AHA Medical/Scientific Statement

Special Report

Guidelines for Implantation of Cardiac Pacemakers and Antiarrhythmia Devices

A Report of the American College of Cardiology/American Heart Association Task Force on Assessment of Diagnostic and Therapeutic Cardiovascular Procedures (Committee on Pacemaker Implantation)

Committee Members
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Preamble
It is becoming more apparent each day that despite a strong national commitment to excellence in health care, the resources and personnel are finite. It is therefore appropriate that the medical profession examine the impact of developing technology on the practice and cost of medical care. Such analysis, carefully conducted, could potentially have an impact on the cost of medical care without diminishing the effectiveness of that care.

To this end, the American College of Cardiology and the American Heart Association in 1980 established a Task Force on Assessment of Diagnostic and Therapeutic Cardiovascular Procedures with the following charge:
The Task Force of the American College of Cardiology and the American Heart Association shall define the role of specific noninvasive and invasive procedures in the diagnosis and management of cardiovascular disease.
The Task Force shall address, when appropriate, the contribution, uniqueness, sensitivity, specificity, indications, contraindications and cost-effectiveness of such specific procedures.
The Task Force shall include a Chairman and six members, three representatives from the American Heart Association, and three representatives from the American College of Cardiology. The Task Force may select ad hoc members as needed upon the approval of the Presidents of both organizations. Recommendations of the Task Force are forwarded to the President of each organization.
The members of the Task Force are George A. Keller, MD, Roman W. DeSanctis, MD, Harold T. Dodge, MD, J. Ward Kennedy, MD, T. Joseph Reeves, MD, Sylvan Lee Weinberg, MD, and Charles Fisch, MD, chairman.
This document was reviewed by the officers and other responsible individuals of the two organizations and received final approval in March 1991. It is being published simultaneously in Circulation and the Journal of the American College of Cardiology. The potential impact of this document on the practice of cardiology and some of its unavoidable shortcomings are clearly set out in the introduction.

Charles Fisch, MD

1. Introduction
This is a revision of the 1984 Guidelines for Permanent Cardiac Pacemaker Implantation. The Joint Subcommittee of the American College of Cardiology and the American Heart Association was chaired by Robert L. Frye, MD, and, in addition to the members of the Joint Task Force, included the following ad hoc members: John J. Collins, MD, Leonard S. Dreifus, MD, Leonard S. Gettes, MD, Paul C. Gillette, MD, and Victor Parsonnet, MD. The present document was reviewed by selected consultants: S. Serge Barold, MD, John D. Fisher, MD, Roger A. Freeman, MD, Richard M. Luceri, MD, Seymour Furman, MD, and Melvin M. Schein-
man, MD. Many of their suggestions have been incorporated in the final text.

**Indications for Permanent Cardiac Pacemakers and Anti-tachycardia Devices**

These recommendations are the subject of this report. Because of the multitude, complexity, and initial cost of currently available pacing systems, the committee has included recommendations regarding selection of devices for specific clinical problems in which pacing or defibrillation is indicated. The committee recommendations are based on current evidence in relation to both knowledge of the natural history of disorders of cardiac rhythm as well as the characteristics of currently available devices. Because of continuing research and development, some of these recommendations may be subject to further modification in the future.

These recommendations apply to permanent pacing and anti-tachycardia devices in the management of chronic, though sometimes intermittent, disorders of cardiac rhythm. For the most part, they do not pertain to identifiable factors that cause transient depression of cardiac impulse formation and conduction, such as drugs, electrolyte or endocrine imbalances, infection, or the acute phase of myocardial infarction. The decision to implant a pacemaker or anti-tachycardia device must be reached by scrupulous adherence to a fundamental principle of clinical medicine: the demand for careful, thoughtful analysis of each patient. Attention must be given to the general medical, emotional, and mental state of the patient as well as to the specifics of the cardiac rhythm disturbance before a proper management decision can be made.

The committee has not offered any recommendations regarding resources required to perform pacemaker or anti-tachycardia device insertions, training of individuals for this purpose, or the appropriate follow-up and monitoring of patients with permanent pacemakers. These critically important topics have been addressed elsewhere. The committee unanimously urges careful review and adoption of the resource guidelines by all institutional administrators, physicians, and surgeons who are responsible for anti-tachycardia device therapy.

The clinical symptoms associated with bradycardia need definition at the outset because they recur throughout the report as major indications for permanent pacemaker therapy. In this report, the term “symptomatic bradycardia” refers to the following clinical manifestations that are directly attributable to the slow heart rate: transient dizziness, light-headedness, near syncope or frank syncope as manifestations of transient cerebral ischemia, and more generalized symptoms such as marked exercise intolerance or frank congestive heart failure. It is acknowledged, however, that some patients may have been symptomatic only in retrospect. How to recognize these individuals a priori is not within the scope of this document. As is mentioned elsewhere, it must be assumed that physicians who implant anti-tachycardia devices are experts and will be skillful enough to make this distinction.

