delivery of high-energy, biphasic shocks.[138]

Sensing of Ventricular Depolarizations by ICDs. Reliable sensing of ventricular depolarization is essential.[4,138,139] The sense amplifier must respond quickly and accurately to rates of 30–360 beats/min or greater and to the varying amplitude and morphology of intracardiac signals during VT or VF. Unfiltered intracardiac electrograms are sent to the sense amplifier. This has a band-pass filter to reject low-frequency T waves and high-frequency noise, automatic gain control (autogain), a rectifier to eliminate polarity dependency, and a fixed or autoadjusting threshold event detector. The sense amplifier produces a set of R-R intervals for the VT and VF detection algorithms to use.

Because the amplitude of intracardiac ventricular electrograms can vary widely between sinus rhythm, VT, and VF, some form of autogain is required.[4,138,139] If gain and sensitivity were fixed, as in pacemakers, and depending on the settings chosen, this could result in VT and VF undersensing or oversensing. In newer ICDs, digital, dynamic autogain continuously adjusts the gain so that the amplitude of the processed signal remains constant. An autoadjusting sensitivity threshold sets the sensitivity

to a proportion of the amplitude of the latest sensed event, and sensitivity then gradually increases until the next event is sensed. Sensed events are analyzed with use of a detection algorithm. This divides all possible ventricular rates into nonoverlapping rate zones (bradycardia, normal rate, VF, and up to three programmable VT zones).

VF Detection and Therapy by ICDs. The ICDs use rate criteria as the sole method for detecting VF.[4,138,139] VF detection algorithms must have high sensitivity but low specificity. This is because the result of not detecting VF is grave. However, if criteria for tracking input signals are too aggressive, the ICD will likely oversense T waves during sinus rhythm. If too conservative, the device will likely undersense some VF but work very well during normal sinus rhythm. Even with autogain and autoadjusting sensitivity threshold, VF detection algorithms must tolerate some degree of undersensing. As a result, an ICD X/Y detector triggers when X of the previous Y sensed ventricular intervals (typically, 70–80% of intervals in a sliding window of 10–24 intervals) are shorter than the VF detection interval.[139] This mechanism successfully ignores the effect of a small number of undersensed events because of the small amplitude of VF intracardiac signals. Any tachycardia with a cycle length less than the VF detection interval will initiate VF therapy. After capacitor charging but before shock delivery, an algorithm confirms the presence of VF. After shock delivery, redetection and episode-termination algorithms determine whether VF has terminated, continued, or changed.

Successful defibrillation may require voltages 125 times greater than the battery voltage.[4,138] This charge is stored in capacitors and delivered between high-energy electrodes to depolarize the ventricles, parts of which may be partially refractory and up to 10 cm away. Output switching is used during capacitor discharge to produce a biphasic shock waveform. In comparison with monophasic shocks, biphasic shocks greatly reduced defibrillation energy requirements[140–142] and were critical to development of smaller ICDs suitable for pectoral implantation.

VT Detection and Therapy by ICDs. In contrast with VF detection algorithms, most VT algorithms in single-chamber ICDs require a programmable number of consecutive R-R intervals shorter than the VT detection interval.[4,139] A longer R-R interval, as might occur during atrial fibrillation, would reset the VT counters. In patients with both supraventricular and ventricular tachydysrhythmias, up to 45% of ICD discharges may be inappropriate if rate is used as the sole criterion for VT therapy.[143] These are poorly tolerated by patients. To increase specificity, VT detection algorithm enhancements are programmed for one or more VT zones in single-chamber ICDs, including criteria for stability of rate, suddenness of onset, and intracardiac QRS morphology.[4,139] Enhancement criteria are not available in the VF zone, where maximum sensitivity is required. In addition, they are programmed only in rate zones that correspond to VT hemodynamically tolerated by the patient.

The rate stability criterion is used to distinguish sustained monomorphic VT with little cycle-length variation from atrial fibrillation with much greater cycle-length variation. For example, one algorithm operates when the VT count reaches four.[139] It then compares the latest R-R interval with each of the three preceding intervals. If the absolute value in milliseconds of any of the interval differences is greater than the programmed VT interval, the VT counter is reset to zero. Another algorithm calculates the R-R interval differences throughout a specified duration of tachycardia and then computes average variance on a beat-to-beat basis.[139] If R-R cycle-length variance at the end of the specified duration is greater than programmed for the VT zone, the rhythm is declared unstable (*i.e.*, not likely to be VT), and VT therapy is inhibited. The suddenness of onset criterion is used to distinguish sinus tachycardia from VT, since VT has a more sudden rate increase. For example, one algorithm finds the maximum difference between adjacent intervals for five intervals on each side of the lowest VT rate boundary.[139] When the maximum difference exceeds the programmable onset parameter by 9–34%, the algorithm selects the shorter of the two intervals as the pivot interval. Then, the difference between the average of four intervals before and three of four intervals after the pivot interval must also be greater than 9–34% to satisfy the onset criterion. Finally, morphology algorithms discriminate VT from SVT on the basis of morphology of intracardiac electrograms.[139] Morphology algorithms were not available in early ICDs because the required calculations were beyond the capabilities of then-available microprocessors. Discussion of the specific methods used for QRS waveform morphology analysis is beyond the scope of this article.[139]

Insufficient specificity of VT detection algorithms, despite optimal enhancements, has been a significant problem with single-chamber ICDs. Dual-chamber ICDs have an atrial lead, which is used for bradycardia pacing and sensing for tachycardia discrimination.[144] Detection algorithms in dual-chamber ICDs use atrial and ventricular timing data to discriminate SVT from VT.[139] For example, the detection algorithm in the Gem DR and Jewel AF ICDs (Medtronic, Minneapolis, MN) is based on several fundamental design principles.[139] High sensitivity of single-chamber, rate-only detection is retained in the enhanced detection algorithm. The devices withhold VT/VF detection only if they can positively identify a specific SVT. The detection algorithm has four key elements: (1) the pattern of atrial and ventricular events; (2) atrial and ventricular rates; (3) regularity of R-R intervals; and (4) presence or absence of atrioventricular dissoci-

ation. The algorithm also uses two methods of atrial and ventricular pattern analysis, which are described and illustrated elsewhere.[139] Nonetheless, limitations of dual-chamber enhancement algorithms include (1) atrial far-field sensing of R waves, leading to rhythm misclassification, (2) trade-offs between undersensing and the necessity for dual-chamber blanking periods to prevent cross-sensing, and (3) distinguishing VT with 1:1 VA conduction from SVT with 1:1 atrioventricular conduction.[139]

Treatment options for tachycardia in the VT zones include ATP, cardioversion, or defibrillation.[4,139] Treatment progresses through a programmable sequence of responses (tiered therapy) until the episode is terminated. Most sustained monomorphic VT can be terminated by a critical pacing sequence.[145] With ATP, usually a train of stimuli are delivered at a fixed percentage of the VT cycle length. Repeated and more aggressive trains can be administered, resulting in termination of VT or progression to cardioversion or defibrillation. Pacing at faster rates increases the likelihood of VT termination and risk of acceleration. ATP is effective, with greater than 90% successful termination of spontaneous VT.[146,147] ATP with backup defibrillation is well-tolerated and reduces the need for painful, high-energy shocks.[148] Finally, the efficacy of ATP and low-energy cardioversion is similar.[149] Both reduce the time to therapy and conserve ICD battery life.[4]

Bradycardia Pacing by ICDs. Ventricular demand pacing for bradycardia is a standard feature of all single-chamber ICDs. Dual-chamber ICDs have all the capabilities of dual-chamber pacemakers, including adaptive-rate pacing and automatic mode-switching. Approximately 20% of ICD recipients require bradycardia pacing, and 80% of these would benefit from dual-chamber pacing.[150] If one includes patients with severe ventricular dysfunction (ejection fraction $<$ 20%) and who would benefit from dual-chamber sensing, it is possible that up to 50% of ICD recipients may benefit from the implantation of a dual-chamber ICD.[4,151–152] Finally, pacing thresholds during pacing for VT and after defibrillation shocks are frequently higher than those needed for routine bradycardia pacing. Pacing thresholds for these conditions are separately programmable in dual-chamber ICDs.[4]

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Cardiac Rhythm Management Devices (Part II)

Perioperative Management

John L. Atlee, M.D., Alan D. Bernstein, Eng.Sc.D.†*

IN the first installment of this two-part communication, we reviewed the indications for an implanted pacemaker or internal cardioverter–defibrillator (ICD), provided a brief overview of how a device is selected, and described the basics of pacemaker and ICD design and function. Here we discuss specific device malfunction, electromagnetic and mechanical interference, and management for patients with a device or undergoing system implantation or revision. As in part I, the NASPE-BPEG (for North American Society for Pacing and Electrophysiology–British Pacing and Electrophysiology Group; sometimes abbreviated as NBG) generic pacemaker code is used to designate pacing modes.[1]

