ACC/AHA PRACTICE GUIDELINES

ACC/AHA Guidelines for Implantation of Cardiac Pacemakers and Antiarrhythmia Devices

A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Pacemaker Implantation)

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Published by Elsevier Science Inc.
0735-1097/98/$19.00
PII S0735-1097(98)00024-2

Preamble

It is important that the medical profession play a significant role in critically evaluating the use of diagnostic procedures and therapies in the management or prevention of disease states. Rigorous and expert analysis of the available data documenting relative benefits and risks of those procedures and therapies can produce helpful guidelines that improve the effectiveness of care, optimize patient outcomes, and impact the overall cost of care favorably by focusing resources on the most effective strategies.

The American College of Cardiology (ACC) and the American Heart Association (AHA) have jointly engaged in the production of such guidelines in the area of cardiovascular disease since 1980. This effort is directed by the ACC/AHA Task Force on Practice Guidelines. Its charge is to develop and revise practice guidelines for important cardiovascular diseases and procedures. Experts in the subject under consideration are selected from both organizations to examine subject-specific data and write guidelines. The process includes additional representatives from other medical practitioner and specialty groups where appropriate. Writing groups are specifically charged to perform a formal literature review, weigh the strength of evidence for or against a particular treatment or procedure, and include estimates of expected health outcomes where data exist. Patient-specific modifiers, comorbidities, and issues of patient preference that might influence the choice of particular tests or therapies are considered as well as frequency of follow-up and cost-effectiveness.

These practice guidelines are intended to assist physicians in clinical decision-making by describing a range of generally acceptable approaches for the diagnosis, management, or prevention of specific diseases or conditions. The guidelines attempt to define practices that meet the needs of most patients in most circumstances. The ultimate judgment regarding care of a particular patient must be made by the physician and patient in light of all of the circumstances presented by that patient.

The Committee on Pacemaker Implantation was chaired by Gabriel Gregoratos, MD, FACC, and included the following members: Melvin D. Cheitlin, MD, FACC; Alicia Conill, MD, FACP; Andrew E. Epstein, MD, FACC; Christopher Fellows, MD, FACC; T. Bruce Ferguson, Jr., MD, FACC; Roger A. Freedman, MD, FACC; Mark A. Hlatky, MD, FACC; Gerald V. Naccarelli, MD, FACC; Sanjeev Saksena, MD, MBBS, FACC; Robert C. Schlant, MD, FACC; and Michael J. Silka, MD, FACC. In October 1997, this document was approved for publication in the *Journal of the American College of Cardiology* and the executive summary for publication in *Circulation*.

The executive summary and recommendations are published in the April 7, 1998 issue of *Circulation*. The full text is published in the April 1998 issue of the *Journal of the American College of Cardiology*. Reprints of both the full text and the executive summary and recommendations are available from both organizations.

James L. Ritchie, MD, FACC
Chair, ACC/AHA Task Force on Practice Guidelines

Introduction

This second revision of the "ACC/AHA Guidelines for Implantation of Cardiac Pacemakers and Antiarrhythmia Devices" updates the previous versions published in 1984 and 1991. Revision of the statement was deemed necessary for two reasons: the publication of major studies that have advanced our knowledge of the natural history of bradyarrhythmias and tachyarrhythmias, which may optimally be treated with device therapy, and major advances in the technology of such devices.

The committee to revise the ACC/AHA Guidelines for Implantation of Cardiac Pacemakers and Antiarrhythmia Devices was composed of both university-affiliated and practicing physicians. It included experts in the area of device therapy and follow-up, senior clinicians skilled in cardiovascular care, a general internist, and a cardiothoracic surgeon. The committee included representatives of the American College of Physicians, the North American Society of Pacing and Electrophysiology (NASPE), and the Society of Thoracic Surgeons. This document was reviewed by three outside reviewers nominated by the ACC, three outside reviewers nominated by the AHA, and individuals representing the American College of Physicians and the North American Society for Pacing and Electrophysiology. The section "Pacing in Children and Adolescents" was reviewed by additional reviewers with special expertise in pediatric electrophysiology. The committee thanks all the reviewers for their comments. Many of their suggestions were incorporated into the final document.

The ACC/AHA Guidelines for Implantation of Cardiac Pacemakers and Antiarrhythmia Devices were approved for publication by the governing bodies of the ACC and the AHA. These guidelines will be reviewed 2 years after publication and yearly thereafter and considered current unless the Task Force on Practice Guidelines revises or withdraws them from circulation.

The recommendations listed in this document are, whenever possible, evidence based. Pertinent medical literature in the English language was identified through a search of library databases, and a large number of publications were reviewed by committee members during the course of their discussions. Additionally the committee reviewed documents related to the subject matter previously published by the ACC, the AHA, and the North American Society for Pacing and Electrophysiology. References selected and published in this document are representative and not all-inclusive.

The committee reviewed and ranked evidence supporting current recommendations with the weight of evidence ranked as level A if the data were derived from multiple randomized clinical trials involving a large number of individuals. The committee ranked available evidence as level B when data were derived from a limited number of trials involving a comparatively small number of patients or from well-designed data analyses of nonrandomized studies or observational data registries. Evidence was ranked as level C when the consensus opinion of experts was the primary source of recommendation. In the narrative portions of these guidelines, evidence is generally presented in chronological order of development. Studies are identified as *observational, randomized, prospective,* or *retrospective*. The committee emphasizes that for certain conditions for which no other therapy is available, the indications for device therapy are based on expert consensus and years of clinical experience and are thus well supported, even though the evidence was ranked as level C. An analogous example is the use of penicillin in pneumococcal pneumonia where there are no randomized trials and only clinical experience. When indications at level C are supported by historical clinical data, appropriate references (case reports, clinical reviews, etc.) are cited if available. When level C indications are based strictly on committee consensus, no references are cited. In areas where sparse data were available (eg, pacing in children and adolescents), a survey of current practices of major centers in North America was conducted to determine if there was a consensus regarding specific pacing indications.

The final recommendations for indications for device therapy are expressed in the standard ACC/AHA format as follows:

Class I: Conditions for which there is evidence and/or general agreement that a given procedure or treatment is beneficial, useful, and effective.

Class II: Conditions for which there is conflicting evidence and/or a divergence of opinion about the usefulness/efficacy of a procedure or treatment.

Class IIa: Weight of evidence/opinion is in favor of usefulness/efficacy.

Class IIb: Usefulness/efficacy is less well established by evidence/opinion.

Class III: Conditions for which there is evidence and/or general agreement that a procedure/treatment is not useful/effective and in some cases may be harmful.

The focus of these guidelines is the appropriate use of devices (pacemakers and implantable cardioverter-defibrillators [ICDs]), not the treatment of cardiac arrhythmias. The fact that use of a device for treatment of a particular condition is listed as a Class I indication (beneficial, useful, and effective) does not preclude the use of other therapeutic modalities that may be equally effective. As with all clinical practice guidelines, the recommendations in this document focus on treatment of an average patient with a specific disorder and may be modified by patient comorbidities, limitation of life expectancy due to coexisting diseases, and other situations that only the primary treating physician may evaluate appropriately.

These guidelines include expanded sections on selection of pacemakers and ICDs, optimization of technology, cost, and follow-up of implanted devices. The follow-up sections are relatively brief because in many instances the type and frequency of follow-up examinations are device specific. The importance of adequate follow-up, however, cannot be overemphasized because optimal results from an implanted device can be obtained only if the device is adjusted to changing clinical conditions.

The committee considered including a section on extraction of failed/unused leads, a topic of current interest, but elected not to do so in the absence of convincing evidence to support specific criteria for timing and methods of lead extraction. An upcoming policy statement on lead extraction from the North American Society of Pacing and Electrophysiology should provide information on this topic. Similarly, the issue of when to discontinue long-term cardiac pacing has not been studied sufficiently to allow formulation of appropriate guidelines despite the publication of isolated case reports (1). The committee therefore decided to defer inclusion of this topic until additional information is available.

The text accompanying the listed indications should be read carefully because it includes the rationale and supporting evidence for many of the indications, and in several instances it includes a discussion of alternative acceptable therapies. Many of the indications are modified by the term "potentially reversible." This term is used to indicate abnormal pathophysiology (eg, complete heart block) that may be the result of reversible factors. Examples include complete heart block due to drug toxicity (digitalis), electrolyte abnormalities, diseases with inflammatory peri-atrioventricular node reaction (Lyme disease), transient injury to the conduction system at the time of open heart surgery, and others. When faced with a potentially reversible situation, the treating physician must decide how long a waiting period is justified before beginning device therapy. The committee recognizes that this statement does not address issues of length of hospital stay *vis a vis* managed-care regulations. It is emphasized that these guidelines are not intended to address this issue, which falls strictly within the purview of the treating physician.