Indications for permanent pacemakers have been grouped according to the following classifications:

Class I: Conditions for which there is general agreement that permanent pacemakers or anti-tachycardia devices should be implanted

Class II: Conditions for which permanent pacemakers or anti-tachycardia devices are frequently used but there is divergence of opinion with respect to the necessity of their insertion

Class III: Conditions for which there is general agreement that pacemakers or anti-tachycardia devices are unnecessary

In those patients being considered for pacemakers or anti-tachycardia devices, decision making may be influenced by the following additional factors:

1. Overall physical and mental state of the patient, including the absence of associated diseases that may result in a limited quality or prognosis for life
2. Presence of associated underlying cardiac disease that may be affected adversely by bradycardia
3. Desire of the patient to operate a motor vehicle
4. Remoteness of medical care, including patients who travel widely or live alone who therefore might be unable to seek medical help if serious symptoms arise
5. Necessity for administering medication that may depress escape heart rates or aggravate atrioventricular (AV) block
6. Slowing of the basic escape rates
7. Significant cerebrovascular disease that might result in a stroke if cerebral perfusion were to suddenly decrease
8. Desires of the patient and family

This report provides brief definitions and descriptions of specific clinical situations in which pacing may be considered and literature references that document the basis for the recommendation.

II. Pacing in Acquired Atrioventricular Block in Adults

Atrioventricular block is classified as first-degree, second-degree, or third-degree (complete) heart block; anatomically, it is defined as supra-His, intra-His, and infra-His. Second-degree heart block may be further classified as type I (progressive prolongation of PR interval before a blocked beat) or type II (no progressive prolongation of PR interval before blocked beats) and is usually associated with a wide QRS complex. Advanced second degree block refers to the block of two or more consecutive P waves. Patients with abnormalities of AV conduction may be asymptomatic or they may experience serious symptoms related to profound bradycardia or ventricular arrhythmias, or both. Decisions regarding the need for a pacemaker are influenced most importantly by
the presence or absence of symptoms that are directly attributable to bradycardia. It is clearly documented4-8 that patients with complete heart block and syncope have an improved survival with permanent pacing. There is no evidence to suggest that survival is prolonged with pacemakers in patients with isolated first-degree AV block. The prognosis in type I second-degree AV block, when due to AV node delay, tends to be benign.9-11 However, in patients with type II second-degree AV block (either intra- or infra-His), symptoms are frequent, prognosis is compromised, and progression to complete heart block is common.9,11,12

Recommendations for inserting a permanent pacemaker in patients with AV block with acute myocardial infarction or congenital AV block are discussed in a separate section. Atrioventricular block in the presence of supraventricular tachyarrhythmia does not constitute an indication for pacemaker insertion except as specifically defined in the recommendations that follow.

**Indications for Permanent Pacing in Acquired Atrioventricular Block in Adults**

**Class I**

A. Complete heart block, permanent or intermittent, at any anatomic level, associated with any one of the following complications:

1. Symptomatic bradycardia (discussed in the Introduction). In the presence of complete heart block, symptoms must be presumed to be due to the heart block unless proved to be otherwise

2. Congestive heart failure

3. Ectopic rhythms and other medical conditions that require drugs that suppress the automaticity of escape pacemakers and result in symptomatic bradycardia

4. Documented periods of asystole ≥3.0 seconds or any escape rate <40 beats/min in symptom-free patients

5. Confusional states that clear with temporary pacing

6. Post-AV junction ablation, myotonic dystrophy

B. Second-degree AV block, permanent or intermittent, regardless of the type or the site of block, with symptomatic bradycardia

C. Atrial fibrillation, atrial flutter or rare cases of supraventricular tachycardia with complete heart block or advanced AV block, bradycardia, and any of the conditions described under IA. The bradycardia must be unrelated to digitalis or drugs known to impair AV conduction

**Class II**

A. Asymptomatic complete heart block, permanent or intermittent, at any anatomic site, with ventricular rates of 40 beats/min or faster

B. Asymptomatic type II second-degree AV block, permanent or intermittent

C. Asymptomatic type I second-degree AV block at intra-His or infra-His levels

**Class III**

A. First-degree AV block (see section IV on bifascicular and trifascicular block

B. Asymptomatic type I second-degree AV block at the supra-His (AV node) level

**III. Pacing in Atrioventricular Block Associated With Myocardial Infarction**

Indications for permanent pacing after myocardial infarction in patients experiencing AV block are related in large measure to the presence of intraventricular conduction defects. The requirement for temporary pacing in acute myocardial infarction does not by itself constitute an indication for permanent pacing. The long-term prognosis in survivors of acute myocardial infarction who have had AV block is related primarily to the extent of myocardial injury and the character of intraventricular conduction disturbances rather than to the AV block itself.6,13-16 Patients with acute myocardial infarction who have intraventricular conduction defects, with the exception of isolated left anterior hemiblock, have an unfavorable short- and long-term prognosis, and an increased incidence of sudden death.12-14 This unfavorable prognosis is not necessarily due to the development of high-grade AV block, although the incidence of such block is higher in postinfarction patients with abnormal intraventricular conduction.14,17 Unlike some other indications for permanent pacing, the criteria in patients with myocardial infarction and AV block do not necessarily depend on the presence of symptoms.