Device Malfunction

Pacemaker Malfunction

Pacing malfunction can occur with an implanted pacemaker or ICD because all contemporary ICDs have at least a backup single-chamber pacing capability, and most have dual-chamber pacing as well. Primary pacemaker malfunction is rare, accounting for less than 2% of all device-related problems in one large center over a 6-yr period.[2] Some devices have programmed behavior that may simulate malfunction, termed pseudomalfunction.[3] For example, failure to pace may be misdiagnosed with programmed rate hysteresis. With rate hysteresis, the pacing cycle duration is longer after a sensed *versus* paced depolarization. This encourages the emergence of intrinsic rhythm. Pacemaker malfunction is classified as failure to pace, failure to capture, pacing at abnormal rates, undersensing (failure to sense), oversensing, and malfunction unique to dual-chamber devices (table 1).[3,4] To diagnose device malfunction, it is necessary obtain a 12-lead electrocardiogram and chest radiograph and to interrogate the device to check pacing and sensing thresholds, lead impedances, battery voltage, and magnet rate.[3,4]

Failure to Pace. With a single-chamber pacemaker and failure to pace, there will be no pacing artifacts in the surface electrocardiogram. The intrinsic rate will be below the programmed lower rate limit, which is obtained from the patient's records or through device interrogation.[3,4] Misdiagnosis of failure to pace is possible if the device is inhibited by intrinsic cardiac depolarizations not apparent in the surface electrocardiogram. With a dual-chamber device, no pacing artifacts may be present, or there may be pacing in only one chamber. With the latter, first it must be determined that the device is not programmed to a single-chamber pacing mode. Failure to pace may be intermittent or continuous.

Failure to pace is often due to oversensing (see Oversensing). Other causes are an open circuit caused by a broken, dislodged, or disconnected lead, lead insulation defects, or malfunction of other system components. In addition, problems with the lead–tissue interface may explain failure to pace. When failure to pace occurs within 48 h of device implantation, lead dislodgement, migration, and myocardial perforation are probable causes. Misdiagnosis of failure to pace may occur with impending battery depletion, evidenced by the "elective replacement indicator." The elective replacement indicator rate is not necessarily the same as the nominally programmed rate. Examples of elective replacement indicators are listed in table 3.[5] Failure to pace may be misdiagnosed with too-rapid strip-chart recording speeds. If so, the intervals between paced beats appear longer than normal. Finally, the sense amplifier may detect isoelectric extrasystoles (*i.e.*, in the surface electrocardiogram) that properly inhibit stimulus delivery.

Failure to Capture. With failure to capture, there will be visible pacing artifacts in the 12-lead surface electrocardiogram but no or intermittent atrial or ventricular depolarizations. To confirm this diagnosis, the device must be interrogated to examine event markers and measured data (*e.g.*, lead impedances and pacing and

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Table 1. Categories of Pacemaker Malfunction, with Electrocardiographic Appearance and Likely Cause for Malfunction

Category of Malfunction	Electrocardiographic Appearance	Cause for Malfunction
Failure to pace	For one or both chambers, either no pacing artifacts will be present in the electrocardiograph, or artifacts will be present for one but not the other chamber	Oversensing; battery failure; open circuit due to mechanical problems with leads or system component malfunction; fibrosis at electrode-tissue interface; lead dislodgement; recording artifact
Failure to capture	Atrial or ventricular pacing stimuli or both are present, with persistent or intermittent failure to capture	Fibrosis at electrode-tissue interface; drugs or conditions that increase pacing thresholds (table 2)
Pacing at abnormal rates	1. Rapid pacing rate (upper rate behavior) 2. Slow pacing rate (below lower rate interval) 3. No stimulus artifact; intrinsic rate below lower rate interval	1. Adaptive rate pacing; tracking atrial tachycardia; pacemaker-mediated tachycardia; oversensing 2. Programmed rate hysteresis, or rest or sleep rates; oversensing 3. Power source failure; lead disruption; oversensing
Undersensing (failure to sense)	Pacing artifacts in middle of normal P waves or QRS complexes	Inadequate intracardiac signal strength; component malfunction; battery depletion; misinterpretation of normal device function
Oversensing	Abnormal pacing rates with pauses (regular or random)	Far-field sensing with inappropriate device inhibition or triggering; intermittent contact between pacing system conducting elements
Malfunction unique to dual-chamber devices	Rapid pacing rate (*i.e.*, upper rate behavior)	Crosstalk inhibition; pacemaker-mediated tachycardia (*i.e.*, runaway pacemaker; sensor-driven tachycardia; tachycardia during MRI; tachycardia 2° to tracking myopotentials or atrial tachycardias; and pacemaker-reentrant tachycardia)

MRI = magnetic resonance imaging.
Compiled from Levine[3] and Mitrani.[4]

sensing thresholds).[3,4] Event markers will identify the release of stimuli and recycling of the device by sensed events. As for causes (table 1), stimulation thresholds may rise during lead maturation (2–6 weeks after implantation), but this has become far less of a problem since the introduction of steroid-eluting leads and other refinements in lead technology. Nonetheless, pacing thresholds may continue to rise until they exceed maximum pulse-generator output (exit block).[3] Transient, metabolic, and electrolyte imbalance,[6–12] as well as drugs and other factors,[3,13–19] may increase pacing thresholds (table 2), a circumstance explaining pacing failure. Anesthetic drugs are not a likely cause. It is notable that addition of equipotent halothane, enflurane,

Table 2. Drugs and Other Factors That Affect or Have No Proven Effect on Pacing Thresholds

Effect	Drugs	Other factors
Increase pacing threshold	Bretylium, encainide, flecainide, moricizine, propafenone, sotalol	Myocardial ischemia and infarction; progression of cardiomyopathy; hyperkalemia; severe acidosis or alkalosis; hypoxemia; after ICD shocks or external cardioversion or defibrillation
Possibly increase pacing threshold	β Blockers, lidocaine, procainamide, quinidine, verapamil	Myxedema; hyperglycemia
Possibly decrease pacing threshold	Atropine, catecholamines, glucocorticoids	Pheochromocytoma; hyperthyroid or other hypermetabolic states
No proven effect on pacing threshold	Amiodarone; anesthetic drugs, both inhalation and intravenous	

ICD = internal cardioverter–defibrillator.

Table 3. Examples of Elective Replacement Indicators That May Affect the Nominal Rate of Pacing

Stepwise change in pacing rate = the pacing rate changes to some predetermined fixed rate or some percentage decrease from the programmed rate.
Stepwise change in magnet rate = the magnet-pacing rate decreases in a stepwise fashion related to the remaining battery life.
Pacing mode change = DDD and DDDR pulse generators may automatically revert to another mode, such as VVI or VOO to reduce current drain and extend battery life.

or isoflurane to opiate-based anesthesia after cardiopulmonary bypass did not increase pacing thresholds.[20] Newer inhalation anesthetics, intravenous agents, narcotics, and anesthetic adjuncts have not been shown to affect thresholds. Finally, failure to capture may be misdiagnosed because of increased latency, which is the delay between stimulation and the onset of myocardial depolarization. Drugs or imbalances that increase pacing thresholds (table 2) may also increase latency.[3]

Pacing at Abnormal Rates. Abnormal pacing rates may be an intended or nonintended device function (table 1).[3,4] An apparently abnormal rate may correspond to the elective replacement indicator (table 3). Alternatively, output is not visible during bipolar pacing because of the low amplitude of bipolar pacing artifacts. Upper rate behavior is normal device function if it occurs in response to an adaptive-rate sensor. In a dual-chamber device, upper rate behavior may be due to pacemaker-mediated tachycardia or tracking atrial tachycardia (see Pacemaker-mediated Tachycardia).

Rarely, very rapid ventricular pacing may be due to pacemaker "runaway." Runaway can occur with a single- or dual-chamber pacemaker, requires at least two system component failures, and may trigger lethal arrhythmias.[3] Newer devices have runaway protection circuits that limit the stimulation rate to less than 200 beats/min. Pacemaker runaway is a major challenge.[21,22] With severe hemodynamic instability, the following measures may be considered: (1) connect the pacing leads to an external pulse generator and then cut or disconnect the leads from the implanted pulse generator or (2) first establish temporary transvenous pacing and then cut or disconnect the leads.[22]

Undersensing (Failure to Sense). The cardiac electrogram must have adequate amplitude and frequency content (slew rate) to be sensed properly.[3] A signal with apparently adequate amplitude may be markedly attenuated by the sense amplifier if it has a reduced slew rate. Therefore, the filtered signal may not be of sufficient size to be recognized as a valid event; consequently, undersensing may occur. Table 4 elaborates on previously identified causes of undersensing.[3,4] As with failure to capture, the onset of undersensing relative to the time of device implantation helps identify the cause. Undersensing occurring shortly after implantation may be due to lead dislodgement or malposition or to cardiac perforation. If it occurs later, it could be due to battery depletion, system component failure, or functional undersensing (see below). In addition, undersensing may be due to altered cardiac signal morphology secondary to disease progression; myocardial ischemia or infarction; inflammatory changes or fibrosis at the lead-tissue interface, transient metabolic or electrolyte imbalance; or the appearance of bundle-branch block or ectopy. Finally, external or internal cardioversion or defibrillation may temporarily or permanently disable sensing function because of transient saturation of the sense amplifier or direct damage to circuitry or the electrode–myocardial interface.

