Anesthesiology 2001; 95:1265–80

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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Received from the Department of Anesthesiology, Medical College of Wisconsin, Milwaukee, Wisconsin, and the Department of Surgery, University of Medicine and Dentistry of New Jersey, Newark, New Jersey. Submitted for publication March 30, 2000. Accepted for publication June 25, 2001. Support was provided solely from institutional and/or departmental sources.

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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.

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.

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]

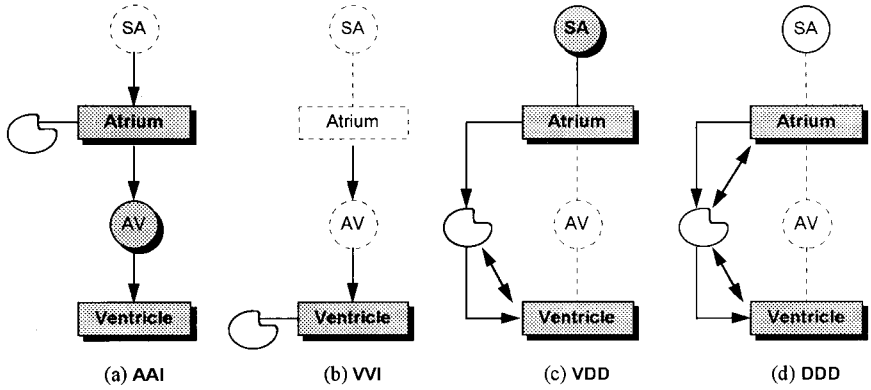

Fig. 1. Examples of antibradycardia pacing modes. (*A*) Atrial-inhibited (AAI) pacing for sinus arrest or bradycardia. The pulse generator is shown with atrial leads only. The atrium is paced, unless pacing is inhibited by sensed spontaneous atrial depolarizations. (*B*) Ventricular-inhibited (VVI) pacing 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.

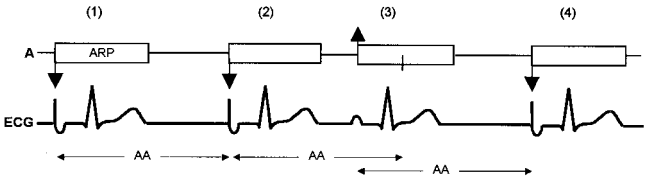

Fig. 2. Depiction of atrial-inhibited pacing, as for sinus bradycardia with intact atrioventricular conduction. In the first beat, the atrium (A) is paced (by convention, an arrow pointing toward the electrocardiogram [ECG] in the atrial timing diagram). The atrial refractory period (ARP) prevents conducted 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

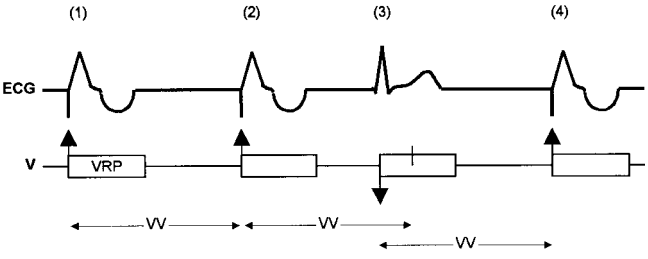

Fig. 3. Depiction of ventricular-inhibited pacing, as for atrioventricular heart block with atrial fibrillation. In the first beat, the ventricle (V) is paced. The pacemaker's ventricular refractory period (VRP) prevents the ensuing T wave from being interpreted as an R wave and inappropriately resetting 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.

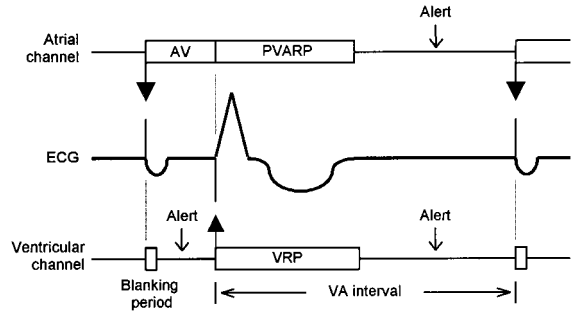

Fig. 4. Basic timing of a dual-chamber pacemaker, with pacing and sensing in both chambers. Atrial and ventricular stimuli are shown in the timing diagrams by arrows pointing toward the electrocardiogram (ECG) from above and below, respectively. The programmed atrioventricular (AV) interval provides 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

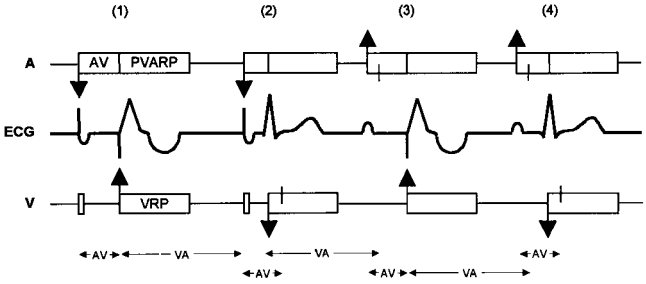

Fig. 5. Basic patterns in dual-chamber pacing. The first beat is fully paced and is an example of atrioventricular (AV) sequential pacing. In the second beat, a spontaneous R wave is sensed in the ventricular (V) channel before the atrioventricular interval times out, initiating a new VA interval (the 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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