The term "symptomatic bradycardia" is used frequently throughout this document. Symptomatic bradycardia is defined as a documented bradyarrhythmia that is directly responsible for development of the clinical manifestations frank syncope or near-syncope, transient dizziness or light-headedness, and confusional states resulting from cerebral hypoperfusion attributable to slow heart rate. Fatigue, exercise intolerance, and frank congestive failure may also result from bradycardia. These symptoms may occur at rest and/or with exertion. Definite correlation of symptoms with a bradyarrhythmia is required to fulfill the criteria defining symptomatic bradycardia. Caution should be exercised not to confuse physiological sinus bradycardia (as occurs in highly trained athletes) with pathological bradyarrhythmias.

In these guidelines the terms "persistent," "transient," and "not expected to resolve" are frequently used. These terms are not specifically defined because the time element varies in different clinical conditions. The treating physician must use appropriate clinical judgment and available data in deciding when a condition is persistent or when it can be expected to be transient. Section I.C., "Pacing for Atrioventricular Block Associated With Acute Myocardial Infarction," overlaps with the "ACC/AHA Guidelines for the Management of Patients With Acute Myocardial Infarction" (2) and includes expanded indications and stylistic changes. The statement "incidental finding at electrophysiological study" is used several times in this document and does not mean that such a study is indicated. Appropriate indications for electrophysiological studies have been published (3).

The section on indications for ICDs has been extensively revised and enlarged to reflect the numerous new developments in this field and the voluminous literature related to the efficacy of these devices in the treatment of sudden cardiac death and malignant ventricular arrhythmias. Indications for ICDs are continuously changing and can be expected to change further as ongoing large-scale trials are reported. Thus, the ICD indications may require revision in the next 2 to 3 years. In this document the term "mortality" is used to indicate "all-cause" mortality unless otherwise specified. The committee elected to use "all-cause mortality" because of the variable definition of "sudden death" and the developing consensus to use all-cause mortality as the most appropriate end point of clinical trials (4,5).

These guidelines are not designed to specify training or credentials required for physicians to use device therapy. Nevertheless, in view of the complexity of both cognitive and technical aspects of device therapy, only appropriately trained physicians should use device therapy. Appropriate training guidelines for physicians have been previously published (6,7).

Finally, because many of the recommended indications are controversial, the committee urges the sponsoring organizations to endorse prospective research to address these issues.

I. Indications for Permanent Pacing

A. Pacing for Acquired Atrioventricular Block in Adults

Atrioventricular (AV) block is classified as first-, second-, or third-degree (complete) block; anatomically it is defined as supra-, intra-, or infra-His. First-degree block is defined as abnormal prolongation of the PR interval. Second-degree AV block is subclassified as type I (progressive prolongation of PR interval before a blocked beat) usually associated with a narrow QRS complex or type II (no progressive prolongation of PR interval before a blocked beat) usually associated with a wide QRS complex. *Advanced AV block* refers to the block of two or more consecutive P waves. Third-degree AV block (complete heart block) is defined as absence of AV conduction.

Patients with abnormalities of AV conduction may be asymptomatic or may experience serious symptoms related to bradycardia, ventricular arrhythmias, or both. Decisions regarding the need for a pacemaker are importantly influenced by the presence or absence of symptoms directly attributable to bradycardia. Furthermore, many of the indications for pacing have evolved over 30 years based on experience without the benefit of comparative, randomized clinical trials, in part because no alternative options exist to treat most bradycardias.

Nonrandomized studies strongly suggest that permanent pacing does improve survival in patients with third-degree AV block, especially if syncope has occurred (8–13). Although there is little evidence to suggest that pacemakers improve survival in patients with isolated first-degree AV block (14), it is now recognized that marked first-degree AV block can lead to symptoms even in the absence of higher degrees of AV block (15). Such marked first-degree AV block may follow catheter ablation of the fast pathway with resultant slow pathway conduction. Marked first-degree AV block for any reason may also be associated with a pseudopacemaker syndrome (16) secondary to close proximity of atrial systole to the preceding ventricular systole that produces hemodynamic consequences similar to those associated with retrograde (ventriculoatrial) conduction. In this instance, atrial contraction occurs before complete atrial filling, ventricular filling is compromised, and an increase in pulmonary capillary wedge pressure and a decrease in cardiac output follow. Small, uncontrolled trials have suggested some symptomatic and functional improvement by pacing of patients with PR intervals >0.30 second by decreasing the time for AV conduction (15). Finally, a long PR interval may identify a group of patients with left ventricular (LV) dysfunction, some of whom may benefit from dual-chamber pacing with a short(er) AV delay (17). Consideration should be given to demonstrating hemodynamic improvement by echocardiographic or invasive assessment before implantation of a permanent pacemaker.

Progression to advanced AV block in patients with type I second-degree AV block, when due to delay in the AV node, is unlikely (18–20), and pacing is usually not indicated. Nevertheless, controversy exists, and pacemaker implantation has been advocated for this finding (21–23). On the other hand, in patients with type II second-degree AV block (either intra- or infra-His), symptoms are frequent, prognosis is compromised, and progression to third-degree AV block is common (18,20,24).

Recommendations for permanent pacemaker implantation in patients with AV block in acute myocardial infarction (AMI), congenital AV block, and AV block associated with enhanced vagal tone are discussed in separate sections. Neurally mediated mechanisms in young patients with AV block should be assessed before proceeding with permanent pacing. Physiological AV block in the presence of supraventricular tachyarrhythmias does not constitute an indication for pacemaker implantation except as specifically defined in the recommendations that follow. In general, the decision regarding implantation of a pacemaker must be considered with respect to whether or not it will be permanent. Reversible causes of AV block such as electrolyte abnormalities should be corrected first. Some diseases may follow a natural history to resolution (eg, Lyme disease), and some AV block can be expected to reverse (eg, perioperative AV block due to hypothermia or inflammation near the AV conduction system after surgery for arrhythmias in this region). Conversely, some conditions may warrant pacemaker implantation due to anticipated adverse consequences or disease progression (eg, sarcoid, amyloid) even if the AV block reverses transiently. Finally, permanent pacing for AV block after valve surgery follows a variable natural history, and therefore the decision for permanent pacing is at the physician's discretion.

Indications for Permanent Pacing in Acquired Atrioventricular Block in Adults

Class I

1. **Third-degree AV block at any anatomic level, associated with any one of the following conditions:**
   a. **Bradycardia with symptoms presumed to be due to AV block.** ***(Level of evidence: C)***
   b. **Arrhythmias and other medical conditions that require drugs that result in symptomatic bradycardia.** ***(Level of evidence: C)***

c. **Documented periods of asystole ≥3.0 seconds (25) or any escape rate <40 beats per minute (bpm) in awake, symptom-free patients (26,27).** ***(Level of evidence: B, C)***
d. **After catheter ablation of the AV junction.** ***(Level of evidence: B, C)*** **There are no trials to assess outcome without pacing, and pacing is virtually always planned in this situation unless the operative procedure is AV junction modification (28,29).**
e. **Postoperative AV block that is not expected to resolve.** ***(Level of evidence: C)*** **(30, 30a)**
f. **Neuromuscular diseases with AV block such as myotonic muscular dystrophy, Kearns-Sayre syndrome, Erb's dystrophy (limb-girdle), and peroneal muscular atrophy.** ***(Level of evidence: B)*** **(31–37)**

2. **Second-degree AV block regardless of type or site of block, with associated symptomatic bradycardia.** ***(Level of evidence: B)*** **(19)**

Class IIa

1. **Asymptomatic third-degree AV block at any anatomic site with average awake ventricular rates of 40 bpm or faster.** ***(Level of evidence: B, C)***
2. **Asymptomatic type II second-degree AV block.** ***(Level of evidence: B)*** **(21,23)**
3. **Asymptomatic type I second-degree AV block at intra- or infra-His levels found incidentally at electrophysiological study performed for other indications.** ***(Level of evidence: B)*** **(19,21–23)**
4. **First-degree AV block with symptoms suggestive of pacemaker syndrome and documented alleviation of symptoms with temporary AV pacing.** ***(Level of evidence: B)*** **(15,16)**

Class IIb

1. **Marked first-degree AV block (>0.30 second) in patients with LV dysfunction and symptoms of congestive heart failure in whom a shorter AV interval results in hemodynamic improvement, presumably by decreasing left atrial filling pressure.** ***(Level of evidence: C)*** **(17)**

Class III

1. **Asymptomatic first-degree AV block.** ***(Level of evidence: B)*** **(14) (See also "Pacing for Chronic Bifascicular and Trifascicular Block.")**
2. **Asymptomatic type I second-degree AV block at the supra-His (AV node) level or not known to be intra- or infra-Hisian.** ***(Level of evidence: B, C)*** **(19)**
3. **AV block expected to resolve and unlikely to recur (38) (eg, drug toxicity, Lyme disease).** ***(Level of evidence: B)***

B. Pacing for Chronic Bifascicular and Trifascicular Block

Bifascicular and trifascicular block refer to electrocardiographic evidence of impaired conduction below the AV node in two or three fascicles of the right and left bundles. In patients with such electrocardiographic abnormalities, there is convincing evidence that symptomatic, advanced AV block is associated with a high mortality rate and a significant incidence of sudden death (9,39).