**Indications for Permanent Pacing After Myocardial Infarction**

**Class I**

A. Persistent advanced second-degree AV block or complete heart block after acute myocardial infarction12-14 with block in the His-Purkinje system (bilateral bundle branch block)14-17

B. Patients with transient advanced AV block and associated bundle branch block14,15

**Class II**

A. Patients with persistent advanced block at the AV node16

**Class III**

A. Transient AV conduction disturbances in the absence of intraventricular conduction defects14

B. Transient AV block in the presence of isolated left anterior hemiblock13

C. Acquired left anterior hemiblock in the absence of AV block

D. Patients with persistent first-degree AV block in the presence of bundle branch block not demonstrated previously14
IV. Pacing in Bifascicular and Trifascicular Block (Chronic)

Bifascicular and trifascicular block refer to electrocardiographic (ECG) evidence of impaired conduction below the AV node in two or three of the fascicles of the right and left bundles. In patients with such ECG abnormalities, there is convincing evidence that advanced heart block with symptoms due to the block is associated with a high death rate and a significant incidence of sudden death.5,19

Syncope is common in patients with bifascicular block. Usually it is not recurrent or associated with an increased incidence of sudden death.20–32 It has been suggested that pacing relieves the transient neurologic symptoms but does not reduce the frequency of sudden death.23 However, there is convincing evidence5 that in the presence of complete heart block, syncope is associated with an increased incidence of sudden death. Thus, being unable to define the cause of syncope in the presence of bifascicular or trifascicular block, it appears reasonable to assume that the syncope may be due to transient complete heart block and thus, in the opinion of some investigators,24,25 prophylactic permanent pacing is indicated.

Although complete heart block is most often preceded by bifascicular block, the evidence is impressive that the rate of progression of bifascicular block to complete heart block is low. Furthermore, no single clinical or laboratory variable, including bifascicular block, identifies patients at high risk of death from a future bradyarrhythmia due to the bundle branch block.26

Of the many laboratory variables, the PR and HV intervals have been singled out as possible predictors of complete heart block and sudden death. Evidence indicates that PR interval prolongation is common in patients with bifascicular block. However, the prolongation is often at the level of the AV node. Furthermore, there is no correlation between the PR and HV intervals or between the length of the PR interval and progression to complete heart block and incidence of sudden death.27,28,32 Although most patients with chronic or intermittent complete heart block demonstrate prolongation of the HV interval during anterograde conduction, some investigators30,31 have suggested that asymptomatic patients with bifascicular block and a prolonged HV interval should be considered for permanent pacing, especially if the HV interval is ≥100 msec.32 The evidence indicates that although the prevalence of prolonged HV is high, the incidence of progression to complete heart block is low. HV prolongation accompanies advanced cardiac disease and is associated with an increased death rate; death is not sudden and is due to the underlying heart disease and not to complete heart block.20,23,26,27,32,33

Attrial pacing as a means of identifying patients at increased risk of future complete heart block probably is not justified. The chance of inducing distal heart block with pacing is low.20,31,34,35 In fact, pacing often fails to induce distal His block in patients with documented abnormal conduction of the His-Purkinje system.20,30,31,36,37 Furthermore, failure to induce distal block cannot be taken as evidence that the patient will not develop complete heart block. However, if atrial pacing induces infra-His block, some consider this to be an indication for pacing.38

Indications for Permanent Pacing in Bifascicular and Trifascicular Block

Class I

A. Bifascicular block with intermittent complete heart block associated with symptomatic bradycardia (as defined)

B. Bifascicular or trifascicular block with intermittent type II second-degree AV block without symptoms attributable to the heart block

Class II

A. Bifascicular or trifascicular block with syncope that is not proved to be due to complete heart block, but other possible causes for syncope are not identifiable

B. Markedly prolonged HV (>100 msec)32

C. Pacing-induced infra-His block38

Class III

A. Fascicular block without AV block or symptoms

B. Fascicular block with first-degree AV block without symptoms

V. Pacing in Sinus Node Dysfunction

Sinus node dysfunction (sick sinus syndrome) constitutes a spectrum of cardiac arrhythmias, including sinus arrhythmia, sinus arrest, sinoatrial block, and paroxysmal supraventricular tachycardia alternating with periods of bradycardia or even asystole. Patients with this condition may be symptomatic from paroxysmal tachycardia, bradycardia, or both. Correlation of symptoms with the specific arrhythmias is essential, although this may be difficult because of the intermittent nature of the episodes. Sinus bradycardia is accepted as a physiologic finding in trained athletes, who not uncommonly have heart rates of 40–50 beats/min while at rest and awake and may have sleeping rates as slow as 30–43 beats/min with sinus pauses or type I AV block producing asystolic intervals as long as 1.6–2.8 seconds.39–41 These characteristics are due to increased vagal tone. Permanent pacing in patients with sinus node dysfunction may not necessarily result in improved survival time,42,43 but severe symptoms related to bradycardia may be relieved.44,45

Indications for Permanent Pacing in Sinus Node Dysfunction

Class I

A. Sinus node dysfunction with documented symptomatic bradycardia. In some patients this will occur
as a consequence of long-term (essential) drug therapy of a type and dose for which there are no acceptable alternatives.