Normal pacemaker function may be misinterpreted as malfunction because of undersensing.[3] For example, reversion to an asynchronous pacing mode during continuous interference is necessary to protect the patient against inappropriate output inhibition. Other examples are triggered pacing modes with fusion or pseudofusion beats. With both, pacing artifacts appear within surface electrocardiographic P waves or QRS complexes. With fusion, there is simultaneous myocardial activation by paced and spontaneous depolarizations. With pseudofusion, pacing stimuli do not produce myocardial depolarization. Fusion or pseudofusion can occur because the pacemaker responds to intracardiac depolarization, which may appear isoelectric in more remote surface electrocardiographic leads. Finally, if too-long refractory periods are programmed, intrinsic cardiac events that should be sensed and should reset pacemaker timing do not. Therefore, the timing interval in effect will time out with delivery of a stimulus. This may be ineffective (pseudofusion) or only partially effective (fusion), de-

Table 4. Causes for Undersensing (Failure to Sense)

Inadequate signal amplitude or slew rate
Deterioration of intrinsic signal over time
Lead maturation
Inflammation, fibrosis
Progression of cardiac disease
Myocardial ischemia–infarction
New bundle branch block
Appearance of ectopic beats
Transient decrease in signal amplitude
After cardioversion or defibrillation shocks
Drugs, metabolic or electrolyte derangements that increase pacing thresholds (table 2)
Component malfunction
Battery depletion
Mechanical lead dysfunction
Recording artifact (pseudomalfunction)
Misinterpretation of normal device function
Triggered pacing modes
Fusion and pseudofusion beats
Functional undersensing (too long refractory periods)
Functional undersensing initiated by oversensing

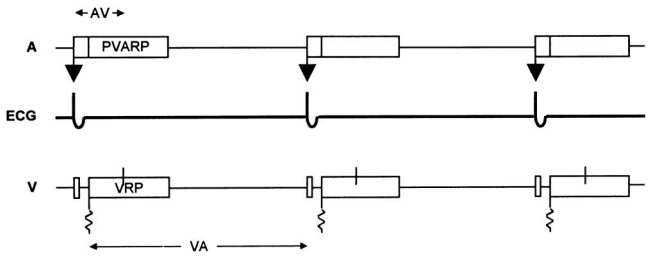

Fig. 1. Cross-talk inhibition. Immediately after the ventricular blanking period (short rectangle; ventricular channel timing overlay), the polarization potential after atrial stimulation is sensed by the ventricular channel (zigzag interference symbol). This is interpreted as an R wave, resetting the ventriculoatrial (VA) interval and ventricular refractory period (VRP). With complete arterioventricular (AV) block and no escape rhythm, ventricular asystole will occur, with atrial pacing faster than the programmed atrial rate. The short vertical lines in the ventricular timing overlay indicate ventricular stimuli inhibited by resetting of the VA interval. ECG = electrocardiography; PVARP = postventricular atrial refractory period. Reprinted with permission from Bernstein AD: Pacemaker timing cycles, American College of Cardiology Learning Center Highlights. Bethesda, American College of Cardiology.

pending on whether the chamber is completely or partially refractory at the time, respectively. This is an example of functional undersensing, because this behavior can be corrected by reprogramming.[3]

Oversensing. Any electrical signal of sufficient amplitude and frequency occurring during the pacemaker alert period can be sensed and can reset the timing. For example, ventricular depolarization sensed by an atrial demand pacemaker may cause inappropriate inhibition of stimulus delivery.[23] This is an example of "far-field" sensing. Far-field potentials arise in other cardiac chambers or are sensed skeletal myopotentials or other electromagnetic interference (EMI). In a device that provides atrial antitachycardia pacing, far-field sensing of ventricular depolarizations may lead to inappropriate delivery of therapy.[24] Far-field sensing of atrial depolarizations by VVI systems is unusual because of the smaller amplitude of P waves.[3] Myopotential inhibition has been reported with sensed succinylcholine-induced muscle fasciculations.[25] Myopotential inhibition is more likely with unipolar systems because of the proximity of the anode (pulse generator housing) to the pectoral muscles, diaphragm, or abdominal muscles, depending on pulse generator location.[3] In addition, intermittent contact between conducting elements of the pacing system may generate small potentials, termed "make-and-break" potentials. If sensed, these may cause inappropriate output inhibition. Any of the described oversensing can be confirmed by programming the pacemaker to an asynchronous mode or by magnet application. If the cause is oversensing, regular asynchronous pacing will resume. However, if the oversensing is due to other causes (*e.g.*, lead-conductor failure, pulse-generator failure, battery depletion, or an open circuit), there will be no pacing.

Malfunction in Dual-chamber Pacemakers. Crosstalk inhibition and pacemaker-mediated tachycardia are examples of malfunction that is specific to devices that both pace and sense in the atria and ventricles.

Crosstalk Inhibition. Crosstalk is the unexpected appearance in the atrial or ventricular sense channel or circuit of electrical signals present in the other.[3] For example, polarization potentials after stimulus delivery may be sensed in the ventricular channel during unipolar atrial pacing. If interpreted as spontaneous ventricular events, they can inhibit ventricular output. In the absence of an escape rhythm, there could be asystole, with only atrial pacing artifacts and P waves visible (fig. 1).[26–28] Such cross-talk inhibition can be prevented by increasing the ventricular sensing threshold, decreasing atrial output, or programming a longer ventricular blanking period, so long as these provide adequate safety margins for atrial capture and ventricular sensing. During the blanking period, ventricular sensing is disabled to avoid overloading of the sense amplifier by voltage generated by the atrial stimulus. If too short (fig. 1), this allows the atrial stimulus to be sensed in the ventricular channel, inappropriately resetting the ventriculoatrial (VA) interval without delivery of ventricular stimuli. If cross-talk cannot be prevented, many dual-chamber pacemakers have a cross-talk management feature, referred to in the pacing industry as nonphysiologic atrioventricular (AV) delay or ventricular safety pacing (fig. 2).[3]

Pacemaker-mediated Tachycardia. Pacemaker-mediated tachycardia is unwanted rapid pacing caused by the device or its interaction with the patient.[3] Pacemaker-mediated tachycardia includes pacemaker runaway; sensor-driven tachycardia; tachycardia during

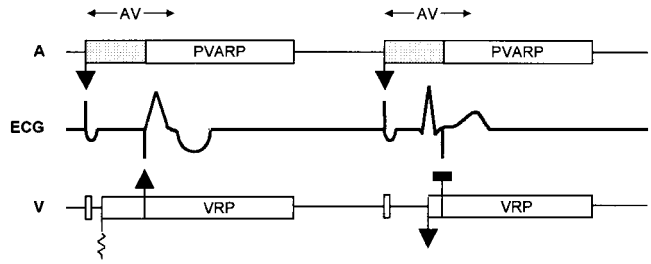

Fig. 2. Nonphysiologic arterioventricular (AV) delay (ventricular safety pacing). Whenever the ventricular channel senses anything during the initial portion of the programmed AV interval (shaded), such as cross-talk interference (zigzag symbol; ventricular timing overlay), a ventricular stimulus is triggered after an abbreviated AV interval to prevent asystole. In beat two, a conducted R wave is sensed and treated as cross-talk because the device does not distinguish spontaneous from paced beats. However, the triggered ventricular stimulus fails to depolarize refractory myocardium (black rectangle; ventricular timing overlay). Furthermore, its premature timing prevents stimulation during the T wave. ECG = electrocardiography; PVARP = postventricular atrial refractory period; VRP = ventricular refractory period. Reprinted with permission from Bernstein AD: Pacemaker timing cycles, American College of Cardiology Learning Center Highlights. Bethesda, American College of Cardiology.

Table 5. Mechanical or Physiologic Interference in the Perioperative Environment That May Be Sensed To Cause Inappropriate High-rate Pacing

Vibration sensor—piezocrystal
Direct pressure on device (prone position)
Bone hammers and saws
Bumpy ride (stretcher; hospital beds)
Impedance-based sensors—minute ventilation
Hyperventilation during induction of anesthesia
Mechanical ventilators
Electrocautery
Environmental 50–60 Hz electrical interference
Evoked QT interval
Catecholamine surge (stress, pain, pheochromocytoma)

magnetic resonance imaging (MRI) or due to tracking myopotentials or atrial tachydysrhythmias; and pacemaker-reentrant tachycardia.

Sensor-driven tachycardia. Adaptive-rate devices that sense vibration, impedance changes, or the QT interval may sense mechanical or physiologic interference to cause inappropriate high-rate pacing (table 5). It is advised that adaptive-rate pacing be disabled, even if electrocautery is not used during surgery.[3,29,30]

Magnetic resonance imaging. Powerful forces exist in the MRI suite, including static magnetic, gradient magnetic, and radiofrequency fields.[31–33] The static magnetic field may exert a torque effect on the pulse generator or close the magnetic reed switch to produce asynchronous pacing. Because devices today contain little ferromagnetic material, the former is considered unlikely.[33] Pacemaker leads can act as an antenna for the gradient magnetic field and radiofrequency field energy applied during MRI.[34] The gradient magnetic field may induce voltage in the pacemaker large enough to inhibit a demand pacemaker but unlikely to cause pacing.[32] The radiofrequency field, however, may generate enough current in the leads to cause pacing at the frequency of the pulsed energy (60–300 beats/min).[32,33] In dual-chamber pacemakers, this may affect one or both channels.[33] Finally, Achenbach *et al.*[31] documented an average temperature increase of 15°C at the electrode tip of 25 electrodes exposed to MRI, with a maximum increase of 63°C.