Syncope is common in patients with bifascicular block. Usually it is not recurrent or associated with an increased incidence of sudden death (40–52). It has been suggested that pacing relieves the transient neurological symptoms but does not reduce the frequency of sudden death (46). Electrophysiological study may be helpful to evaluate and direct the treatment of inducible ventricular arrhythmias (53,54) that are common in patients with bifascicular and trifascicular block. However, there is also convincing evidence that in the presence of permanent or transient third-degree AV block, syncope is associated with an increased incidence of sudden death regardless of the results of electrophysiological study (9,54,55). Thus, if the cause of syncope in the presence of bifascicular or trifascicular block cannot be determined with certainty or if treatments used (such as drugs) may exacerbate AV block, prophylactic permanent pacing is indicated, especially if syncope may have been due to transient third-degree AV block (40,52).

Although third-degree AV block is most often preceded by bifascicular block, there is impressive evidence that the rate of progression of bifascicular block to third-degree AV block is slow. Furthermore, no single clinical or laboratory variable, including bifascicular block, identifies patients at high risk of death from a future bradyarrhythmia due to bundle branch block (48).

Of the many laboratory variables, the PR and HV intervals have been identified as possible predictors of third-degree AV 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 third-degree AV block and incidence of sudden death (43,45,49). Although most patients with chronic or intermittent third-degree AV block demonstrate prolongation of the HV interval during anterograde conduction, some investigators (50,51) 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 milliseconds (49). The evidence indicates that although the prevalence of prolonged HV is high, the incidence of progression to third-degree AV block is low. Because HV prolongation accompanies advanced cardiac disease and is associated with increased mortality, death is often not sudden or due to AV block but rather due to the underlying heart disease itself and nonarrhythmic cardiac causes (43,46–49,51,54–56).

Atrial pacing at electrophysiological study in asymptomatic patients as a means of identifying patients at increased risk of future high- or third-degree AV block is probably not justified. The probability of inducing block distal to the AV node (ie, intra- or infra-His) with rapid atrial pacing is low (47,50,51,57–60). Furthermore, failure to induce distal block cannot be taken as evidence that the patient will not develop third-degree AV block in the future. However, if atrial pacing induces nonphysiological infra-His block, some consider this an indication for pacing (57).

Indications for Permanent Pacing in Chronic Bifascicular and Trifascicular Block

Class I

1. **Intermittent third-degree AV block.** ***(Level of evidence: B)*** **(8–13,39)**
2. **Type II second-degree AV block.** ***(Level of evidence: B)*** **(18,20,24)**

Class IIa

1. **Syncope not proved to be due to AV block when other likely causes have been excluded, specifically ventricular tachycardia (VT).** ***(Level of evidence: B)*** **(40–51,53–58)**
2. **Incidental finding at electrophysiological study of markedly prolonged HV interval (≥100 milliseconds) in asymptomatic patients.** ***(Level of evidence: B)*** **(49)**
3. **Incidental finding at electrophysiological study of pacing-induced infra-His block that is not physiological.** ***(Level of evidence: B)*** **(57)**

Class IIb

None.

Class III

1. **Fascicular block without AV block or symptoms.** ***(Level of evidence: B)*** **(43,45,48,49)**
2. **Fascicular block with first-degree AV block without symptoms.** ***(Level of evidence: B)*** **(43,45,48,49)**

C. Pacing for Atrioventricular Block Associated With Acute Myocardial Infarction

Indications for permanent pacing after MI in patients experiencing AV block are related in large measure to the presence of intraventricular conduction defects. Unlike some other indications for permanent pacing, the criteria in patients with MI and AV block do not necessarily depend on the presence of symptoms. Furthermore, the requirement for temporary pacing in AMI does not by itself constitute an indication for permanent pacing (see ACC/AHA Guidelines for Management of Patients With Acute Myocardial Infarction [2]). The long-term prognosis for survivors of AMI who have had AV block is related primarily to the extent of myocardial injury and the character of intraventricular conduction disturbances rather than the AV block itself (11,61–64). Patients with AMI who have intraventricular conduction defects, with the exception of isolated left anterior fascicular block, have an unfavorable short- and long-term prognosis and an increased incidence of sudden death (11,24,61,63). This unfavorable prognosis is not necessarily due to development of high-grade AV block, although the incidence of such block is higher in postinfarction patients with abnormal intraventricular conduction (61,65).

When AV or intraventricular conduction block complicates AMI, the type of conduction disturbance, location of infarction, and relation of electrical disturbance to infarction must be considered as permanent pacing is contemplated. Even with data available, the decision is not always straightforward, because the reported incidence and significance of various conduction disturbances vary widely (66). Despite the use of thrombolytic therapy, which has decreased the incidence of AV block in AMI, mortality remains high in this patient group if AV block occurs (67–70).

Although more severe disturbances in conduction are in general associated with greater arrhythmic and nonarrhythmic mortality (61–66), the impact of preexisting bundle branch block on mortality after AMI is controversial (52,66). However, a particularly ominous prognosis is associated with left bundle branch block combined with advanced or third-degree AV block and with right bundle branch block combined with left anterior or left posterior fascicular block (41,52,62,64). Irrespective of whether the infarction is anterior or inferior, the development of an intraventricular conduction delay reflects extensive myocardial damage rather than an electrical problem in isolation (64). Although AV block that occurs during inferior MI can be associated with a favorable long-term clinical outcome, in-hospital survival is impaired, irrespective of temporary or permanent pacing in this situation (67,68,71,72). Furthermore, pacemakers should not be implanted if the peri-infarctional AV block is expected to resolve or not negatively impact long-term prognosis, as in the case of inferior MI (69).

Indications for Permanent Pacing after the Acute Phase of Myocardial Infarction*

Class I

1. **Persistent second-degree AV block in the His-Purkinje system with bilateral bundle branch block or third-degree AV block within or below the His-Purkinje system after AMI.** ***(Level of evidence: B)*** **(24,61–65)**
2. **Transient advanced (second- or third-degree) infranodal AV block and associated bundle branch block. If the site of block is uncertain, an electrophysiological study may be necessary.** ***(Level of evidence: B)*** **(61,62)**
3. **Persistent and symptomatic second- or third-degree AV block.** ***(Level of evidence: C)***

Class IIa

None.

Class IIb

1. **Persistent second- or third-degree AV block at the AV node level.** ***(Level of evidence: B)*** **(23)**

Class III

1. **Transient AV block in the absence of intraventricular conduction defects.** ***(Level of evidence: B)*** **(61)**

*These recommendations generally follow the ACC/AHA Guidelines for the Management of Patients With Acute Myocardial Infarction (2).

2. **Transient AV block in the presence of isolated left anterior fascicular block.** ***(Level of evidence: B)*** **(63)**
3. **Acquired left anterior fascicular block in the absence of AV block.** ***(Level of evidence: B)*** **(61)**
4. **Persistent first-degree AV block in the presence of bundle branch block that is old or age indeterminate.** ***(Level of evidence: B)*** **(61)**

D. 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 tachyarrhythmias alternating with periods of bradycardia or even asystole. Patients with this condition may be symptomatic from paroxysmal tachycardia or bradycardia or both. Correlation of symptoms with the above arrhythmias using an electrocardiogram (ECG), ambulatory electrocardiographic monitoring, or an event recorder is essential. This correlation may be difficult because of the intermittent nature of the episodes.

Sinus node dysfunction may express itself as chronotropic incompetence in which there is an inadequate sinus response to exercise or stress. Rate-responsive pacemakers have clinically benefited patients by restoring physiological heart rate during physical activity (73–75).

Sinus bradycardia is accepted as a physiological finding in trained athletes, who not uncommonly have a heart rate of 40 to 50 bpm while at rest and awake and may have a sleeping rate as slow as 30 bpm with sinus pauses or type I second-degree AV block producing asystolic intervals as long as 2.8 seconds (76–78). These findings are due to increased vagal tone.

Although sinus node dysfunction is frequently the primary indication for implantation of permanent pacemakers (73), permanent pacing in patients with sinus node dysfunction may not necessarily result in improved survival time (26,79), although symptoms related to bradycardia may be relieved (27,80). *Nonrandomized observational* studies suggest that dual-chamber pacing may improve survival compared with ventricular pacing (81–83). However, one *randomized prospective* study (84) of 225 patients with sinus node disease and intact AV nodal conduction followed for a mean of 40 months demonstrated no difference in overall or cardiac mortality between the groups receiving atrial versus ventricular pacing. Multiple small studies suggest that dual-chamber pacing improves quality of life and decreases morbidity (atrial fibrillation, stroke). Multiple prospective trials are ongoing to assess the superiority of dual-chamber versus ventricular-based pacing systems in this population (84a).