Class II

A. Sinus node dysfunction, occurring spontaneously or as a result of necessary drug therapy, with heart rates <40 beats/min when a clear association between significant symptoms consistent with bradycardia and the actual presence of bradycardia has not been documented

Class III

A. Sinus node dysfunction in asymptomatic patients, including those in whom substantial sinus bradycardia (heart rate <40 beats/min) is a consequence of long-term drug treatment

B. Sinus node dysfunction in patients in whom symptoms suggestive of bradycardia are clearly documented not to be associated with a slow heart rate

VI. Pacing in Hypersensitive Carotid Sinus and Neurovascular Syndromes

The hypersensitive carotid sinus syndrome is defined as syncope resulting from an extreme reflex response to carotid sinus stimulation. It is an uncommon cause of syncope. There are two components of the reflex:

1. Cardioinhibitory, resulting from increased parasympathetic tone and manifested by slowing of the sinus rate or prolongation of the PR interval and advanced AV block, alone or in combination

2. Vasodepressor, secondary to a reduction in sympathetic activity resulting in hypotension

Before concluding that permanent pacing is clinically indicated, the physician must determine the relative contribution of the two components of carotid sinus stimulation to the individual patient’s symptom complex. Hyperactive response to carotid sinus stimulation is defined as asystole due to sinus arrest or AV block of more than 3 seconds, or a substantial symptomatic decrease in systolic blood pressure, or both. Such heart rate and hemodynamic responses may occur in normal subjects and patients with coronary artery disease; a conclusion of a cause-and-effect relation between the hypersensitive carotid sinus and the patient’s symptoms must be made with great caution. Spontaneous syncope that is then reproduced by carotid sinus stimulation should alert the physician to the presence of this syndrome. Minimal pressure on the carotid sinus in elderly patients or patients receiving digitalis may result in marked changes in heart rate and blood pressure, yet not be of clinical significance. Permanent pacing for patients with pure excessive cardioinhibitory response to carotid stimulation is effective in relieving symptoms. Because 10–20% of patients with this syndrome may have an important vasodepressor component of this reflex response, it is necessary to define this component before concluding that all symptoms are related to asystole alone. In patients whose reflex response includes both cardioinhibitory and vasodepressor components, attention to the latter is essential for effective therapy in patients undergoing permanent pacing.

Indications for Permanent Pacing in Hypersensitive Carotid Sinus and Neurovascular Syndromes

Class I

A. Recurrent syncope associated with clear, spontaneous events provoked by carotid sinus stimulation; minimal carotid sinus pressure induces asystole of >3 seconds’ duration in the absence of any medication that depresses the sinus node or AV conduction

Class II

A. Recurrent syncope without clear, provocative events and with a hypersensitive cardioinhibitory response

B. Syncope with associated bradycardia reproduced by a head-up tilt with or without isoproterenol or other forms of provocative maneuvers and in which a temporary pacemaker and a second provocative test can establish the likely benefits of a permanent pacemaker

Class III

A. A hyperactive cardioinhibitory response to carotid sinus stimulation in the absence of symptoms

B. Vague symptoms, such as dizziness or light-headedness, or both, with a hyperactive cardioinhibitory response to carotid sinus stimulation

C. Recurrent syncope, light-headedness, or dizziness in the absence of a cardioinhibitory response

VII. Use of Pacemakers in Children

Although the indications for pacemakers in children are similar to those in adults, there are some special considerations. As in the adult, the optimal indication for pacemaker implantation in a child is the concurrent observation of symptoms with bradycardia—for example, syncope and complete AV block or syncope with severe bradycardia (30 beats/min). Concurrence of symptoms and bradycardia can be determined by 24-hour ambulatory electrocardiography or transtelephonic electrocardiography.

Sinus node dysfunction (sick sinus syndrome), although becoming more frequently recognized in pediatric patients, is not in and of itself an indication for pacemaker implantation. In patients with sinus node dysfunction, even greater emphasis is placed on concurrence of sinus bradycardia or exit block with symptoms. Symptomatic bradycardia (as defined in the Introduction) with sinus node dysfunction is considered to be an indication for a pacemaker, assuming that another cause of such symptoms has been excluded. Such alternative causes to be considered include seizures, breath holding, infantile apnea, and autonomic dysfunction.

The bradycardia–tachycardia syndrome is frequently an indication for a pacemaker in children,
particularly if an antiarrhythmic drug other than digitalis is necessary. The use of quinidine or other type I drugs is particularly dangerous in children with this syndrome. Propranolol and amiodarone also severely depress sinus node function, and their use may require pacing in children with the bradycardia–tachycardia syndrome.

**Indications for Permanent Pacing in Children**

**Class I**

A. Second- or third-degree AV block with symptomatic bradycardia, as defined
B. Advanced second- or third-degree AV block with moderate to marked exercise intolerance
C. External ophthalmoplegia with bifascicular block
D. Sinus node dysfunction with symptomatic bradycardia, as defined
E. Congenital AV block with wide QRS escape rhythm or with block below the His bundle
F. Advanced second- or third-degree AV block persisting 10–14 days after cardiac surgery

**Class II**

A. Bradycardia–tachycardia syndrome with need for an antiarrhythmic drug other than digitalis or phenytoin
B. Second- or third-degree AV block within the bundle of His in an asymptomatic patient
C. Prolonged subsidiary pacemaker recovery time
D. Transient surgical second- or third-degree AV block that reverts to bifascicular block
E. Asymptomatic second- or third-degree AV block and a ventricular rate <45 beats/min when awake. Complete AV block when awake, with an average ventricular rate <50 beats/min
F. Complete AV block with double or triple rest cycle length pauses or minimal heart rate variability
G. Asymptomatic neonate with congenital complete heart block and bradycardia in relation to age
H. Complex ventricular arrhythmias associated with second- or third-degree AV block or sinus bradycardia
I. Long QT syndrome

**Class III**

A. Asymptomatic postoperative bifascicular block
B. Asymptomatic postoperative bifascicular block with first degree AV block
C. Transient surgical AV block that returns to normal conduction in <1 week
D. Asymptomatic type I second-degree AV block
E. Asymptomatic congenital heart block without profound bradycardia in relation to age

**VIII. Pacing for Tachyarrhythmia**

The decision to use a pacemaker to control tachycardias should be made only after careful observation and electrophysiologic study by those experienced in this complex field.

