Guidelines for Permanent Cardiac Pacemaker Implantation, May 1984

A report of the Joint American College of Cardiology/American Heart Association Task Force on Assessment of Cardiovascular Procedures (Subcommittee on Pacemaker Implantation).

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Background

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 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 Joint Task Force on Assessment of Cardiovascular Procedures with the following charge:

The Joint 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 and contraindications and cost-effectiveness of such specific procedures.

The Task Force shall include a Chairman and four members, two representatives from the American Heart Association and two 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 Joint Task Force are: Roman W. DeSanctis, MD, Harold T. Dodge, MD, T. Joseph Reeves, MD, Sylvan L. Weinberg, MD and Charles Fisch, MD, Chairman.

The Subcommittee on Pacemaker Implantation 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.

This document was reviewed by the officers and other responsible individuals of the two organizations and received final approval on May 2, 1984. It is being published simultaneously in Circulation and 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.

I. Introduction

The joint American College of Cardiology/American Heart Association Ad Hoc Task Force on Assessment of Cardio-
vascular Procedures was formed to make recommendations regarding the appropriate utilization of technology in the diagnosis and treatment of patients with cardiovascular disease. One such important technique is that of cardiac pacing. Rapid progress in a number of areas has led to extraordinary and still evolving advances in implantable cardiac pacemakers and in other devices which electrically stimulate the heart. For this reason, and also because of allegations of abuses of this technology, by the medical profession, the Task Force was assigned the task of defining current indications for permanent cardiac pacemakers. These recommendations are the subject of this report. Because of the multitude, complexity and initial cost of currently available pacing systems, the Subcommittee has included recommendations regarding selection of devices for specific clinical problems in which pacing is indicated. The Subcommittee 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 pacemakers. Because of continuing research and development, some of these recommendations may be subject to modification in even the near future.

These recommendations apply to permanent pacing in the management of chronic, though sometimes intermittent, disorders of cardiac rhythm. For the most part, they do not pertain to identifiable factors which 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 must be reached by scrupulous adherence to a fundamental principle of clinical medicine which demands a careful, thoughtful analysis of each individual patient by the responsible physician. 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 decision with respect to pacing can be made.

The Subcommittee has not offered any recommendations regarding resources required to perform pacemaker 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 (1). The Subcommittee unanimously urges careful review of the resource guidelines by all institutional administrators, physicians and surgeons who are responsible for pacemaker therapy. The clinical symptomatology associated with bradycardia needs definition at the outset since it recurs throughout the report as a major indication for permanent pacemaker therapy. In this report, the term "symptomatic bradycardia" is used to refer to the following clinical manifestations which 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.

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

Class I: Conditions for which there is general agreement that permanent pacemakers should be implanted.

Class II: Conditions for which permanent pacemakers 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 are unnecessary.

In those patients being considered for pacemakers, 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 prognosis for life;
2) presence of associated underlying cardiac disease that may be adversely affected 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; and
8) desires of the patient and family.

The format of this report consists of a brief definition and description of specific clinical situations in which pacing may be considered, and literature references to document the basis for the recommendations.

II. Pacing in Acquired Atrioventricular (AV) Block in Adults

Clinically, atrioventricular (AV) block is classified as first degree, second degree or third degree (complete) heart block; anatomically, it is defined as supra-His, intra-His and/or 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). "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 and/or ventricular arrhythmias. 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 documented that patients with complete heart block and syncope have an improved survival with permanent pacing (2–5). 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 nodal delay, tends to be benign (6–8). 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 (6,8,9).

Recommendations for insertion of permanent pacemakers in patients with AV block with acute myocardial infarction or congenital AV block are discussed in a separate section. AV 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 AV 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 patients with these symptoms in the presence of complete heart block, the symptoms must be presumed to be due to the heart block unless proven to be otherwise.
2. Congestive heart failure.
3. Ventricular ectopy and other conditions that require treatment with drugs which suppress the automaticity of escape foci.
4. Documented periods of asystole of 3.0 seconds or longer, or any escape rate of less than 40 beats/min in symptom-free patients.
5. Confusional states which clear with temporary pacing.

B. Second degree AV block, permanent or intermittent, regardless of the type or the site of the 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 I-A. 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 on bi-trifascicular block).