Indications for Permanent Pacing in Sinus Node Dysfunction

Class I

1. **Sinus node dysfunction with documented symptomatic bradycardia, including frequent sinus pauses that produce symptoms. In some patients, bradycardia is iatrogenic and will occur as a consequence of essential long-term drug therapy of a type and dose for which there are no acceptable alternatives.** ***(Level of evidence: C)*** **(27,73,79)**
2. **Symptomatic chronotropic incompetence (73–75).** ***(Level of evidence: C)*** **(27,73,79)**

Class IIa

1. **Sinus node dysfunction occurring spontaneously or as a result of necessary drug therapy, with heart rate <40 bpm when a clear association between significant symptoms consistent with bradycardia and the actual presence of bradycardia has not been documented.** ***(Level of evidence: C)*** **(26,27,73,78–80)**

Class IIb

1. **In minimally symptomatic patients, chronic heart rate <30 bpm while awake.** ***(Level of evidence: C)*** **(26,27,73,78–80)**

Class III

1. **Sinus node dysfunction in asymptomatic patients, including those in whom substantial sinus bradycardia (heart rate <40 bpm) is a consequence of long-term drug treatment.**
2. **Sinus node dysfunction in patients with symptoms suggestive of bradycardia that are clearly documented as not associated with a slow heart rate.**
3. **Sinus node dysfunction with symptomatic bradycardia due to nonessential drug therapy.**

E. Prevention and Termination of Tachyarrhythmias by Pacing

Under certain circumstances, an implanted pacemaker may be useful for treating patients with recurrent symptomatic ventricular and supraventricular tachycardias (85–94). Pacing can be useful in preventing and terminating arrhythmias. Reentrant rhythms including atrial flutter, paroxysmal reentrant supraventricular tachycardia, and VT may be terminated by a variety of pacing patterns, including programmed stimulation and short bursts of rapid pacing (95,96). 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.

Prevention of arrhythmias by pacing has been demonstrated in certain situations. In some patients with the long QT syndrome, recurrent pause-dependent VT may be prevented by continuous pacing (97). A combination of pacing and β-blockade has been reported to shorten the QT interval and help prevent sudden cardiac death (98,99).

Atrial synchronous ventricular pacing may prevent recurrences of reentrant supraventricular tachycardia (100), although this technique is rarely used, given the availability of catheter ablation and other alternative therapies. Although ventricular ectopic activity may be suppressed by such pacing in other conditions, serious or symptomatic arrhythmias are rarely prevented (101). In some patients with bradycardia-dependent atrial fibrillation, atrial pacing may be effective in reducing the frequency of recurrence (92). Dual-site right atrial pacing may offer additional benefits to single-site right atrial pacing in patients with symptomatic drug-refractory atrial fibrillation and concomitant bradyarrhythmias (93). In patients with sick sinus syndrome and intraatrial block (P wave >180 milliseconds), biatrial pacing may lower recurrence rates of atrial fibrillation (94).

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 (VF). Patients for whom an antitachycardia pacemaker has been prescribed have usually been unresponsive to antiarrhythmic drugs or were receiving agents that could not control their cardiac arrhythmias. When permanent antitachycardia pacemakers detect and interrupt supraventricular tachycardia, all pacing should be done in the atrium because adverse interactions have been reported (85,102) with use of ventricular pacing to interrupt supraventricular arrhythmias. Permanent antitachycardia pacing as monotherapy for VT is not appropriate, given that antitachycardia pacing algorithms are available in tiered-therapy ICDs that have the capability of cardioversion and defibrillation in cases when antitachycardia pacing is ineffective or causes acceleration of the treated tachycardia.

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

Class I

1. **Symptomatic recurrent supraventricular tachycardia that is reproducibly terminated by pacing after drugs and catheter ablation fail to control the arrhythmia or produce intolerable side effects.** ***(Level of evidence: C)*** **(86–88,90,91)**
2. **Symptomatic recurrent sustained VT as part of an automatic defibrillator system.** ***(Level of evidence: B)*** **(103–105)**

Class IIa

None.

Class IIb

1. **Recurrent supraventricular tachycardia or atrial flutter that is reproducibly terminated by pacing as an alternative to drug therapy or ablation.** ***(Level of evidence: C)*** **(85–88,90,91)**

Class III

1. **Tachycardias frequently accelerated or converted to fibrillation by pacing.**
2. **The presence of accessory pathways with the capacity for rapid anterograde conduction whether or not the pathways participate in the mechanism of the tachycardia.**

Pacing Indications to Prevent Tachycardia

Class I

1. **Sustained pause-dependent VT, with or without prolonged QT, in which the efficacy of pacing is thoroughly documented.** ***(Level of evidence: C)*** **(97,98)**

Class IIa

1. **High-risk patients with congenital long QT syndrome.** ***(Level of evidence: C)*** **(97,98)**

Class IIb

1. **AV reentrant or AV node reentrant supraventricular tachycardia not responsive to medical or ablative therapy.** ***(Level of evidence: C)*** **(87,88,92)**
2. **Prevention of symptomatic, drug-refractory, recurrent atrial fibrillation.** ***(Level of evidence: C)*** **(93,94)**

Class III

1. **Frequent or complex ventricular ectopic activity without sustained VT in the absence of the long QT syndrome.**
2. **Long QT syndrome due to reversible causes.**

F. Pacing in Hypersensitive Carotid Sinus and Neurally Mediated Syndromes

The hypersensitive carotid sinus syndrome is defined as syncope or presyncope 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 loss of vascular tone and hypotension. This effect is independent of heart rate changes.

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 (106). Pauses up to 3 seconds during carotid sinus massage are considered to be within normal limits. Such heart rate and hemodynamic responses may occur in normal subjects and patients with coronary artery disease. The cause-and-effect relation between the hypersensitive carotid sinus and the patient's symptoms must be made with great caution (107). Spontaneous syncope 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 (108,109). Because 10% to 20% of patients with this syndrome may have an important vasodepressor component of their reflex response, it is desirable 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 pacing.

Neurally mediated syncope accounts for 10% to 40% of syncope patients. Neurally mediated syncope and neurally mediated syndromes refer to a variety of clinical scenarios in which triggering of a neural reflex results in a usually self-limited episode of systemic hypotension characterized by both bradycardia and peripheral vasodilation (110). Vasovagal syncope is a term used to denote one of the most common clinical scenarios within the category of neurally mediated syncopal syndromes.

Considerable controversy exists concerning the role of permanent pacing in refractory neurally mediated syncope associated with significant bradycardia or asystole. Approximately 25% of patients have a predominant vasodepressor reaction without significant bradycardia (111). An additional large percentage of patients will have a mixed vasodepressor/vasoinhibitory component of their symptoms. While one group of investigators have noted some benefit of pacing in these patients (112,113), another study using a pacing rate 20% higher than the resting heart rate demonstrated that pacing did not prevent syncope any better than pharmacotherapy (106). Because most individuals with neurally mediated syncope have a slowing of heart rate after the fall in blood pressure, pacing may be ineffective in most patients. However, dual-chamber pacing, carefully prescribed on the basis of tilt-table test results, may be effective in reducing symptoms if the patient has a significant cardioinhibitory component to the cause of their symptoms (114). Preliminary results from a recent *randomized trial* (115) in highly symptomatic patients with bradycardia demonstrated that permanent pacing increased the time to first syncopal event ($P < .0007$). The actuarial rate of recurrent syncope at 1 year was 18.5% for pacemaker patients and 59.7% for control patients. Although spontaneous or provoked prolonged pauses are a concern in this population, the prognosis without pacing is excellent (116). However, several investigators have concluded that some patients with syncope of undetermined origin may benefit from pacing if findings strongly suggestive of bradycardic etiology are discovered or provoked at electrophysiological study (117,118).

The evaluation of patients with syncope of undetermined origin should take into account clinical status and not overlook other more serious causes of syncope such as ventricular tachyarrhythmias.