Tachycardia due to myopotential tracking. The atrial channel of a unipolar, dual-chamber device that tracks P waves (*i.e.*, programmed to VAT, VDD, or DDD) may sense myopotentials from muscle beneath the pulse generator, with triggered ventricular pacing up to the programmed maximum atrial tracking rate. This is unlikely with bipolar sensing, currently preferred by many implanting physicians.[3]

Tachycardia secondary to tracking atrial tachydysrhythmias. Atrial dysrhythmias, notably atrial fibrillation or flutter, may be tracked by ventricular pacing at or near the device's upper rate interval if programmed to an atrial-tracking mode (VAT, VDD, DDD). Medication to suppress the dysrhythmia or cardioversion may be necessary. In most instances, placing a magnet over the pulse generator to disable sensing (see Response of Pacemaker to Magnet Application) will terminate high-rate atrial tracking.[4] Some dual-chamber pacemakers have algorithms to detect fast, nonphysiologic atrial tachycardia and then switch to a nontracking pacing mode (*i.e.*, automatic mode-switching).[35–37] This is a useful feature with complete AV heart block and susceptibility to intermittent atrial tachyarrhythmias. Methods to prevent high rate atrial tracking are shown in figures 3 and 4.

Pacemaker-reentrant tachycardia. Pacemaker-reentrant tachycardia (PRT) can occur in any dual-chamber pacemaker programmed to an atrial-tracking mode (*e.g.*, VAT, VDD, DDD). It is a type of reentrant tachycardia that incorporates the pacemaker in the reentry circuit. The patient must have retrograde VA conduction through the AV node or an accessory AV pathway for PRT to occur. Approximately 80% of patients with sick sinus syndrome and 35% of those with AV block have retrograde VA conduction,[38–40] so more than 50% of patients receiving dual-chamber pacemakers are susceptible to PRT.[38] Furthermore, 5–10% of patients with absent VA conduction at the time of device implantation later acquire VA conduction.[38,41] Normally, PRT is initiated by a premature ventricular beat. This conducts to the atria and is sensed, provided it occurs outside the total atrial refractory period. The sensed retrograde P wave initiates the AV interval, which times out with

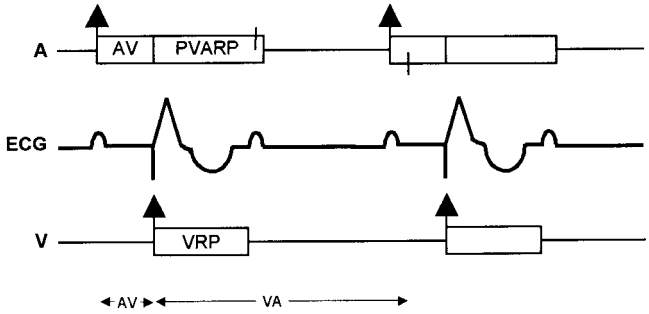

Fig. 3. Prevention of high-rate atrial tracking. When sensed P waves fall within the postventricular atrial refractory period (PVARP; first beat; short upward vertical line; atrial timing overlay), it does not trigger ventricular pacing or reset the arterioventricular (AV) interval. The next anticipated paced event is atrial stimulation at the end of the ventriculoatrial (VA) interval (second beat; short vertical line; atrial timing overlay). However, as shown, a spontaneous P wave is sensed, and this initiates a new AV interval before the VA interval times out with delivery of an atrial stimulus. Such intentional failure to track P waves within the PVARP produces "n-to-one block" (as shown, 2:1 block), limiting the minimum ventricular interval to the sum of the AV interval and PVARP. With AV block, as the atrial rate increases above the maximum tracking rate, only every other P wave is tracked, halving the paced ventricular rate. If still faster, two or more P waves may fall within the total atrial refractory period (AV + PVARP) and fail to trigger ventricular stimuli. ECG = electrocardiography; VRP = ventricular refractory period. Reprinted with permission from Bernstein AD: Pacemaker timing cycles, American College of Cardiology Learning Center Highlights. Bethesda, American College of Cardiology.

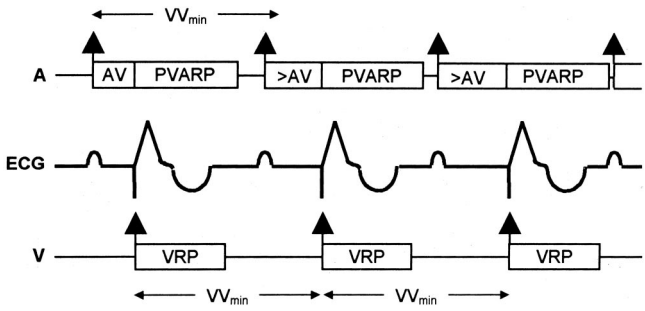

Fig. 4. Alternative prevention for high-rate atrial tracking. The minimum ventricular interval (VV_{min}) is lower than in fig. 3 but greater than the arterioventricular (AV) + postventricular atrial refractory period (PVARP; atrial channel). When the P-P interval is between AV + PVARP and VV_{min} (as shown), the P wave falls outside PVARP and is tracked by ventricular pacing, but after an extended AV interval (> AV), because the ventricular stimulus is delayed until the end of VV_{min}. Therefore, the interval between sensed P waves and the ventricular stimulus increases with each beat until a P wave falls within PVARP and is not tracked (not shown). This produces "pacemaker" or "pseudo" Wenckebach. ECG = electrocardiography; VRP = ventricular refractory period. Reprinted with permission from Bernstein AD: Pacemaker timing cycles, American College of Cardiology Learning Center Highlights. Bethesda, American College of Cardiology.

ventricular stimulation. PRT also occurs when paced ventricular beats are conducted back to the atria to trigger ventricular stimulation (fig. 5). To prevent PRT, a longer postventricular atrial refractory period is programmed,[3] but this limits the upper atrial tracking rate of the device. For example, some patients have VA conduction times greater than 430 ms.[3] Thus, if the postventricular atrial refractory period is 450 ms and the AV interval is 150 ms, the total atrial refractory period is 600 ms. This limits the maximum paced ventricular rate to 100 beats/min, possibly too slow for an active patient. In some devices, the postventricular atrial refractory period can be programmed to a longer duration after premature ventricular beats to prevent sensing of retrograde P waves. In addition, placing a magnet over the pulse generator will terminate PRT in most devices by disabling sensing and producing asynchronous (DOO) pacing. However, PRT may recur after the magnet is removed.

Response of Pacemaker to Magnet Application. Most pulse generators respond to magnet application by pacing asynchronously in a device-specific single-chamber (SOO) or dual-chamber pacing mode (DOO). (An SOO device paces a single chamber, either the atrium or the ventricle.) This corresponds to the programmed magnet mode.[42,43] For example, Thera DR or D devices (Medtronic, Minneapolis, MN) pace SOO or DOO at 85 beats/min.[43] However, with impending power source depletion, the magnet rate may differ, because it becomes the end-of-life (EOL) or elective replacement indicator. Again, the EOL or elective replacement indicator rate is characteristic for specific devices (*e.g.*, VOO at 65 beats/min for the Thera DR and D devices).[43]

The first few paced beats after magnet application may occur at a rate or output other than that seen later, providing device identification data on the strip-chart electrocardiographic recording as well as information regarding integrity of the pulse generator and leads.[42] Magnet application during electrocardiographic monitoring also confirms the ability of the system to capture the appropriate chamber at the programmed output settings.[43] In addition, magnets may be useful diagnostically and therapeutically.[43] In a patient whose intrinsic rhythm inhibits the device, magnet application may serve to identify the programmed mode when the correct programmer is not available for telemetry.[43] Furthermore, with device malfunction due to malsensing, magnet-initiated asynchronous pacing may temporarily correct the problem, confirming the presence of far-field sensing, cross-talk inhibition, T-wave sensing, or pacemaker-mediated tachycardia. Finally, in pacemaker-dependent patients, magnet application may ensure pacing if EMI inhibits output (*e.g.*, in surgical electrocautery). However, if the device has reverted to an asynchronous interference mode (fig. 6), the magnet response may not be the same as when the device is not in the interference mode.[42]

Finally, it is widely assumed that placing a magnet over any pacemaker pulse generator will invariably cause asynchronous pacing as long as the magnet remains in place. However, in some pacemakers, the magnet response may have been programmed off. In others a variety of magnet responses may have been programmed, some of which do not provide immunity to EMI sensing. In still others, the device will continue to pace asynchronously or pacing will cease after a programmed number of intervals.[42] Thus, if possible, one should determine before EMI exposure which pulse generator is present and what must be done to provide

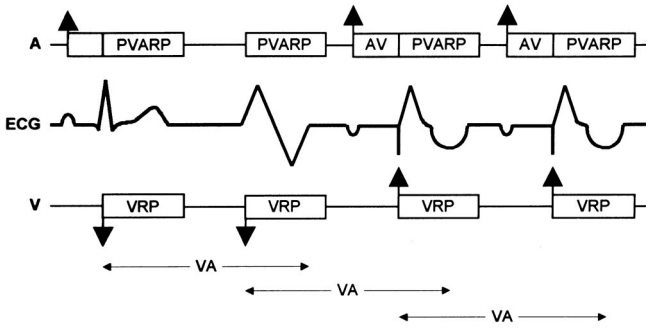

Fig. 5. Pacemaker-reentrant tachycardia occurs when a premature ventricular beat with retrograde P wave (second beat) resets the arterioventricular (AV) interval, triggering a ventricular stimulus earlier than expected (*i.e.*, when the ventriculoarterial [VA] interval times out). Pacemaker-reentrant tachycardia may also occur if paced ventricular beats produce retrograde P waves. ECG = electrocardiography; PVARP = postventricular atrial refractory period; VRP = ventricular refractory period. Reprinted with permission from Bernstein AD: Pacemaker timing cycles, American College of Cardiology Learning Center Highlights. Bethesda, American College of Cardiology.