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

**III. Pacing in Atrioventricular (AV) 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 per se (10–14). 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 increased incidence of sudden death (10–12). 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 (12). 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. Patients with persistent advanced second degree AV block or complete heart block after acute myocardial infarction (12,14). Decision for insertion of pacemaker should be made before discharge in this group of patients.

**Class II.**

A. Patients with persistent first degree AV block in the presence of bundle branch block not documented previously (13).

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

**Class III.**

A. Patients in whom AV conduction disturbances are transient in the absence of intraventricular conduction defects (12).
B. Patients with transient AV block in the presence of isolated left anterior hemiblock (11).
C. Patients with acquired left anterior hemiblock in the absence of atrioventricular (AV) block.

IV. Pacing in Bifascicular and Trifascicular Block (Chronic)

Bifascicular and trifascicular block refer to electrocardiographic 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 electrocardiographic abnormalities, there is convincing evidence that advanced heart block with symptoms due to the block is associated with a high mortality and a significant incidence of sudden death (16-18).

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Syncope is common in patients with bifascicular block. It is usually not recurrent, nor is it associated with an increased incidence of sudden death (16-18). It has been suggested that although pacing relieves the transient neurologic symptoms, it does not reduce mortality from sudden death (19). There is convincing evidence, however, that in the presence of complete heart block, either permanent or transient, syncope is associated with an increased incidence of sudden death (5). 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, prophylactic permanent pacing is indicated (20,21).

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 (22).

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 prolongation is common in patients with bifascicular block. However, the prolongation is most often at the level of the AV node. Furthermore, there is no correlation between the PR and HV intervals, nor is there a correlation between the length of the PR interval and progression to complete heart block and incidence of sudden death (23,24,28). Although most patients with chronic or intermittent complete heart block demonstrate prolongation of the HV interval during anterograde conduction, and some investigations (26,27) have suggested that asymptomatic patients with bifascicular block and a prolonged HV interval be considered for permanent pacing, the evidence indicates that while 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 mortality; death is not sudden and is due to the underlying heart disease, and not to complete heart block (16,19, 23,28,29). The prolonged HV interval is, thus, not an independent marker for sudden death (22).

Atrial pacing as a means of identifying patients at increased risk of future complete heart block probably is not justified. The chance of induction of distal heart block with pacing is low (16,27,30,31). In fact, pacing often fails to induce distal His block in patients with documented abnormal conduction of the His-Purkinje system (16,26,27,32,33). 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, this may be considered an indication for pacing by some (34).

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 block with intermittent type II second degree AV block with symptoms attributable to the heart block.

Class II.
A. Bifascicular or trifascicular block with intermittent type II second degree AV block without symptoms.
B. Bifascicular or trifascicular block with syncope that is not proven to be due to complete heart block, but other possible causes for syncope are not identifiable.
C. Pacing-induced infra-His block.

Class III.
A. Fascicular blocks without AV block or symptoms.
B. Fascicular blocks 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 bradycardia, 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. This may be difficult, however, because of the intermittent nature of the episodes. Sinus bradycardia is accepted as a physiologic finding in trained athletes, in whom awake resting heart rates of 40 to 50 beats/min are not uncommon and minimal heart rates during sleep may
be as slow as 30 to 43 beats/min with sinus pauses as long as 1.6 to 2.8 seconds (35–37). This is due to increased vagal tone. Permanent pacing in patients with sinus node dysfunction may not necessarily result in an improvement in survival (38,39), but severe symptoms related to bradycardia may be relieved (40,41).

**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 is no acceptable alternative.

**Class II.**
A. Sinus node dysfunction, occurring spontaneously or as a result of necessary drug therapy, with heart rates below 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 Syndrome**

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 to the reflex:

1) **Cardioinhibitory,** resulting from increased parasympathetic tone and manifested by slowing of the sinus rate and/or prolongation of the PR interval and advanced AV block; and
2) ** Vasodepressor,** secondary to a reduction in sympathetic activity resulting in hypotension.