Indications for Permanent Pacing in Hypersensitive Carotid Sinus Syndrome and Neurally Mediated Syncope

Class I

1. **Recurrent syncope caused by carotid sinus stimulation; minimal carotid sinus pressure induces ventricular asystole of >3 seconds' duration in the absence of any medication that depresses the sinus node or AV conduction.** ***(Level of evidence: C)*** **(108,109)**

Class IIa

1. **Recurrent syncope without clear, provocative events and with a hypersensitive cardioinhibitory response.** ***(Level of evidence: C)*** **(108,109)**
2. **Syncope of unexplained origin when major abnormalities of sinus node function or AV conduction are discovered or provoked in electrophysiological studies.** ***(Level of evidence: C)***

Class IIb

1. *Neurally mediated syncope with significant bradycardia reproduced by a head-up tilt with or without isoproterenol or other provocative maneuvers. (Level of evidence: B)* **(112–115)**

Class III

1. **A hyperactive cardioinhibitory response to carotid sinus stimulation in the absence of symptoms.**
2. **A hyperactive cardioinhibitory response to carotid sinus stimulation in the presence of vague symptoms such as dizziness, light-headedness, or both.**
3. **Recurrent syncope, light-headedness, or dizziness in the absence of a hyperactive cardioinhibitory response.**
4. **Situational vasovagal syncope in which avoidance behavior is effective.**

G. Pacing in Children and Adolescents

The indications for permanent cardiac pacemaker implantation in the child or adolescent may be broadly considered as (1) symptomatic sinus bradycardia, (2) recurrent bradycardia-tachycardia syndromes, (3) congenital AV block, and (4) advanced second- or third-degree AV block, either surgical or acquired. Although the general indications for pacemaker implantation in children are similar to those in adults, there are several important considerations in young patients. First, an increasing number of patients are surviving complex surgical procedures for congenital heart disease that result in palliation rather than correction of circulatory physiology. The residua of impaired ventricular function and abnormal physiology may result in symptomatic bradycardia at rates that do not produce symptoms in persons with normal cardiovascular physiology. Hence, the indications for pacemaker implantation in these patients need to be based on correlation of symptoms with relative bradycardia rather than absolute heart rate criteria. Second, the clinical significance of bradycardia is age dependent: a heart rate of 45 bpm may be a normal finding in an adolescent, whereas the same rate in a newborn or infant indicates profound bradycardia.

Bradycardia and associated symptoms in children are often transient (eg, paroxysmal AV block or sinus arrest) and difficult to document. Although sinus node dysfunction (sick sinus syndrome) is increasingly recognized in pediatric patients, it is not itself an indication for pacemaker implantation. In the young patient with sinus bradycardia, the primary criterion for a pacemaker is the concurrent observation of a symptom (eg, syncope) with bradycardia (eg, heart rate <35 to 40 bpm or asystole >3 seconds) (25,27,119). In general, correlation of symptoms with bradycardia is determined by 24-hour ambulatory or transtelephonic electrocardiography. Symptomatic bradycardia (as defined) is considered an indication for pacemaker implantation, provided that other causes of the symptom(s) have been excluded. Alternative causes to be considered include seizures, breath holding, apnea, or neurally mediated mechanisms.

Bradycardia-tachycardia syndrome (sinus bradycardia alternating with atrial flutter or reentrant atrial tachycardia) is an increasingly frequent problem in young patients following surgery for congenital heart disease. Substantial morbidity and mortality have been observed in young patients with recurrent or chronic atrial flutter with the loss of sinus rhythm an independent risk factor for subsequent development of atrial flutter (120,121). Thus, both long-term atrial pacing at physiological rates as well as atrial antitachycardia pacing have been reported for treatment of sinus bradycardia and prevention or termination of recurrent episodes of tachycardia (122,123). To date the results of pacing for the bradycardia-tachycardia syndrome in children have been equivocal and the source of considerable controversy (124,125). It is clear that long-term drug therapy (eg, propranolol or amiodarone) deemed essential for the control of atrial flutter may result in symptomatic bradycardia in some patients, whereas in others the use of antiarrhythmic agents (eg, quinidine) may potentially increase the risk of ventricular arrhythmias or sudden death in the presence of profound bradycardia. Thus, in young patients with recurrent arrhythmias associated with the bradycardia-tachycardia syndrome, permanent pacing should be considered as an adjunctive form of therapy.

Indications for permanent pacing in young patients with congenital complete AV block have evolved on the basis of improved definition of the natural history of the disease as well as advances in pacemaker technology and diagnostic methods. For example, in recent studies it has been observed that pacemaker implantation may improve long-term survival and prevent syncopal episodes among asymptomatic patients with congenital complete AV block (126,127). Several criteria (average heart rate, pauses in the intrinsic rate, associated structural heart disease, prolonged QT interval, and exercise tolerance) must be considered in the asymptomatic patient with congenital complete AV block (128–130).

The use of cardiac pacing with β-blockade for prevention of symptoms in patients with the congenital long QT syndrome is supported by recent studies (98,131). This applies in particular to patients with pause-dependent initiation of ventricular tachyarrhythmias (132) or those with sinus bradycardia or advanced AV block in association with the congenital long QT syndrome (133,134). Although pacemaker implantation may reduce the incidence of symptoms in these patients, long-term benefit on risk of sudden cardiac arrest remains to be determined (98,131,133).

A poor prognosis has been established for patients with permanent postsurgical AV block who do not receive permanent pacemakers for rate support (135). Hence, the presence of advanced second- or third-degree AV block persisting for 7 to 14 days after cardiac surgery is considered a Class I indication for pacemaker implantation (136). However, the need for pacing in patients with transient AV block with residual bifascicular block is less certain, whereas patients in whom AV conduction returns to normal generally have a favorable prognosis (137).

Additional considerations that need to be made in pacemaker implantation in young patients include risk of paradoxic embolism with an endocardial lead system in the presence of residual intracardiac defects and the lifelong need for permanent cardiac pacing (138,139). Therefore, decisions about pacemaker implantation must also take into account implantation technique (transvenous versus epicardial) and long-term vascular access.

Indications for Permanent Pacing in Children and Adolescents

Class I

1. **Advanced second- or third-degree AV block associated with symptomatic bradycardia, congestive heart failure, or low cardiac output.** *(Level of evidence: C)*
2. **Sinus node dysfunction with correlation of symptoms during age-inappropriate bradycardia. The definition of bradycardia varies with the patient's age and expected heart rate.** *(Level of evidence: B)* **(25,27,119)**
3. **Postoperative advanced second- or third-degree AV block that is not expected to resolve or persists at least 7 days after cardiac surgery.** *(Level of evidence: B, C)* **(135,136)**
4. **Congenital third-degree AV block with a wide QRS escape rhythm or ventricular dysfunction.** *(Level of evidence: B)* **(127,129)**
5. **Congenital third-degree AV block in the infant with a ventricular rate <50 to 55 bpm or with congenital heart disease and a ventricular rate <70 bpm.** *(Level of evidence: B, C)* **(129,130)**
6. **Sustained pause-dependent VT, with or without prolonged QT, in which the efficacy of pacing is thoroughly documented.** *(Level of evidence: B)* **(97,98,131,132)**

Class IIa

1. **Bradycardia-tachycardia syndrome with the need for long-term antiarrhythmic treatment other than digitalis.** *(Level of evidence: C)* **(123,124)**
2. **Congenital third-degree AV block beyond the first year of life with an average heart rate <50 bpm or abrupt pauses in ventricular rate that are two or three times the basic cycle length.** *(Level of evidence: B)* **(128)**
3. **Long QT syndrome with 2:1 AV or third-degree AV block.** *(Level of evidence: B)* **(133,134)**

4. **Asymptomatic sinus bradycardia in the child with complex congenital heart disease with resting heart rate <35 bpm or pauses in ventricular rate >3 seconds.** ***(Level of evidence: C)***

Class IIb

1. **Transient postoperative third-degree AV block that reverts to sinus rhythm with residual bifascicular block.** ***(Level of evidence: C)*** **(137)**
2. **Congenital third-degree AV block in the asymptomatic neonate, child, or adolescent with an acceptable rate, narrow QRS complex, and normal ventricular function.** ***(Level of evidence: B)*** **(126,127)**
3. **Asymptomatic sinus bradycardia in the adolescent with congenital heart disease with resting heart rate <35 bpm or pauses in ventricular rate >3 seconds.** ***(Level of evidence: C)***

Class III

1. **Transient postoperative AV block with return of normal AV conduction within 7 days.** ***(Level of evidence: B)*** **(136,137)**
2. **Asymptomatic postoperative bifascicular block with or without first-degree AV block.** ***(Level of evidence: C)***
3. **Asymptomatic type I second-degree AV block.** ***(Level of evidence: C)***
4. **Asymptomatic sinus bradycardia in the adolescent with longest RR interval <3 seconds and minimum heart rate >40 bpm.** ***(Level of evidence: C)*** **(140)**

H. Pacing in Specific Conditions

Hypertrophic Obstructive Cardiomyopathy Early *observational* studies suggested that pacing the right ventricular apex would reduce the LV outflow gradient. In patients with severely symptomatic hypertrophic cardiomyopathy, implantation of a dual-chamber pacemaker with a short AV delay has been shown to decrease the magnitude of LV outflow obstruction and alleviate symptoms (141–143). These findings come from *nonrandomized unblinded* studies. The mechanisms by which pacing improves the LV outflow gradient are not completely understood. Pacing therapy can change the ventricular contraction pattern by prematurely activating part of the ventricle, creating a regional dyssynchrony. This early paced portion of the ventricle faces low chamber pressure and stress and contracts against a lower afterload (144). Altered LV activation causes disordered ventricular contractility with late septal activation, increases LV systolic dimension, and reduces systolic anterior motion of the mitral valve. Thus, LV outflow obstruction is reduced and the atrial contribution to LV filling is maintained. Selection of an optimal AV delay appears to be critical in achieving an optimal hemodynamic result (142,145). The optimal AV delay appears to be the longest AV interval that consistently results in a completely paced QRS morphology (146). Some patients with too short a native AV delay may benefit from AV junction ablation so that the paced and sensed AV delay can be optimized (147). Pacing may cause thinning of the LV wall and decrease outflow obstruction (142,148). Two recent *observational* studies have suggested that a decrease in LV outflow gradient produced by temporary dual-chamber pacing may have adverse effects on ventricular filling and cardiac output (149,150). Another small *observational* study of dual-chamber pacing in hypertrophic cardiomyopathy patients without outflow obstruction failed to show significant hemodynamic or short-term benefit (151).