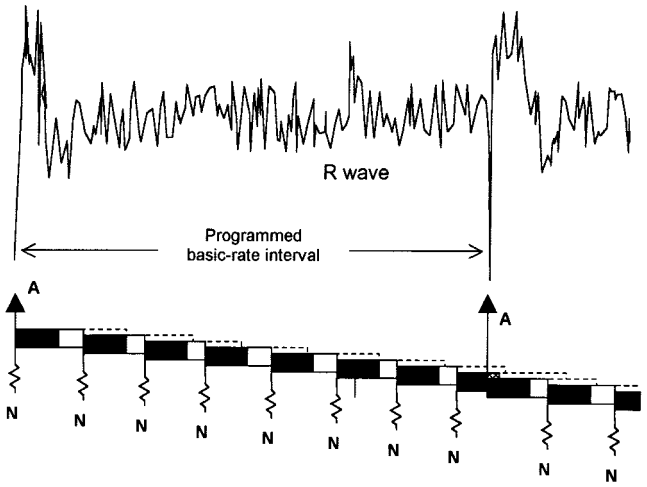

Fig. 6. Asynchronous interference mode in a VVI pacemaker, with temporary VOO pacing at the programmed basic-rate interval. The ventricular refractory period (VRP) begins with a noise-sampling period (black rectangles), during which time the sense amplifier is off. During the rest of the VRP, repeated noise (N) above a minimal frequency (*e.g.*, 7 Hz = 420 events/min) is interpreted as interference and restarts the VRP. Preempted portions of the VRP are indicated by dashed rectangles. Thus, so long as the noise persists, the device remains refractory, with the escape timing determined solely by the programmed basic-rate interval (asynchronous pacing at A). Note that a spontaneous R wave and the second paced beat occur in the noise-sampling period. Neither are sensed, but the latter initiates a new VRP. Reprinted with permission from Bernstein AD: Pacemaker timing cycles, American College of Cardiology Learning Center Highlights. Bethesda, American College of Cardiology.

protection. If this is not possible, then one can observe the magnet response during EMI to ascertain whether there is protection from EMI sensing. During electrocautery, for example, if despite magnet application the electrocautery triggers rapid pacing or inhibits pacing stimuli in a pacemaker-dependent patient, then the cautery must be limited to short bursts.

ICD Malfunction

Specific ICD malfunctions include inappropriate shock delivery, failure to deliver therapy, ineffective shocks, and interactions with drugs or devices affecting the efficacy of therapy.[44–46] Because all ICDs feature single- or dual-chamber pacing, there is potential for pacing malfunction as well (discussed previously).

Inappropriate Delivery of Shocks. Electrical artifacts consequent to lead-related malfunction may be interpreted as tachycardia, with inappropriate shock delivery.[46] Electrocautery artifact may be similarly misinterpreted.[47] Rapid supraventricular or nonsustained ventricular tachycardia (VT) may be misdiagnosed as sustained VT or ventricular fibrillation (VF),[44,46] especially if rate-only criteria are used for diagnosis.[48] Finally, R and T wave oversensing during ventricular bradycardia pacing has led to inappropriate shocks.[49]

Failure to Deliver Therapy or Ineffective Shocks. Magnet application may disable sensing and therefore the ability to deliver therapy (see Response of an ICD to Magnet Application). Especially after repeated subthreshold shocks for VF, tachydysrhythmias may be undersensed and may be the cause of failure to deliver therapy.[50] Exposure to diagnostic radiography or computed tomography scanning does not adversely affect shock delivery. Lead-related problems, including conductor fracture, lead migration, and lead insulation defects, may also be responsible for failure to deliver shocks or ineffective shocks.[46] Acute myocardial infarction, hypoxia, and severe acid-based or acute electrolyte imbalance may increase defibrillation thresholds, leading to ineffective shocks.[44] Any of the latter could also affect the rate or morphology of VT and the ability to diagnose VT. Finally, isoflurane and propofol anesthesia do not affect defibrillation thresholds.[51] The effect of other anesthetics or drugs used to supplement anesthesia is not known.

Drug–Device Interactions Affecting Efficacy of ICD Therapy. Antiarrhythmic drugs are prescribed along with ICDs to suppress (1) recurring sustained VT and the need for frequent shocks; (2) nonsustained VT that triggers unnecessary shocks and causes premature power source depletion; and (3) atrial fibrillation and inappropriate shocks.[46] In addition, they may be used to slow VT to make it better tolerated or more amenable to termination by antitachycardia pacing and to slow AV nodal conduction with atrial fibrillation. Possible adverse effects of combined drug and ICD therapy are that (1) amiodarone slows VT to below the programmed rate-detection threshold; (2) prodysrhythmia (the provocation of new or worse dysrhythmias) occurs with many antidysrhythmic drugs, increasing the need for shocks; (3) defibrillation thresholds may increase; (4) hemodynamic tolerance of VT may be reduced; (5) possible PR, QRS, or QT interval increases can cause multiple counting and spurious shocks; and (6) possible morphologic alterations or reductions in amplitude of cardiac electrograms may lead to failure to detect VT or VF.[44,46,52,53] Lidocaine, long-term amiodarone, class 1C drugs (*e.g.*, flecainide), and phenytoin increase defibrillation thresholds.[46] Class 1A drugs (*e.g.*, quinidine) and bretylium do not affect defibrillation thresholds.[46]

Device–Device Interactions Affecting Efficacy of Therapy. Formerly, pacemakers were used for bradycardia and antitachycardia pacing in patients with ICDs. Today, ICDs incorporate both pacing capabilities. However, there still may be an occasional patient with both devices.[46] Adverse interactions between devices include the following: (1) sensed pacing artifacts or depolarizations lead to multiple counting, misdiagnosis as VT/VF, and unnecessary shocks; (2) antitachycardia pacing artifacts may be misdiagnosed as VT, triggering shocks; (3)

ICD shocks may reprogram a pacemaker or cause failure to capture or undersensing.[46]

Response of an ICD to Magnet Application. Depending on the manufacturer and model of the ICD and how it is programmed (*e.g.*, magnet switch inactivated[54]), tachycardia sensing and delivery of therapy may be inactivated during exposure to a magnet. However, except for CPI devices (CPI, St. Paul, MN), sensing is inhibited only while the magnet is directly over the pulse generator.[55] With CPI devices, magnet application for less than 30 s temporarily disables sensing, whereas that longer than 30 s requires magnet reapplication for longer than 30 s to reactivate sensing.

Electromagnetic and Mechanical Interference

Pacemakers and ICDs are subject to interference from nonbiologic electromagnetic sources.[33] In addition, temperature extremes or irradiation may cause malfunction. In general, devices in service today are effectively shielded against EMI, and increasing use of bipolar sensing has further reduced the problem. EMI frequencies above 10^9 Hz (*i.e.*, infrared, visible light, ultraviolet, x-rays, and gamma rays) do not interfere with pacemakers or ICDs because the wavelengths are much shorter than the device or lead dimensions.[33] However, high-intensity therapeutic x-rays and irradiation can directly damage circuitry.[33]

EMI enters a pacemaker or ICD by conduction or radiation, depending on whether it is in direct contact with the source or the leads act as an antenna, respectively.[33] These devices are protected from EMI by shielding the circuitry, reducing the distance between the electrodes to minimize the antenna (*e.g.*, use of a bipolar *vs*. unipolar lead configuration for sensing), and filtering incoming signals to exclude noncardiac signals. If EMI does enter the pulse generator, noise protection algorithms in the timing circuit help reduce its effect on the patient. However, EMI signals between 5 and 100 Hz are not filtered because these overlap the frequency range of intracardiac signals. Therefore, EMI in this frequency range may be interpreted by a device as intracardiac signals, giving rise to abnormal behavior. Possible responses to EMI include (1) inhibition or triggering of pacing stimulation; (2) asynchronous pacing; (3) mode resetting; (4) damage to the pulse generator circuitry; and (5) triggering of unnecessary ICD shocks.[33]

Output Inhibition or Triggering and Asynchronous Pacing. To protect the pacemaker against inappropriate inhibition of paced output, some devices will revert to asynchronous pacing at the basic-rate interval when exposed to continuous EMI above a certain frequency (fig. 6). In others, rather than timing out at the basic-rate interval, repetitive detection of noise in the noise-sampling period causes temporary reversion to a specific "noise mode," typically VOO or DOO.[33] Whether EMI noise causes inhibition or asynchronous pacing depends on signal duration and field strength.[56] At the lowest field strength, there is no effect. However, as field strength increases, there is a greater tendency to inhibition because the noise may be sensed intermittently. Thus, it may not be sensed in the noise-sampling period but in the alert period before the next pacing pulse. With higher field strengths, noise is sensed continuously, and asynchronous pacing occurs. There is considerable variation between pacemakers and their susceptibility to noise.[33,56] Another approach for handling EMI is to program a triggered pacing mode (*i.e.*, VVT, AAT).[33] Continuous EMI will then trigger pacing at an upper rate determined by the ventricular or atrial refractory period. This is usually set at approximately 400 ms to limit the maximum triggered rate to 150 beats/min.