**Indications for Permanent Pacing for Tachyarrhythmias**

Under certain circumstances, an implanted pacemaker may be useful in the treatment of patients with recurrent symptomatic ventricular and supraventricular tachycardias. Reentrant rhythms may be interrupted by a variety of pacing patterns, including programmed stimulation and short bursts of rapid pacing. These antitachyarrhythmia devices may detect tachycardia and automatically activate a pacing sequence or they may respond only to an external instruction—for example, application of a magnet. In some patients with the long QT syndrome, recurrent ventricular tachycardia may be prevented by continuous pacing. Atrial synchronous ventricular pacing may prevent recurrences of reentrant supraventricular tachycardia. Although ventricular ectopic activity may be suppressed by such pacing in other conditions, serious or symptomatic arrhythmias are rarely prevented.

Potential recipients of antitachyarrhythmia devices that interrupt arrhythmias should undergo extensive testing before implantation to ensure that the devices safely and reliably terminate the ectopic mechanism without accelerating the tachycardia or inducing ventricular fibrillation. These patients usually have been unresponsive to antiarrhythmic drugs or were receiving agents that were inappropriate to control the cardiac arrhythmias. When permanent pacemakers employing programmed extrastimulation or rapid overdrive pacing are used to detect and interrupt supraventricular tachycardia, all pacing should be done in the atrium. Although these pacemakers may be effective, adverse interactions have been reported with use of ventricular pacing to interrupt supraventricular arrhythmias.

**Indications for Permanent Pacemakers That Automatically Detect and Pace to Terminate Tachycardias**

**Class I**

A. Symptomatic recurrent supraventricular tachycardia when drugs fail to control the arrhythmia or produce intolerable side effects
B. Symptomatic recurrent ventricular tachycardia after an automatic defibrillator has been implanted or incorporated in the device and recurrence of ventricular tachycardia is not prevented by drug therapy or when no other therapy is applicable

**Class II**

A. Recurrent supraventricular tachycardia as an alternative to drug therapy

**Class III**

A. Tachycardias that are accelerated or converted to fibrillation by pacing
B. The presence of accessory pathways having the capacity for rapid anterograde conduction whether or not the pathways participate in the mechanism of the tachycardia

**Indications for Externally Manually Activated Antitachyarrhythmia Devices That Act to Terminate Tachycardia**

**Class I**

A. Recurrent, symptomatic ventricular tachycardia uncontrolled by drugs when surgery, catheter ablation, or the implantation of an automatic pacemaker or cardioverter-defibrillator is not indicated

**Class III**

A. Recurrent tachycardia that produces syncope

**Indications for Overdrive or Atrial Synchronous Ventricular Pacemakers Intended to Prevent Tachycardia Occurrence**

**Class I**

A. Atrioventricular reentrant or AV node reentrant supraventricular tachycardia not responsive to medical therapy

**Class II**

A. Sustained ventricular tachycardia in other conditions when all other therapies are ineffective or inapplicable and efficacy of pacing is thoroughly documented

B. Long QT syndrome

**Class III**

A. Frequent or complex ventricular ectopic activity without sustained ventricular tachycardia associated with coronary artery disease, cardiomyopathy, mitral valve prolapse; or a normal heart and in the absence of the long QT syndrome

B. The long QT syndrome due to remediable causes

**IX. Indications for Implantation of Automatic Defibrillator Devices**

Early clinical reports suggest that an automatic implanted cardioverter-defibrillator is effective in preventing sudden cardiac death in patients presumed to be at risk for life-threatening ventricular tachyarrhythmias. Subsequent experience continues to support these impressions. There have been no prospective randomized studies comparing this or other devices to other treatments, but several less rigorous comparisons have been published.

Concern remains about the value of drug therapy for preventing ventricular fibrillation and sudden death. Unfortunately, there is still no complete evidence that drug therapy aimed at ventricular arrhythmias reduces the incidence of sudden death due to coronary heart disease. However, the prognosis of repeated ventricular tachycardia or ventricular tachyarrhythmia resistant to therapy with antiarrhythmic drugs is known to be poor; patients so affected frequently die suddenly. By comparison, survival in patients treated with the implanted cardioverter-defibrillator is quite favorable. The actuarial survival of patients receiving these devices was compared with the projected survival rate if the first clinically appropriate shock had been ineffective. Two such studies showed significant improvements in survival in patients with the defibrillator. However, this study design tends to overestimate mortality because some shocks might have been untriggered or might have interrupted an arrhythmia that would not have been lethal. Another study using concurrent medically treated populations for comparison showed a better outcome in subjects with the implanted cardioverter-defibrillator.