One study (142) demonstrated that dual-chamber pacing eliminated or ameliorated symptoms in 74 of 88 patients. Patients in this study were not required to have a beneficial hemodynamic response to temporary pacing as a selection criterion for permanent pacing. A recent *randomized* study (152) demonstrated that DDD pacing reduced outflow tract gradient and improved New York Heart Association (NYHA) functional class. One long-term study (153) in eight patients supported the long-term benefit of dual-chamber pacing in this group of patients. The outflow gradient was reduced even after cessation of pacing, suggesting some ventricular remodeling had occurred secondary to pacing. Although these data are encouraging, a recent *randomized, double-blind* crossover study (154) of 19 patients demonstrated no significant subjective or exercise capacity improvement in the paced versus nonpaced group at 2 to 3 months of follow-up, despite a significant decrease in LV outflow gradient. However, several individual patients in this study demonstrated symptomatic and hemodynamic improvement from dual-chamber pacing. Dual-chamber pacing may improve symptoms and LV outflow gradient in pediatric patients. However, rapid atrial rates, rapid AV conduction, and congenital mitral valve abnormalities may preclude effective pacing in some patients (155).

The lack of large, prospective, placebo-controlled data makes this indication for permanent pacing controversial. Currently there are no data available to support that pacing alters the clinical course of the disease or improves survival. Therefore, routine implantation of dual-chamber pacemakers should not be advocated in all patients with symptomatic hypertrophic obstructive cardiomyopathy.

Pacing Indications for Hypertrophic Cardiomyopathy

Class I

Class I indications for sinus node dysfunction or AV block as previously described. ***(Level of evidence: C)***

Class IIa

None.

Class IIb

1. **Medically refractory, symptomatic hypertrophic cardiomyopathy with significant resting or provoked LV outflow obstruction.** ***(Level of evidence: C)*** **(142,145,146)**

Class III

1. **Patients who are asymptomatic or medically controlled.**
2. **Symptomatic patients without evidence of LV outflow obstruction.**

Idiopathic Dilated Cardiomyopathy Several *observational* studies have shown limited improvement in patients with symptomatic dilated cardiomyopathy refractory to medical therapy with dual-chamber pacing with a short AV delay (156–159). Theoretically, a short AV delay may optimize the timing of mechanical AV synchrony and ventricular filling time. In patients with prolonged PR intervals >200 milliseconds, diastolic filling time may be improved by dual-chamber pacing with a short AV delay (17). In one study (157), cardiac output was increased 38% by shortening AV delay when the average PR interval was 283 milliseconds before pacing. Permanent pacing in symptomatic patients with drug-refractory dilated cardiomyopathy and a prolonged PR interval may be useful if short-term benefit is demonstrated in acute studies. However, at this time no long-term data are available, and there is no consensus of opinion for this indication. The mechanisms by which dual-chamber pacing might benefit patients with dilated cardiomyopathy are poorly understood. One hypothesis is that a well-timed atrial contraction primes the ventricles and decreases mitral regurgitation, thus augmenting stroke volume and arterial pressure. Several studies have not demonstrated improvement in cardiac output with dual-chamber pacing in patients with congestive heart failure (160,161). One *randomized controlled* trial of 12 patients showed no significant benefit of VDD pacing through a range of PR intervals despite the presence of both tricuspid and mitral regurgitation (160). One study (162) in 89 patients with LV dysfunction suggested that VVI pacing in the right ventricular outflow tract (simulating a normal high to low ventricular activation) improved cardiac output by 18.8% when compared with pacing the right ventricular apex. Preliminary data (163,164) suggest that simultaneous biventricular pacing may improve cardiac hemodynamics and thus lead to subjective and objective symptom improvement. *Prospective controlled* trials are under way to confirm these initial findings and further define the benefit of biventricular pacing in patients with symptomatic, drug-refractory dilated cardiomyopathy. Overall there are sparse long-term data to show improvement in hemodynamics, symptom relief, or survival for pacing in dilated cardiomyopathy. Even less data exist in patients with ischemic cardiomyopathy.

Pacing Indications for Dilated Cardiomyopathy

Class I

Class I indications for sinus node dysfunction or AV block as previously described. *(Level of evidence: C)*

Class IIa

None.

Class IIb

1. **Symptomatic, drug-refractory dilated cardiomyopathy with prolonged PR interval when acute hemodynamic studies have demonstrated hemodynamic benefit of pacing.** *(Level of evidence: C)* **(17,156–158)**

Class III

1. **Asymptomatic dilated cardiomyopathy.**
2. **Symptomatic dilated cardiomyopathy when patients are rendered asymptomatic by drug therapy.**
3. **Symptomatic ischemic cardiomyopathy.**

Cardiac Transplantation The incidence of bradyarrhythmias after cardiac transplantation varies from 8% to 23% (165–167). The majority of bradyarrhythmias are associated with sinus node dysfunction. Because of symptoms and impaired recovery and rehabilitation, some transplant programs recommend more liberal use of cardiac pacing for persistent postoperative bradycardia. About 50% of patients show improvement within 6 to 12 months, and long-term pacing is often unnecessary in a large number of patients (168–170). Significant bradyarrhythmias and asystole have been associated with reported cases of sudden death (171). No predictive factors have been identified to indicate which patients will develop post-transplantation bradyarrhythmias. In some patients the need for pacing may be transient. The benefits of the atrial contribution to cardiac output and chronotropic competence may optimize the patient's functional status. Attempts to temporarily treat bradycardia with measures such as theophylline (172) may minimize the need for pacing. Post-transplant patients who have irreversible sinus node dysfunction or AV block with previously stated Class I indications should have permanent pacemakers.

Pacing Indications After Cardiac Transplantation

Class I

1. **Symptomatic bradyarrhythmias/chronotropic incompetence not expected to resolve and other Class I indications for permanent pacing.** *(Level of evidence: C)*

Class IIa

None.

Class IIb

1. **Symptomatic bradyarrhythmias/chronotropic incompetence that, although transient, may persist for months and require intervention.** *(Level of evidence: C)*

Class III

1. **Asymptomatic bradyarrhythmias after cardiac transplantation.**

I. Selection of Pacemaker Device

Once the decision has been made to implant a pacemaker in a given patient, the clinician must decide among a large number of available pacemaker generators and leads. Generator choices include single- versus dual-chamber devices, unipolar versus bipolar configuration, presence and type of sensor for rate response, advanced features such as automatic mode switching, size, battery capacity, and cost. Lead choices include polarity, type of insulation material, fixation mechanism (active versus passive), presence of steroid elution, and typical pacing impedance. Other factors that importantly influence the choice of pacemaker system components include the capabilities of the pacemaker programmer, which provides

JACC Vol. 31, No. 5
April 1998:1175–209

Table 1. Guidelines for Choice of Pacemaker Generator in Selected Indications for Pacing

	Sinus Node Dysfunction	AV Block	Neurally Mediated Syncope or Carotid Sinus Hypersensitivity
Single-chamber atrial pacemaker	• No suspected abnormality of AV conduction and not at increased risk for future AV block • Maintenance of AV synchrony during pacing desired • Rate response available if desired	Not appropriate	Not appropriate (unless AV block systematically excluded)
Single-chamber ventricular pacemaker	• Maintenance of AV synchrony during pacing not necessary • Rate response available if desired	• Chronic atrial fibrillation or other atrial tachyarrhythmia or maintenance of AV synchrony during pacing not necessary • Rate response available if desired	• Chronic atrial fibrillation or other atrial tachyarrhythmia • Rate response available if desired
Dual-chamber pacemaker	• AV synchrony during pacing desired • Suspected abnormality of AV conduction or increased risk for future AV block • Rate response available if desired	• AV synchrony during pacing desired • Atrial pacing desired • Rate response available if desired	• Sinus mechanism present • Rate response available if desired
Single-lead, atrial-sensing ventricular pacemaker	Not appropriate	• Normal sinus node function and no need for atrial pacing • Desire to limit the number of pacemaker leads	Not appropriate

AV = atrioventricular.

the link between the pacemaker system and the physician, and local availability of technical support.