Mode Resetting and Reprogramming. EMI noise may cause a change to another mode that persists after the noise stops.[33] This is usually the backup or reset mode, often VVI, and the same as the elective replacement indicator or impending battery depletion mode.[33] If so, a pacemaker that has been affected by EMI may be wrongly assumed to have reached battery depletion and be replaced. Alternatively, an operator knowing that a device has been subject to EMI may reprogram one that has truly reached battery depletion.[33] Some pacemakers may be reset to the VOO mode, resulting in competition between paced and intrinsic rhythm. To our knowledge, EMI has not reprogrammed ICD antitachycardia therapies or affected bradycardia pacing in ICDs with single- or dual-chamber pacing capability. Although random reprogramming of a pre-1990s pacemaker by electrocautery EMI has occurred,[57] such reprogramming is highly unlikely with newer pacemakers, because unique radiofrequency sequences are required to enable programming of these devices.

Damage to Circuitry. There can be direct EMI damage to pacemaker or ICD circuitry, resulting in output failure, pacemaker runaway,[21] or other malfunction that necessitates pulse generator replacement.[33] Pacemakers and ICDs are protected from damage by high-energy current or shocks by special circuitry that electronically regulates the voltage entering the circuitry and should prevent high current from being conducted to the myocardium. Even so, extremely high energies may overcome such protection, causing damage to the device or heart. Bipolar devices appear more resistant than unipolar devices.[33]

Triggered Shocks. Reports of inappropriate ICD shocks due to EMI oversensing are infrequent.[47] A recent report described aborted shock delivery in a patient during facial electrosurgery.[58] In this case, EMI was interpreted by the device as VF, but spurious shocks were averted because the noise did not continue beyond the 9-s capacitor charging period.

Table 6. Potential Sources of Electromagnetic Interference and Their Effects on Pacemakers with Relevance to Perioperative Management

EMI Source	Generator Damage	Complete Inhibition	One-beat Inhibition	Asynch Pacing	Rate Increase
Electrocautery	Yes	Yes	Yes	Yes	Yes*†
External DCDF	Yes	No	No	Yes	Yes
MRI scanner	Possible	No	Yes	Yes	Yes
Lithotripsy	Yes†	Yes‡	Yes‡	Yes‡	Yes§
RF ablation	Yes	Yes	No	No	Yes
ECT	No	Yes	Yes	Yes	Yes†
TENS	No	Yes	No	Yes	Yes
Radiation therapy	Yes	No	No	No	Yes
Diagnostic radiation	No	No	No	No	Yes

* Impedance-based adaptive-rate pulse generators. † Piezoelectric crystal-based pulse generators. ‡ Remote potential for interference. § DDD mode only.
Asynch = asynchronous; DCDF = direct current cardioversion or defibrillation; MRI = magnetic resonance imaging; RF = radiofrequency; ECT = electroconvulsive therapy; TENS = transcutaneous electrical nerve stimulation.
Compiled from Hayes and Strathmore[33] and Levine and Love.[3]

Specific Electromagnetic and Mechanical Interference. EMI sources with relevance to perioperative physicians, along with their potential effects on pacemakers, are listed in table 6.[3,33] Although devices programmed to a bipolar lead configuration are more sensitive to locally generated signals, they are relatively insensitive to more remote signals. The most important EMI sources are surgical electrocautery and high-energy shocks for cardioversion or defibrillation. Mechanical ventilators and bone hammers or saws may interfere with vibration, acceleration, or minute-ventilation adaptive-rate pacemakers.

Surgical Electrocautery. The current generated by unipolar electrocautery is related to the distance and orientation of the cautery tool and grounding plate with respect to the pacemaker or ICD pulse generator and leads.[59] The greater the distance, the smaller is the voltage difference measured by the sensing circuit. High current is generated in the pulse generator circuitry if the cautery cathode (bovie tool) is close to the pulse generator, and even higher current is generated if the pulse generator is between the cathode and anode (grounding plate).[33] Bipolar cautery produces smaller voltage differences in the sensing circuits. Possible anomalous behavior with electrocautery EMI is described in the section Electromagnetic and Mechanical Interference. In addition, electrocautery may overwhelm the impedance-measuring circuit of a minute ventilation adaptive-rate pacemaker to cause pacing at the upper rate limit.[60] Finally, induced currents in the pacing leads may cause heating at the electrode-tissue interface, leading to tissue damage and elevated pacing or sensing thresholds. This is infrequently documented and usually transient.[33]

Defibrillator or Cardioverter Shocks. External cardioversion or defibrillation produces sufficient energy near a pacemaker or ICD to cause damage to the pulse generator or electrode–myocardial interface.[33] Transient elevation of thresholds for pacing and sensing is not uncommon after external or internal defibrillation.[33] Unipolar pacing systems are more susceptible.[33,61] ICDs deliver smaller amounts of energy but also can interfere with pacemaker function.[62] ICD shocks likely will activate the backup or reset modes or the elective replacement indicator. However, in devices with programmable lead configuration, unipolar pacing will be delivered by these modes. Because unipolar pacing pulses are more likely to be detected by an ICD, it is essential that a pacemaker in a patient with an ICD be programmed to a bipolar configuration or that the unipolar configuration first be tested to ensure there is no undersensing or oversensing by the defibrillator.[33] A pacemaker without programmable lead configurations is preferred for ICD patients.[33]

Miscellaneous EMI Sources. In general, it is recommended that patients with pacemakers not routinely undergo MRI.[33] Recent studies suggest that MRI may be safe, at least with some models of pacemakers or ICDs, provided the pulse generator and leads are not inside the magnet bore.[32,63] If MRI must be performed, program the device to its lowest voltage and pulse width or to the OOO mode if the patient has adequate spontaneous rhythm.[44,64] The pulse waveform should be closely monitored in pacemaker-dependent patients, and an external defibrillator must be available.[33,65,66] Device function must be checked after MRI.

Diagnostic radiation has no effect on pacemakers or ICDs. Therapeutic radiation did not affect the earliest pacemakers but can cause pulse generator failure in newer pacemakers that incorporate complementary metal oxide semiconductor–integrated circuit technology.[33,67–69] ICDs may also fail when exposed to radiation. Radiation causes leakage currents between the insulated parts of the circuit, leading to inappropriate charge accumulation in silicon oxide layers, which eventually leads to circuit failure. Therapeutic radiation involves doses up to 70 Gy, and pacemakers may fail with as little

as 10 Gy.[33] Failure is unpredictable and may involve changes in sensitivity, amplitude, or pulse width.[33] In addition, loss of telemetry, failure of output, or runaway rates may occur.[33,70] If unalterable malfunction occurs, replacement of the device is necessary.[33,44] Although some changes may resolve in hours, long-term reliability of the device is suspect. Before a course of radiation therapy is begun, the device must be identified and its function evaluated.[33,44,67,69,71] Radiation to any part of the body away from the site of the pulse generator should not cause a problem with the pulse generator, but the pulse generator should be shielded to avoid scatter.[33] If this is not possible, the device should be removed and reimplanted as far as possible from beams of radiation. The cumulative dose of radiation energy to which the pulse-generator is exposed should be recorded after each session. Device function should be monitored during therapy and regularly evaluated by telemetry during and after the course of treatment.