A special case for consideration is the patient with a documented episode of aborted sudden death who does not have an inducible tachyarrhythmia at electrophysiologic study. In patients surviving an episode of sudden cardiac death due to ventricular tachycardia or fibrillation, invasive electrophysiologic testing with programmed stimulation can be used to define therapy if ventricular arrhythmias are inducible. In a significant fraction of patients with aborted sudden death, clinical ventricular arrhythmias are not inducible by accepted laboratory protocols. Thus, it is not possible to estimate the true relative risks. It is clear that despite therapy, sudden death is relatively common in patients with inducible tachyarrhythmias; 14% died suddenly at a mean follow-up interval of 20 months.

Another special case is the patient in whom it is not possible to predict subsequent efficacy of therapy by ambulatory ECG monitoring, electrophysiologic study, or other methods. This situation occurs under three circumstances in which a “fail-safe” therapy such as an implanted cardioverter-defibrillator is reasonable:

1. Tachyarrhythmia is not inducible and spontaneous ventricular ectopic activity is infrequently observed or absent
2. Previous predictions of efficacy were incorrect
3. Efficacy assessment is contraindicated, refused, or infeasible

Before a patient is considered to be a candidate for an implanted defibrillator, the arrhythmia in question must have been demonstrated to be life threatening, producing sudden death, syncope, or severe hemodynamic compromise. Remediable causes of the arrhythmia must have been ruled out, such as acute myocardial infarction, myocardial ischemia, electrolyte imbalance, and drug toxicity. Device selection mandates the participation of an electrophysiologist. Indications for implanted cardioverter-defibrillator implantation remain evolutionary and controversial. Therefore, these guidelines are fairly liberal, pending further definitions.

**Class I**

A. One or more documented episodes of hemodynamically significant ventricular tachycardia or ven-
tricular fibrillation in a patient in whom electrophysiologic testing and ambulatory monitoring cannot be used to accurately predict efficacy of therapy.

B. One or more documented episodes of hemodynamically significant ventricular tachycardia or ventricular fibrillation in a patient in whom no drug was found to be effective or no drug currently available and appropriate was tolerated.

C. Continued inducibility at electrophysiologic study of hemodynamically significant ventricular tachycardia or ventricular fibrillation despite the best available drug therapy or despite surgery or catheter ablation if drug therapy has failed.

Class II

A. One or more documented episodes of hemodynamically significant ventricular tachycardia or ventricular fibrillation in a patient in whom drug efficacy testing is possible.

B. Recurrent syncope of undetermined origin in a patient with hemodynamically significant ventricular tachycardia or ventricular fibrillation induced at electrophysiologic study in whom no effective or no tolerated drug is available or appropriate.

Class III

A. Recurrent syncope of undetermined cause in a patient without inducible tachyarrhythmias.

B. Arrhythmias not due to hemodynamically significant ventricular tachycardia or ventricular fibrillation.

C. Incessant ventricular tachycardia or fibrillation.

X. Clinical Applications of Various Pacing Modes and Device Selection

This section lists the conditions for which various pacing modes might be selected. The acceptability of a given mode of pacing is divided into three classes according to the following definitions:

Class I: Conditions for which there is general agreement that such a mode of pacing is appropriate.

Class II: Conditions for which a given mode of pacing may be used but there is divergence of opinion with respect to the necessity of that mode of pacing.

Class III: Conditions for which there is general agreement that such a mode of pacing is inappropriate.

Two varieties of pulse generators are available for permanent implantation:

1. Single-chamber pacemakers for use in either atrium or ventricle.

2. Dual-chamber pacemakers for use in atrial and ventricular chambers (usually programmable to single-chamber pacemaker modes as well).

Virtually all modern pacemakers are multiprogrammable, which renders them more or less adaptable to changing clinical situations. (Some pacing modes that were originally found as specific pacemaker models, such as ventricular asynchronous pacing mode [VOO], P wave synchronized pacing mode [VAT], and ventricular-triggered pacing mode [VVT], and that still retain some useful, though minor, functions are not discussed.102 These modes are optional settings of some multiprogrammable pacemakers.) Many new pacemakers provide telemetry of stored and variable data that, on command, can provide information about pacemaker function and clinical performance. Both programmability and telemetry are helpful in optimizing pacemaker function, avoiding reoperation, and extending pulse generator life.

Many pacemakers also incorporate alternate sensors that respond to variables (signals) other than P waves to increase the pacing rate.102 Adaptive rate pacemakers (rate modulated, “rate responsive”) utilize various types of sensors that respond to physical, chemical, or electrical signals. They can be classified roughly in the following way:

A. Physical

1. Motion—physical activity

2. Temperature—mixed right atrial venous blood temperature

B. Chemical

1. Venous oxygen saturation

2. pH

C. Electrical phenomena

1. Impedance variations—respiratory rate, minute ventilation, right ventricular stroke volume, rate of change of right ventricular pressures, and stroke volume (dP/dt and dV/dt), and prejection period

2. Intracardiac potentials

a. Stimulus to T wave intervals

b. Evoked potentials (Wilson’s ventricular depolarization gradient)

Single-chamber pacemakers incorporating at least one of these alternate variables are widely used. Alternate sensors also have been added to dual-chamber devices. Pacemakers using two variables in addition to the P wave are also making their appearance.

Adaptive rate pacemakers cost more than their nonadaptive equivalents. They may require special electrodes and leads that are not compatible with preexisting or future models.