Even after selecting and implanting the pacing system, the physician has a number of options for programming the device. In modern single-chamber pacemakers, programmable features include pacing mode, lower rate, pulse width and amplitude, sensitivity, and refractory period. Dual-chamber pacemakers have the same programmable features as well as maximum tracking rate, AV delay, and others. Rate-responsive pacemakers require programmable features to regulate the relation between sensor output and pacing rate and to limit the maximum sensor-driven pacing rate. These programmable parameters must be individually adjusted for each patient, and the choice of one programmable parameter will often depend on the availability of another parameter. For example, in a patient with complete AV block and paroxysmal atrial fibrillation, a dual-chamber pacemaker without mode-switching capability most appropriately might be programmed to DDIR* mode, whereas in the same patient, a pacemaker with mode-switching capability most appropriately might be programmed to DDDR mode with mode switching. In recent years, with the advent of more sophisticated pacemaker generators, optimal programming of pacemakers has become increasingly complex and device specific and requires specialized knowledge on the part of the physician.

Many of these considerations are beyond the scope of this document. The discussion below focuses on the most fundamental choices the clinician has with respect to the pacemaker prescription: those that have the greatest impact on procedural time and complexity, follow-up, patient outcome, and cost: (1) the choice among single-chamber ventricular pacing, single-chamber atrial pacing, and dual-chamber pacing, and (2) whether or not to use a generator that incorporates a sensor for rate-responsive pacing.

Table 1 gives brief guidelines on the appropriateness of different pacemakers for the most commonly encountered indications for pacing. Figure 1 is a decision tree for selecting a pacing system in a patient with AV block. Figure 2 is a decision tree for selecting a pacing system in a patient with sinus node dysfunction.

An important challenge in selecting a pacemaker system is anticipating progression of abnormalities of automaticity and conduction and selecting a system that will best accommodate these developments. Thus, it is reasonable to select a pacemaker with more extensive capabilities than needed at the time of implantation but that may prove useful in the future. Some patients with sinus node dysfunction and paroxysmal atrial fibrillation, for example, may develop AV block in the future (as a result of natural progression of disease, drug therapy, or catheter ablation) and may ultimately benefit from a dual-chamber pacemaker with mode-switching capability. Patients who are likely to develop ventricular tachyarrhythmias, for which an ICD would be warranted, should receive a pacemaker that is compatible with ICDs.

Newer Technical Innovations *Rate-responsive pacemakers.* An increasing percentage of pacemakers implanted in the United States incorporate sensors to detect states of exercise and trigger accelerations in pacing rate. An industrywide survey in 1996 indicates that 83% of all generators implanted in 1996 in the United States had rate response as a programmable option. Among pacemaker patients who are chronotropically incompetent (ie, unable to increase sinus node rate appropriately with exercise), rate-responsive pacemakers allow

*This and other three- or four-letter notations conform to the NASPE/BPEG generic pacemaker code (173).

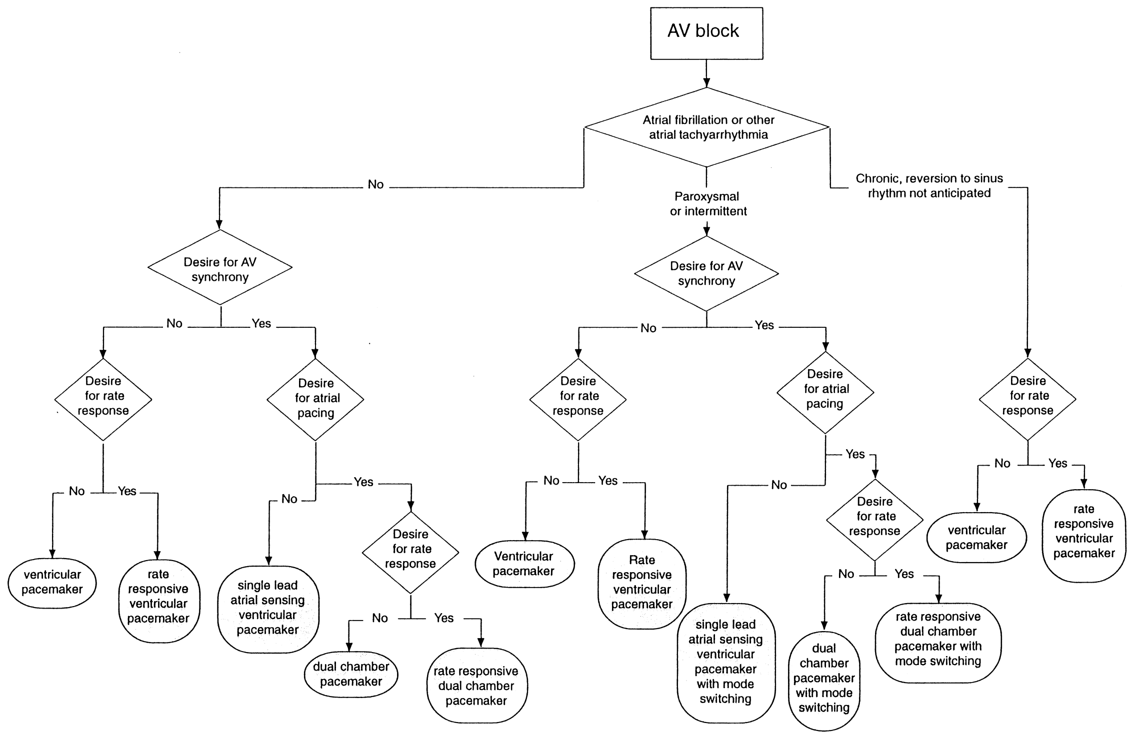

Figure 1. Selection of pacemaker systems for patients with atrioventricular (AV) block.

for increases in pacing rates with exercise and have been shown to improve exercise capacity and quality of life.

In the United States, the vast majority of sensors incorporated into rate-responsive implantable pacemakers are piezoelectric crystals or accelerometers that detect motion, vibration, pressure, or acceleration. Other technologies using sensors that measure minute ventilation or QT interval may provide a heart rate response more proportional to exercise than piezoelectric sensors or accelerometers. An advantage of all of these sensor technologies is that they do not require specialized pacemaker leads, although minute ventilation sensing requires a bipolar lead. An older technique that measured circulating blood temperature has largely been abandoned.

The challenge of appropriately adjusting the response to exercise of these generators in individual patients is becoming increasingly recognized. To facilitate optimal programming of rate-response capability, recently introduced generators incorporate procedures for initial programming of rate-response parameters, subsequent automatic adjustment of these parameters, and retrievable diagnostic data (such as heart rate histograms or heart rate plots) to assess the appropriateness of the rate response.

Single-lead VDD pacemaker systems. Despite advances in rate-responsive pacemakers, it is widely appreciated that the best signal to guide heart rate response to exercise (and other forms of physiological stress) is a normally functioning sinus node. Most commonly, dual-chamber pacemakers incorporating separate atrial and ventricular leads are used to detect atrial depolarization. However, single transvenous lead pacing systems have been developed that are capable of sensing atrial depolarization. The distal end of the lead is positioned in the right ventricle for ventricular pacing and sensing; a pair of electrodes is incorporated in the more proximal portion of the lead body lying within the right atrial cavity for atrial sensing. With current technology, single-lead VDD pacing systems are not capable of atrial pacing. The atrial signal sensed by single-lead VDD pacemakers has a less consistent amplitude than that typically sensed by conventional dual-chamber pacemakers and varies significantly with posture, but sensing performance is generally satisfactory (174). Single-lead VDD pacemaker systems are a reasonable alternative to dual-chamber pacemakers in patients with AV block in whom atrial pacing is not required and in whom simplicity of implantation or avoidance of two leads is desired.