Before concluding that permanent pacing is clinically indicated, determination of the relative contribution of the two components of carotid sinus stimulation to the individual patient’s symptom complex is essential. Hyperactive response to carotid sinus stimulation is defined as asystole due either to sinus arrest or AV block of more than 3 seconds and/or a substantial symptomatic decrease in systolic blood pressure. However, such heart rate and hemodynamic responses may occur in normal subjects and patients with coronary artery disease (42,43), and a conclusion of a cause and effect relation between the hypersensitive carotid sinus and the patient’s symptoms must be made with great caution. Minimal pressure on the carotid sinus in the elderly 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 (44–46). Since 10 to 20% of patients with this syndrome may have an important vasodepressor component, it is necessary to define this before concluding that all symptoms are related to asystole alone. In patients with both cardioinhibitory and vasodepressor components, attention to the latter in patients undergoing permanent pacing is essential for effective therapy.

**Indications for Permanent Pacing in Hypertensive Carotid Sinus Syndrome**

**Class I.**
A. Patients with recurrent syncope associated with clear, spontaneous events provoked by carotid sinus stimulation, in whom minimal carotid sinus pressure induces asystole of greater than 3 seconds in the absence of any medication that depresses the sinus node or AV conduction.

**Class II.**
A. Patients with recurrent syncope without clear, provocative events and with a hypersensitive cardioinhibitory response.

**Class III.**
A. Asymptomatic patients with a hyperactive cardioinhibitory response to carotid sinus stimulation.
B. Patients with vague symptoms, such as dizziness and/or light-headedness, and with hyperactive cardioinhibitory response to carotid sinus stimulation.
C. Patients with recurrent syncope, light-headedness or dizziness in whom the vasodepressor response is the cause for symptoms.

**VII. The Use of Pacemakers in Children**

Although the indications for pacemakers in children are similar to those in adults, there are some special considerations. The optimal indication for a pacemaker implantation in a child, as in an adult, is the concurrent observation of symptoms with bradycardia. For example, a patient with syncope who is observed electrophysiologically to have complete AV block or a patient with syncope who is noted on physical examination to have severe bradycardia such as a heart rate of 30 beats/min. Concurrence of symptoms and bradycardia can also be obtained by 24 hour ambulatory electrocardiography or by transtelephonic electrocardiography. Sometimes several 24 hour recordings are necessary.
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 tachycardia. In patients with sinus node dysfunction, even greater emphasis is placed on concurrence of sinus bradycardia or exit block with symptoms. Sinus node dysfunction is not likely to be a fatal arrhythmia in infants or children. Therefore, more time can be spent trying to document the presence of an arrhythmia during symptoms.

Symptomatic bradycardia (as defined in the Introduction) with sinus node dysfunction is considered to be an indication for a pacemaker, assuming that another etiology to account for such symptoms has been excluded. Such alternate etiologies to be considered include seizures resulting in hypoxia, breathholding or infantile apnea.

It is sometimes hard to differentiate whether apnea or bradycardia occurs first in symptomatic patients. The bradycardia-tachycardia syndrome is frequently an indication for pacemakers in children, particularly if an antiarrhythmic drug other than digitalis is necessary. It appears that the use of quinidine or other type I drugs is particularly dangerous in children with bradycardia-tachycardia syndrome. Propranolol and amiodarone also severely depress sinus node function and their use may require the use of a pacemaker 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 (47).
D. Sinus node dysfunction with symptomatic bradycardia as defined.
E. Bradycardia-tachycardia syndrome in a child with a need for antiarrhythmic drugs other than digitalis.
F. Congenital AV block with wide QRS escape rhythm (48).
G. Asymptomatic patients after cardiac surgery with advanced second or third degree AV block persisting 10 to 14 days postoperatively (49).

Class II.
A. Second or third degree AV block within the bundle of His in an asymptomatic patient (49).
B. Prolonged subsidiary pacemaker recovery time (50).
C. Transient surgical second or third degree AV block, which reverts to bifascicular block.
D. Asymptomatic children with second or third degree AV block and a ventricular rate of less than 45 beats/minute when awake (51).
E. Asymptomatic infra-His, second or third degree AV block (49).

F. An asymptomatic neonate with congenital complete heart block with bradycardia in relation to age (52).
G. Complex ventricular arrhythmias associated with second or third degree AV block or sinus bradycardia (53).