Adaptive-rate pacemakers that sense mechanical vibration or acceleration may malfunction during orthopedic surgery.[33] Positive-pressure ventilation may adversely affect measurement of minute ventilation by adaptive-rate pacemakers.[72–74] Electroconvulsive therapy appears safe for patients with pacemakers since little current flows within the heart because of the high impedance of body tissues.[33] However, the seizure may generate sufficient myopotentials for pacemaker inhibition (unipolar devices) or ventricular tracking (adaptive-rate devices).[33] Extracorporeal shock wave lithotripsy (ESWL) appears safe with pacemakers, provided shocks are synchronized to electrocardiographic R/S waves and dual-chamber devices have the cross-talk management feature enabled (fig. 2).[33,71] There may be a rate increase in an activity-sensing pacemaker after ESWL shocks. If this is undesirable, the adaptive-rate feature should be programmed off. Programming a DDD pacemaker to VVI, VOO, or DOO is advised to avoid irregularities in pacing rate, tracking of ESWL-induced supraventricular tachyarrhythmias, or triggering of ventricular output by sensed EMI.[33] It is best to disable tachycardia detection during ESWL and to thoroughly test the ICD following the procedure.[33] Transcutaneous electric nerve stimulation units probably can be used safely in patients with pacemakers or ICDs with bipolar lead polarity.[75,76] Nevertheless, it is reasonable to monitor pacemaker or ICD-dependent patients during initial application of transcutaneous electric nerve stimulation. Pacemaker-mediated tachycardia has been induced by intraoperative somatosensory evoked potential stimuli.[77] Finally, the effects of radiofrequency catheter ablation for termination of tachydysrhythmias are similar to those of electrocautery and include inappropriate inhibition, asynchronous pacing, and reset to a backup pacing mode.[78,79]

Management for the Patient with a Pacemaker or ICD

Preoperative Evaluation. Most patients with pacemakers or ICDs, especially the latter, have significant cardiovascular disease. Many have coexisting systemic disease as well. Special attention is paid to progression of disease, functional status, current medications, and compliance with treatment. No special laboratory tests or radiographs are required because the patient has an implanted pacemaker or ICD. However, results of recent 12-lead electrocardiography and any indicated diagnostic and recent laboratory tests (*e.g.*, for electrolyte status) should be available.

Device Identification and Evaluation. Unless the proposed surgery or intervention is truly emergent or poses little risk to the pulse generator or leads (*e.g.*, extremity, ophthalmologic, or other minimally invasive surgery in which bipolar cautery is used), identify the device, as well as date of and indication(s) for its implantation. Because all implanted pacemakers and ICDs are programmable, device interrogation with a compatible programmer is the most reliable, efficient way to determine function, battery status, programmed settings, pacing thresholds, lead impedances, electrode configuration, intrinsic rhythm, and magnet response. These should be recorded and rechecked after the surgery or intervention.

Most hospitals today have a pacemaker or ICD clinic or service (or access to one) that should be consulted for device interrogation and reprogramming. For the pacemaker-dependent patient, it is advised that the device be reprogrammed to an asynchronous mode if EMI is likely to cause significant malfunction (*e.g.*, unipolar electrocautery for surgery involving the upper abdomen or chest wall). For patients with adaptive-rate devices (including ICDs), this feature should be programmed off during surgery or exposure to other EMI that might cause device malfunction (table 6). Magnet-activated testing should be programmed off.[42] For patients with an ICD, tachycardia sensing should be programmed off. Further, if the patient is also pacemaker-dependent, an asynchronous pacing mode should be programmed if EMI might cause significant inhibition or other undesired function. After the planned procedure, it is necessary to have device function tested by qualified personnel, with the device reprogrammed or replaced if necessary.

In smaller hospitals and freestanding surgical or ambulatory care facilities, there may be no one immediately available to perform device interrogation and reprogramming. We strongly advise that under no circumstance should elective surgery or intervention proceed in this circumstance if the patient is at risk for device malfunction that could jeopardize his or her health. In other words, just as for the patient with uncontrolled hypertension or unstable coronary disease, it is necessary to optimize the patient's status before elective surgery or

Table 7. North American Manufacturers of Pacemakers and ICD, with 24-h Hotlines and Web Sites

Manufacturer	Hotline and Website	Products
Biotronik, Inc. 6024 Jean Road Lake Oswego, Oregon 97035-5369	1-800-547-9001 1-503-635-9936 (Fax) www.biotronik.com	Single- and dual-chamber pacemakers; single-chamber ICD
Guidant Corporation CRM* 4100 Hamline Avenue North St. Paul, Minnesota 55112-5798 (CPI, Intermedics)	1-800-CARDIAC (227-3422) 1-800-582-4166 (Fax) www.guidant.com	Single- and dual-chamber pacemakers (Intermedics, CPI); single- and dual-chamber ICDs (CPI)
Medtronic Corporation 7000 Central Avenue NE Minneapolis, Minnesota 55432	1-800-328-2518 1-800-824-2362 (Fax) www.medtronic.com	Single- and dual-chamber pacemakers; single- and dual-chamber ICDs
St. Jude Medical* Cardiac Rhythm Management Division 15900 Valley View Court Sylmar, California 91342 (Pacesetter, Ventritex)	1-800-777-2237 1-800-756-7223 (Fax) www.sjm.com	Single- and dual-chamber pacemakers (Pacesetter); single-chamber ICD (Ventritex)

* Parent company, with recently acquired or merged companies shown below in parentheses.

ICD = internal cardioverter–defibrillator.

intervention. In this case, however, instead of optimizing the patient's physical status, the physician is configuring a device to minimize risk for complications related to system failure or malfunction. If the planned surgery or intervention is urgent and risk of EMI-related malfunction certain, there still may be time to have the device interrogated and reprogrammed by qualified personnel. The next best strategy for reducing risk is to identify the device and contact the manufacturer for suggested management (table 7).

At the time of device implantation, all patients receive a card that identifies the model and serial numbers of the pacemaker or ICD, the date of implantation, and the implanting physician or clinic (fig. 7). The manufacturer also has this information in its registry. If the patient does not have an identification card, the information should be in the patient's medical records. If not, a chest radiograph of the pulse generator area may reveal the unique radiopaque code (*i.e.*, x-ray or radiographic "signatures") that can be used to identify the manufacturer and model of the device.[80] These radiographic signatures, which are on most pacemakers and ICDs in existence—as well as other useful information regarding specific devices, models, and leads (such as NBG code for functional capability, lead configuration, battery end-of-life or elective replacement indicator, and nominal longevity)—appear in generic reference guides available from all manufacturers listed in table 7. Consideration should be given to keeping a current guide in the vicinity of the operating suite or preoperative holding area for reference purposes. Once the device has been identified, the manufacturer should be contacted for further information through its Web site or telephone hotline (table 7).

If the surgery or procedure is truly emergent and it is not possible to identify the device, basic function of most suppressed pacemakers can be confirmed by placing a magnet over the pulse generator to cause asynchro-

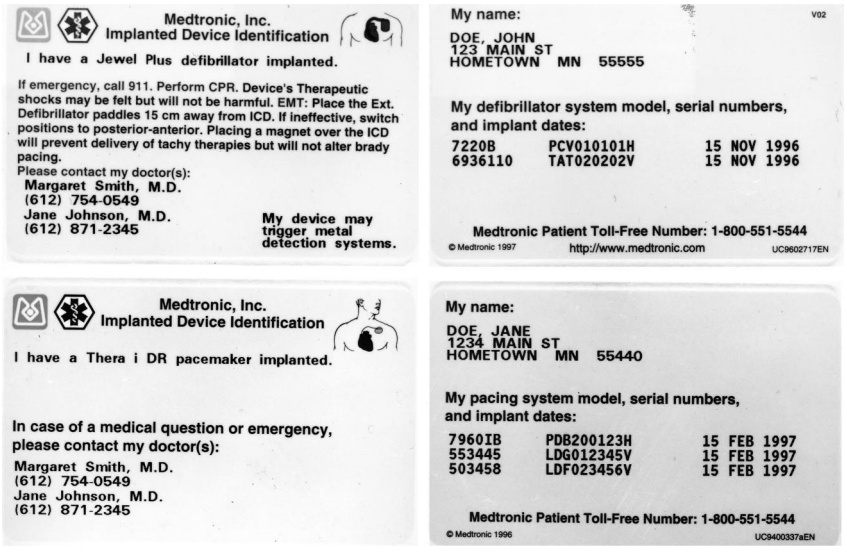

Fig. 7. Sample device identification cards for a Medtronic Jewel Plus internal cardioverter–defibrillator (*top*) and Thera I DR pacemaker (*bottom*). Front (*left*) and back (*right*) of respective cards. With this information, the manufacturer can be contacted through the toll-free number for patients (see cards) or *via* a hotline (table 7) for further advice concerning device management. Cards courtesy of Medtronic, Minneapolis, Minnesota.

nous pacing, provided the magnet function has not been programmed off. Cholinergic stimulation (*e.g.*, with Valsalva maneuver, carotid sinus massage, or 6–12 mg intravenous adenosine) might also be considered to slow the intrinsic rate sufficiently for release of pacing stimuli.

Perioperative Management: Surgery Unrelated to Device. The chief concern with perioperative management for the patient with a pacemaker or ICD is to reduce as much as possible the risk of adverse effects such as hemodynamic instability (resulting from inhibition or triggering of pacing stimuli or antitachycardia therapies) or upper rate pacing behavior. If EMI is likely to cause device malfunction and the patient does not have an adequate intrinsic rhythm, the pacemaker should be programmed to an asynchronous mode, preferably one that maintains AV synchrony, especially with impaired ventricular function. If the device is an ICD, tachycardia sensing should be programmed off. If the patient also requires pacing, an appropriate asynchronous mode should be programmed. If a pacemaker or ICD also has adaptive-rate pacing, this feature should be programmed off.