Some believe that single-chamber adaptive rate pacemakers are equivalent to standard dual-chamber pacemakers in terms of physiologic effectiveness. Although this may be less important at rapid heart rates, at slow heart rates it is almost always desirable to maintain AV synchrony. Long-term absence of AV synchrony increases the incidence of atrial fibrillation and stroke and may reduce patient life expectancy, particularly in patients with impaired ventricular function, idiopathic hypertrophic subaortic stenosis, or aortic stenosis.103–108 Therefore, the concept that the single-chamber pacemaker with adaptive-rate functions is equivalent to the dual-chamber pacemaker cannot be supported as a general rule.

In choosing between pacemakers with or without adaptive rate functions, the following factors should be considered:

1. The cardiac conduction abnormality

2. The nature and severity of comorbidities

3. The presence of coronary heart disease and angina pectoris

4. The degree of left ventricular dysfunction
5. The impact of present and future drug therapy
6. The level of anticipated activity
7. The availability of support services, such as follow-up clinics, industrial backup, and availability
8. The expertise of implant teams familiar with the device and its programmable function
9. Costs

The primary indication for an adaptive rate pacemaker is to permit a heart rate increase in the absence of an appropriate spontaneous increase in heart rate. This may be the case if the P waves are undetectably small, or absent as in atrial fibrillation, or are unresponsive to stress or exercise. The latter condition has come to be known as chronotropic incompetence. For practical purposes, chronotropic incompetence might be said to exist if the heart rate does not reach 100 beats/min in response to an exercise test.

There are advantages and disadvantages to each; perhaps the most important consideration is whether there is a need for a special lead. Devices that may be used with standard unipolar or bipolar electrodes are those that sense the stimulus to T wave interval, evoked potentials, vibration, ventricular prejection period, and ventricular volume change. Conditions that require special electrodes are temperature, oxygen saturation, dP/dt, and ventricular pressure. The characteristics of the variables to be considered are input and output feedback (closed or open loop), speed of rate response and rate of recovery, appropriateness of the rate response to the activity, the susceptibility of the sensor to extraneous signals (“noise”), the complexity of programming and follow-up, the projected influence on power consumption, and the longevity of the battery.

Many pulse generators incorporate other adaptive functions. These include differing AV intervals after a paced or sensed P wave, shortening of the AV interval and the atrial refractory period with increasing heart rate or metabolic activity, extension of the atrial refractory period after a ventricular premature beat to prevent sensing of retrograde atrial activity, and rate-smoothing adaptation for management of irregular atrial rates. Furthermore, some specialized pacemakers also can be utilized for noninvasive electrophysiologic and antiarrhythmic studies. Clearly, further data are needed to identify the relative benefit of these adaptive functions. The physician must decide how many of these features, if any, are clinically necessary. It is assumed that pacemakers will be implanted by experts who are able to select the device that offers the best clinical advantage at reasonable cost.

Single Chamber Pacemakers

I. Atrial--AAI: Atrial pacing inhibited by sensed atrial activity
   Class I
   A. Symptomatic sinus node dysfunction (sick sinus syndrome), provided AV conduction is shown to be adequate by appropriate studies
   Class II

A. Hemodynamic enhancement through rate adjustment in patients with bradycardia and symptoms of impaired cardiac output, provided AV conduction is shown to be adequate by appropriate tests

Class III
A. Preexisting AV conduction delay or block or if decremental AV conduction is demonstrated by appropriate tests
B. Inadequate intracavitary atrial complexes
   AAIR+: As in classes I and II but with chronotropic incompetence and an anticipated moderate to high level of physical activity, normal AV conduction, and with little likelihood of progression of AV block or induction of AV block as the result of drug therapy.
II. Ventricular--VVI: The classic prototypical pacing mode: ventricular pacing inhibited by sensed spontaneous ventricular activity.

Class I
A. Any symptomatic bradycardia where pacing simplicity is a prime concern in cases of:
   1. No significant atrial hemodynamic contribution (persistent or paroxysmal atrial flutter/fibrillation, giant atria)
   2. No evidence of pacemaker syndrome due to loss of atrial contribution or negative atrial kick (a replacement pacemaker)†

Class II
A. Symptomatic bradycardia where pacing simplicity is a prime concern in cases of:
   1. Senility (for life-sustaining purposes only)
   2. Terminal disease
   3. Domicile remote from a follow-up center
   4. Absent retrograde ventriculoatrial (VA) conduction

Class III
A. Known pacemaker syndrome or symptoms produced by temporary ventricular pacing at the time of initial pacemaker implantation
B. The need for maximum atrial contribution because of
   1. Congestive heart failure
   2. Special need for rate response

VVIR‡: As in classes I and II but with chronotropic incompetence and anticipated moderate to high level of physical activity. VVIR pacemakers are particularly contraindicated in the presence of retrograde conduction.

‡Atrial pacing inhibited by sensed atrial activity. R in position 4 indicates the presence of an adaptive rate function.
†The pacemaker syndrome was first defined as light-headedness or syncope related to long periods of AV asynchrony that occurred at times during ventricular-inhibited (VVI) or VOO pacing. The definition is now expanded to include 1) episodic weakness or syncope associated with alternating AV synchrony and asynchrony; 2) inadequate cardiac output associated with continued absence of AV synchrony or with fixed asynchrony (persistent VA conduction); and 3) patient awareness of beat-to-beat variations in cardiac contractile sequence, often as a result of a) cannon A waves, b) V waves transmitted to the atria or pulmonary veins, and c) bundle branch block patterns of ventricular contraction with a paced beat.
‡Ventricular-VVIR: The classic prototypical pacing mode; ventricular pacing inhibited by sensed spontaneous ventricular activity. R in position 4 indicates the presence of adaptive rate function.
VA conduction or when angina pectoris or congestive failure is aggravated by fast rates.