Class III.
A. Postoperative bifascicular block in the asymptomatic patient.
B. Postoperative bifascicular block with first degree AV block in the asymptomatic patient.
C. Transient surgical AV block that returns to normal conduction in less than 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 Tachyarrhythmias

The use of implantable cardiac pacemakers to terminate supraventricular or ventricular tachycardias is just beginning. We will not discuss the use of overdrive pacemakers for the termination of ventricular tachycardia, since there is no clinically approved device for this indication and since the use of this device is still extremely controversial with risks perhaps outweighing benefits in some patients. The decision for chronic use of 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

Class I.
A. Patients with symptomatic supraventricular tachycardia which has not responded to a well planned medical regimen including documentation of adequate serum drug concentrations, or in whom the medical treatment causes major side effects or in whom the necessity for taking drugs seriously inhibits the patient's ability to carry out normal daily function. Before implantation of an antitachycardia pacemaker, an electrophysiologic study should be carried out and the various proposed modes of termination of tachycardia tested to determine which one is most appropriate for the particular patient. An external form of the implantable device should be available during electrophysiologic study to document the exact settings that will be used and will in fact terminate the patient's tachycardia. The physician who implants the pacemaker should be prepared to reprogram the pacemaker to new settings when the patient is again active.
IX. Clinical Applications of Various Pacing Modes

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 (SCP) for use in either atrium or ventricle; and
2) dual chamber pacemakers (DCP) for use in both chambers (usually programmable to SCP 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 VOO, VAT and VVT) are not discussed. These modes are now optional settings of multiprogrammable pacemakers. Many new pacemakers also 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.

It is essential that the selection process be individualized to the needs of the patient, with appropriate consideration given to complication, complexity and 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 tests.

Class II.

A. Overdrive of supraventricular or ventricular arrhythmias.

B. Hemodynamic enhancement through rate adjustment in patients with bradycardia and symptoms of impaired cardiac output.

Class III.

A. Pre-existing AV conduction delay or block or if PR interval is inappropriately prolonged by atrial pacing.

B. Inadequate intracavitary atrial complexes.

II. Ventricular (VVI): The classic prototypical pacing mode; ventricular pacing inhibited by sensed spontaneous ventricular activity.

Class I.

A. Any symptomatic bradyarrhythmia, but particularly when there is:
   1) no significant atrial hemodynamic contribution (atrial flutter/fibrillation, giant atria), or
   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 (life-sustaining only),
   2) terminal disease,
   3) domicile remote from a follow-up center, or
   4) intact retrograde VA (ventriculoatrial) conduction.

Class III.

A. Known pacemaker syndrome (a replacement pacemaker) or symptoms produced by temporary ventricular pacing at the time of initial pacemaker implantation.

B. The need for maximal atrial contribution, because of:
   1. congestive heart failure, or
   2. special need for rate responsiveness.

Dual Chamber Pacemakers

I. VDD: Ventricular pacing in synchrony with sensed atrial activity, inhibited by sensed ventricular activity. (Although these units are rate-responsive, 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.)

†The pacemaker syndrome was first defined as the light-headedness or syncope related to long cycles of AV asynchrony that occurred at times during 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.

*The pacemaker mode is identified according to the Inter-Society Commission for Heart Disease Resources (lCHD) code (1).
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 in patients:
1) requiring atrial contribution for hemodynamic benefit, or
2) with previous or anticipated pacemaker syndrome.

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

Class III.
A. Frequent or persistent supraventricular tachyarhythmias, including atrial fibrillation or flutter.
B. Inadequate intracavitary atrial complexes.

III. DVI: Pacing of both chambers at a preselected minimal rate, inhibited by ventricular but not atrial activity.

Class I.
A. The need for synchronous atrial-ventricular contraction in patients with symptomatic bradycardia and a slow atrial rate.
B. Patients with previously documented pacemaker syndrome.

Class II.
A. Overdrive of certain arrhythmias.
B. Frequent supraventricular arrhythmias in which combined pacing and drugs have been shown to be therapeutically effective.
C. 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 tachyarhythmias, including atrial fibrillation or flutter.

III. 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, and
3. pacemaker syndrome during previous pacemaker experience, or a reduction in systolic blood pressure of more than 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. Any patient in whom simultaneous control of atrial and ventricular rates inhibits tachyarrhythmias or in whom the pacemaker can be adjusted to a mode designed to interrupt the arrhythmia.

Class III.
A. Frequent or persistent supraventricular tachyarhythmias, including atrial fibrillation or flutter.
B. Inadequate intracavitary atrial complexes.

References