Because disabling ICD sensing will also prevent delivery of tachycardia therapies, an external cardioverter-defibrillator must be available. If it is not possible to reprogram a device through a compatible programmer and there is significant hemodynamic instability resulting from EMI-related malfunction that is largely unavoidable (namely there is massive hemorrhage: surgery is in the vicinity of the pulse generator or leads, and a short burst of electrocautery is impractical), then it is reasonable to place a magnet directly over the pulse generator of a pacemaker. This will cause most devices to pace asynchronously until the magnet is removed, unless the magnet mode has been programmed off. However, some devices will pace asynchronously only for a programmed number of intervals.[42] As for ICD, without knowing what device it is or how it is programmed, or what the magnet response is, it is advised that a magnet not be placed over the ICD pulse generator to disable tachycardia sensing (written communication, David L. Hayes, M.D., Professor of Medicine, Mayo Medical School, Rochester, MN, March 2001). Nonetheless, this must be considered if EMI triggers antitachycardia pacing or repeated shocks that destabilize the patient.

Unipolar electrocautery interference can be reduced by having the grounding plate located as far as possible from the cautery tool.[33] The pacemaker or ICD pulse generator and leads should not be between the bovie tool and grounding plate. Pacing function is confirmed by palpation of the pulse or by monitoring of the heart sounds or pulse waveform (*e.g.*, oximetry or direct arterial pressure). Only the lowest possible energies and brief bursts of electrocautery should be used, especially with hemodynamic instability due to related device malfunction. If electrocautery must be used in the vicinity of (less than 15 cm from) the pulse generator or leads, the device should be identified so that its response to sensed continuous, strong EMI (*i.e.*, backup or reset mode) will be known. If the backup pacing mode might compromise the patient by reduction of AV synchrony, asynchronous pacing, or too slow a rate, a compatible programming device must be available in the operating room, the pulse generator must be accessible to the programming head, and someone experienced in programming should be present.[33] Finally, a recent report suggests that the ultrasonic scalpel may provide a safe alternative to surgical electrocautery.[81] However, this requires more study before recommendations can be made. In addition, the ultrasonic scalpel may not be useful for all types of surgery.

External cardioverter/defibrillator shocks will probably cause at least temporary inhibition. Transient loss of capture or sensing should be anticipated, and the stimulus amplitude may need to be increased. This is done automatically by ICDs with a backup bradycardia pacing capability[71] (virtually all ICDs in service today). Pulse generator damage is related to the distance of the external paddles from the pulse generator. All device manufacturers recommend the anteroposterior paddle configuration, with the paddles located at least 10 cm from the pulse generator. Furthermore, it is advised that the lowest possible energies be used for cardioversion or defibrillation. After cardioversion or defibrillation the pacemaker or ICD must be interrogated to ensure proper function. Reprogramming or lead replacement may be necessary.[33]

Perioperative Management: Surgery Related to Device. Most pacemakers and ICDs have transvenous lead systems. A thoracotomy is no longer required for system implantation. Both the pulse generator and leads can be implanted with use of local anesthesia with conscious sedation.[82–86] However, a thoracotomy and general anesthesia are required for most infants and small children because epicardial lead systems are still widely used. General anesthesia or monitored anesthesia care and heavy sedation may be requested in some centers for system implantation or revision in adults, especially if the procedure involves extensive electrophysiologic testing with repeated induction of tachydysrhythmias and shocks. Therefore, the following management recommendations must be considered. (1) Temporary pacing is advised for disadvantageous bradycardia due to any cause. Alternatively, chronotropic drugs and backup external pacing should be available. (2) Reliable pulse monitoring (*i.e.*, direct arterial blood pressure monitoring or pulse oximetry) is necessary. Some centers require direct arterial blood pressure monitoring.[82] (3) For surface electrocardiographic monitoring, select the best leads for P waves and ischemia diagnosis. (4) Pulmonary artery catheters, formerly recommended,[47,87,88] are seldom used today because of the widespread use of nonthora-

Table 8. Suggested Management for Patients with Pacemakers or ICD Undergoing Unrelated Surgery

Elective Surgery*
Contact pacemaker or ICD clinic or manufacturer during the preoperative evaluation. Identify and interrogate the device, and reprogram if necessary (*i.e.*, nature or location of planned surgery, unipolar cautery, and so on).
With a pacemaker-dependent patient, reprogram the device to a triggered or asynchronous mode. Program magnet-activated testing and adaptive-rate pacing off.
With ICD, program tachycardia sensing off. Do not use magnet to disable sensing unless the magnet response is known. Have an external cardioverter–defibrillator available.
If possible, locate the cautery grounding plate so that the pulse generator and leads are not in the current pathway between it and the bovie tool. Also, the grounding plate should be located as far as possible from the pulse generator and leads. Use the lowest possible cautery energy and short bursts to minimize adverse effects of EMI.
Monitor arterial pulse waveform and heart sounds to detect EMI-related hemodynamic instability, which is unlikely. Should this occur, proceed as during urgent or emergent surgery (below).
If external defibrillation is required, locate defibrillation pads or paddles at least 10 cm from the pulse generator and implanted electrodes. Use apex- (anterior-) posterior position if possible. As near as possible, current flow between the paddles should be perpendicular to the major lead axis.
After surgery, arrange to have device function tested by pacemaker or ICD clinic, and reprogram or replace the device if necessary.
Urgent or Emergent Surgery
If time permits, identify the implanted device from the patient's medical record, identification card, or "x-ray signature." Contact the manufacturer (table 7) and follow their recommendations.
Institute electrocardiography and arterial pulse waveform and heart sounds monitoring. If no pacing artifacts are seen and the device is a pacemaker, place a magnet over the pulse generator to determine whether the device is functional. Alternatively, consider a vagal maneuver or drug to slow the intrinsic rate.
If EMI-related pacemaker malfunction is hemodynamically destabilizing, program the device to a triggered or asynchronous mode. If this is not possible, a magnet over the pulse generator will convert many (but not all) devices to an asynchronous pacing mode.
If the device is an ICD, without knowing what it is or how it is programmed, or what the magnet response is, it is generally advised not to place a magnet over the pulse generator to disable tachycardia sensing. However, this should be considered if repeated shocks or antitachycardia pacing in response to sensed EMI are hemodynamically destabilizing.
After surgery, arrange to have device function tested by pacemaker or ICD clinic, and reprogram or replace the device if necessary.

* It is assumed that for patients having elective surgery and at risk for related device malfunction, the pacemaker or ICD clinic or manufacturer will have been consulted regarding appropriate perioperative management, including device interrogation and reprogramming if necessary.

ICD = internal cardioverter–defibrillator; EMI = electromagnetic interference.

cotomy lead systems and smaller pulse generators. In addition, pulmonary artery catheters may interfere with ICD lead positioning. (5) If the procedure requires multiple defibrillation threshold testing and extensive subpectoral dissection, general anesthesia should be considered.[82,89] (6) Techniques and drugs for monitored anesthesia care or general anesthesia vary among institutions. Available inhalation or intravenous agents are not known to increase defibrillation thresholds[90] and are selected more with a view to hemodynamic tolerance. Older volatile agents (halothane, enflurane, and isoflurane) affected inducibility of ventricular tachydysrhythmias,[91–94] which is a consideration during electrophysiologic testing. Whether desflurane and sevoflurane have such an effect is not known. It is possible that anesthetic drugs could affect the morphology of sensed intracardiac electrograms, but to our knowledge, this has not been examined. Small amounts of lidocaine for vascular access should not affect electrophysiologic testing or defibrillation thresholds; larger amounts of lidocaine or bupivacaine for regional anesthesia (*e.g.*, field blocks) might.[90] Although it has not been reported, procaine probably does not because it is similar to procainamide, which also does not affect defibrillation thresholds.[90] (7) An external cardioverter–defibrillator must be available and functioning. (8) If the ICD is active at any time during the procedure, tachycardia sensing should be disabled when unipolar electrocautery is used.

Summary and Recommendations

Perioperative management for patients with cardiac rhythm management devices may be challenging, given the increased sophistication of these devices and the potential for adverse effects during exposure to electromagnetic or mechanical interference. Improved shielding and increased use of bipolar lead configurations with current devices has reduced the risk of device malfunction during exposure to EMI. Nevertheless, perioperative device malfunction is a real possibility without appropriate precautions. First, it is necessary to understand why the device was prescribed and what it is expected to do for the patient and medical circumstances. Second, basic understanding of pacemaker timing and how ICDs detect and diagnose dysrhythmias is required for recognition of device malfunction. These considerations are addressed in the first installment of this article. Herein we have discussed specific pacemaker and ICD malfunctions and EMIs that are likely to be encountered by anesthesiologists. In addition, we have outlined management for patients undergoing surgery related or unrelated to such a device. For the latter, suggested management is summarized in table 8. However, anesthesiologists must recognize that this is a very complex and constantly evolving field of technology. It is strongly encouraged that they make use of resources available to them for advice regarding perioperative management issues. Thus, whenever pos-

sible, the clinic or service responsible for pacemaker and ICD follow-up and the device manufacturers should be consulted regarding optimal management for specific devices and circumstances.

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