**Dual-Chamber Pacemakers**

I. *VDD*: Ventricular pacing in synchrony with sensed atrial activity inhibited by sensed ventricular activity. (Although these units are rate modulated at a slow atrial rate below the set rate of the pacemaker, only the ventricle is paced, in which case the pacemaker functions as a VVI unit). With the recent development of “single-pass” leads that incorporate ventricular pacing electrodes at the tip and a set of sensing electrodes in the right atrium on the shaft of the lead, this mode has regained some popularity. This system does *not* provide atrial pacing.

**Class I**

A. Requirements for ventricular pacing when adequate atrial rates and adequate intracavitary atrial complexes are present. This includes the presence of complete AV block when

1. Atrial contribution is needed for hemodynamic benefit
2. Pacemaker syndrome had existed or is anticipated

**Class II**

A. Normal sinus rhythm and normal AV conduction in patients needing ventricular pacing intermittently

**Class III**

A. Frequent or persistent supraventricular tachyarrhythmias, including atrial fibrillation or flutter
B. Inadequate intracavitary atrial complexes
C. Intact VA conduction

II. *DVI*: Pacing of both chambers at a preselected minimal rate with both outputs inhibited by ventricular but not atrial complexes.

**Class I**

A. The need for synchronous atrial–ventricular contraction in symptomatic bradycardia and a slow atrial rate
B. Previously documented pacemaker syndromes

**Class II**

A. Frequent supraventricular arrhythmias in which combined pacing and drugs have been shown to be therapeutically effective
B. Bradycardia–tachycardia syndrome, provided adjustment of atrial rate and AV interval terminates or prevents the emergence of supraventricular arrhythmias with or without concomitant drug administration

**Class III**

A. Frequent or persistent supraventricular tachyarrhythmias, including atrial fibrillation or flutter

*DVI*: An option in some adaptive rate pacemakers. There is no atrial sensing, and therefore atrial synchrony at fast rates does not occur.

III. *DDI*: Dual-chamber pacing, sensing in both chambers, and inhibited by sensed events in both chambers. This mode is like DDD and DVI pacing in that it provides AV synchrony at slow rates. It is unlike DDD pacing in that there is no atrial tracking and unlike DVI pacing in that the atrial output will be inhibited by an atrial or ventricular event, thus avoiding the provocation of an atrial arrhythmia through competitive pacing. Thus, it is useful in patients requiring dual-chamber pacing who have frequent, but not constant, supraventricular arrhythmias.

**DDIR**: A particularly useful mode in chronotropic incompetence when a moderate to high level of activity is anticipated and when there are fairly frequent atrial arrhythmias or in those individuals who need dual-chamber pacing intermittently.

IV. *DDD*: Pacing of both chambers, sensing of both chambers, inhibition of atrial or ventricular output by sensed atrial or ventricular activity; triggering of ventricular output by sensed atrial activity.

**Class I**

A. Requirement for AV synchrony over a wide range of rates such as

1. The active or young patient with atrial rates responsive to clinical need
2. Significant hemodynamic need
3. Pacemaker syndrome during previous pacemaker experience or a reduction in systolic blood pressure >20 mm Hg during ventricular pacing at the time of pacemaker implantation (with or without evidence of VA conduction)

**Class II**

A. Complete heart block or sick sinus syndrome and stable atrial rates
B. When simultaneous control of atrial and ventricular rates can be shown to inhibit tachyarrhythmias or when the pacemaker can be adjusted to a mode designed to interrupt the arrhythmia

**Class III**

A. Frequent or persistent supraventricular tachyarrhythmias, including atrial fibrillation or flutter
B. Inadequate intracavitary atrial complexes
C. Angina pectoris aggravated by rapid heart rates

**DDD**: This mode is indicated in patients with chronotropic incompetence who have an anticipated moderate or high level of activity and in whom there is a stable atrial rhythm. It is particularly applicable in those patients who have persistent VA conduction.

**Comment**

Pacemakers have grown immensely complex, rendering interpretation of the paced electrocardiogram difficult for even the most experienced cardiologist. The subtleties of indications, device selection, and, especially, follow-up require constant study and hands-on usage. Thus, pacing should be performed by individuals with appropriate training who maintain their skills by participating in an adequate number of operations.
The implanting physician must also assure that patients receive regular follow-up care in a facility in which special attention is paid to the adequacy of pacemaker function and to its optimal physiologic effectiveness. Simple ECG monitoring or transtelephonic transmission of signals from complex pacemakers at the present state of the art in themselves are not satisfactory surveillance methods. Some implanters use sales and other industrial representatives to assist them with their implantations. The sales representatives thus act as hospital technicians and may not be adequately protected by liability insurance. It is a practice that should not be condoned.

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_Circulation_. 1991;84:455-467
doi: 10.1161/01.CIR.84.1.455

_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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