Automatic mode switching. When nonphysiological atrial tachyarrhythmias, such as atrial fibrillation or flutter, occur paroxysmally in a patient with a dual-chamber pacemaker programmed to conventional DDD or DDDR mode, the tachyarrhythmia will generally be tracked near the programmed maximum tracking rate, leading to an undesirable acceleration of ventricular pacing rate. Newer dual-chamber generators incorporate algorithms for detecting rapid, nonphysiological atrial rates and automatically switch modes to one that does not track atrial activity, such as DDI or DDIR. When the atrial tachyarrhythmia terminates, the pacemaker automatically reverts back to the DDD or DDDR mode. This

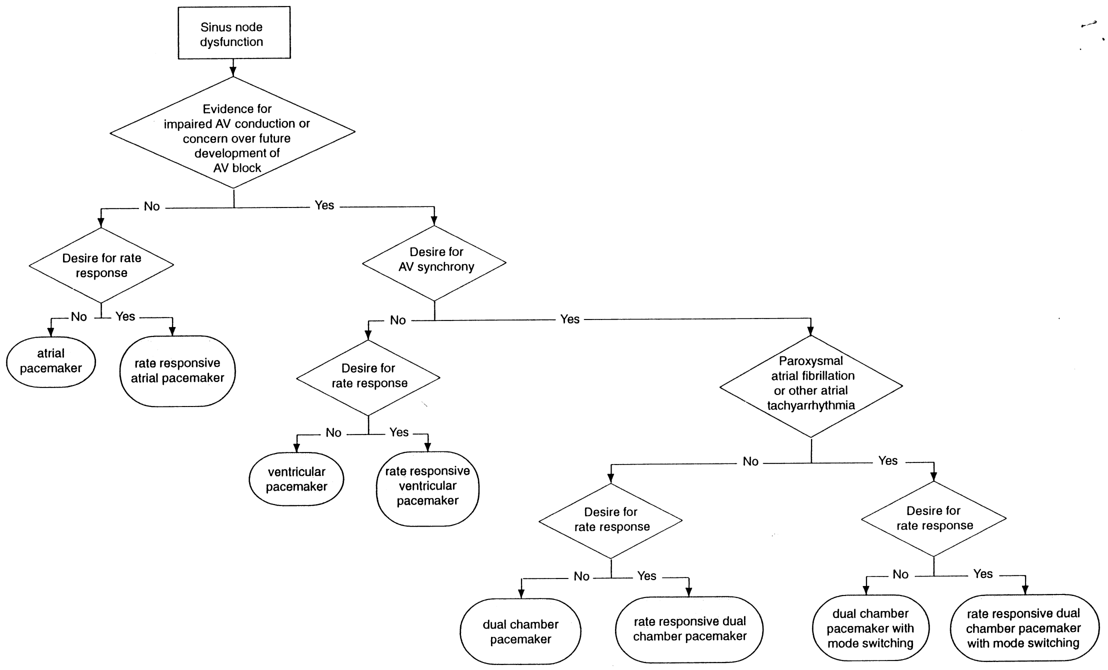

Figure 2. Selection of pacemaker systems for patients with sinus node dysfunction. AV indicates atrioventricular.

automatic mode-switch feature is especially helpful in patients with AV block and paroxysmal atrial fibrillation and expands the usefulness of dual-chamber pacemakers in such patients.

Pacemaker leads. The vast majority of implanted pacemakers use transvenous endocardial leads, with the remainder using epicardial leads. Transvenous leads may be bipolar or unipolar in configuration. Bipolar configurations have the advantage of avoiding myopotential inhibition and skeletal muscle stimulation, and an increasingly important advantage is that, unlike most unipolar pacing systems, they are compatible with concomitantly implanted ICDs. However, some manufacturers' bipolar leads have higher failure rates than their unipolar leads.

The insulation material used in pacemaker leads is either silicone rubber or polyurethane. Polyurethane-insulated leads have a thinner diameter and better handling characteristics than silicone-insulated leads. However, some bipolar lead models with polyurethane insulation have shown unacceptably high failure rates due to degradation of the insulation. It is possible that more recently introduced polyurethane leads, using different polymers and different manufacturing processes, will avoid these unacceptably high failure rates.

Active fixation leads, in which the distal tip of the lead incorporates a small helical screw for fixation to the endocardium, are an alternative to passive fixation leads. Active fixation leads allow for more alternatives in the site of endocardial attachment. For instance, whereas a passive fixation ventricular lead generally must be positioned in the right ventricular apex, an active fixation lead may be positioned in the apex, outflow tract, or inflow tract of the right ventricle. Active fixation leads have an additional advantage of greater ease of extraction after long-term implantation. A disadvantage of active fixation leads is that they generally have higher chronic capture thresholds than do passive fixation leads.

An important advance in pacemaker leads is the development of leads with lower capture thresholds, which result in less battery consumption during pacing. Steroid eluting leads incorporate at their distal tip a small reservoir of corticosteroid that slowly elutes into the interface between the lead electrode and the endocardium, reducing the inflammation and fibrosis that normally occur at this interface. As a result, steroid-eluting leads have significantly lower long-term capture thresholds than leads not incorporating steroid. The benefit of steroid elution was originally demonstrated in passive fixation transvenous leads (175); more recently, the benefit has also been demonstrated in active fixation transvenous leads (176) and epicardial leads (177). Similar improvements in capture thresholds have been achieved with modification in electrode shape, size, and composition (178).

Methodology of Comparing Different Pacemaker Generators and Configurations Two or more pacemaker modes can be compared with respect to exercise capacity, quality of life, clinical end points (such as death, heart failure, atrial fibrillation, and stroke), and cost. For end points such as exercise capacity or quality of life, pacemaker modes can be compared using a randomized crossover study design, provided that the patients have pacing systems that can be programmed to each of these modes. (For example, dual-chamber, rate-responsive pacemakers can be crossed over between VVIR and DDDR pacing). Studies that compare clinical end points require long-term follow-up without crossover. In long-term studies patients can be randomly assigned to receive different types of pacemakers (eg, single-chamber ventricular pacemakers versus single-chamber atrial pacemakers), or all patients may receive a single type of pacemaker system (eg, dual-chamber, rate-responsive) and be randomly assigned to different modes (eg, VVIR versus DDDR).

Quality-of-life measures have recently been emphasized as important end points when comparing different modes of pacing, and there are important considerations in the choice of the instrument used to measure quality of life (179–181). Although the quality of life experienced with different modes of pacing may be compared using short-term crossover studies, long-term studies that include quality-of-life end points may reflect effects of chronic adaptation to stimulation not detectable in short-term comparisons. Several recent or ongoing long-term randomized comparisons of pacing modes have quality-of-life end points (83).

Pacing in Sinus Node Dysfunction *Short-term outcomes.* Short-term crossover studies in patients with sinus node dysfunction have shown improved quality of life in dual-chamber versus ventricular pacing (180,182). There are conflicting data regarding any improvement in maximum exercise performance in rate-responsive dual-chamber compared with rate-responsive ventricular pacing (182,183).

Long-term outcomes. Over the past decade a number of *nonrandomized observational* studies have been published comparing atrial-based pacing (either atrial pacemakers or dual-chamber pacemakers) to ventricular pacing in patients with sinus node dysfunction. These studies have recently been reviewed (83,184,185). A consistent finding is that the incidence of atrial fibrillation is lower in patients receiving atrial-based pacemakers than in those receiving ventricular pacemakers; atrial-based pacing is associated with a reduction in risk of atrial fibrillation averaging 74% (185). The findings of the studies were mixed with regard to mortality end points: some studies showed a lower mortality in atrial-based pacemaker patients and some showed no significant difference. These studies suffer from limitations common to all nonrandomized studies, most importantly, uncertainty as to the clinical equivalence of the patient groups. In some of these studies the patient groups appear to be well matched, whereas in others there is insufficient information to assess their comparability.

Andersen et al (84) published a *randomized* study comparing pacemaker modes with long-term follow-up in patients with sinus node dysfunction. Two hundred twenty-five patients were randomly assigned to atrial and ventricular pacing. During a mean of 40 months of follow-up, there were significantly fewer thromboembolic events in the atrial paced patients. There was a trend toward less atrial fibrillation in the atrial paced group, but it did not reach statistical significance. The study was not powered to detect a mortality difference between the two patient groups. However, when follow-up was extended to 8 years, atrial pacing was associated with significantly decreased "all-cause" and "cardiovascular" mortality compared to ventricular pacing (84a).

In summary, available data suggest that in patients with sinus node dysfunction, the incidence of atrial fibrillation in patients receiving atrial or dual-chamber pacemakers may be lower than in patients receiving ventricular pacemakers. Published studies do not adequately address the issues of other clinical end points, such as heart failure, mortality, or quality of life.

Role of single-chamber atrial pacemakers. Single-chamber atrial pacemakers, with rate-responsive capability if appropriate, have been advocated for patients with sinus node dysfunction but no evidence of AV block (21,179,186–188). Use of single-chamber pacemakers is limited by concerns about subsequent development of AV block. The risk of developing significant AV block after atrial pacemaker implantation for sinus node dysfunction has been estimated to be 0.6% to 3.0% per year, with bundle branch block but not AV Wenckebach rate being predictive of a higher likelihood of subsequent AV block (186,187,189,190). In selected patients with sinus node dysfunction, use of single-chamber atrial pacemakers is an acceptable approach that maintains normal AV synchrony without the added cost and extra lead of a dual-chamber pacemaker system but with a small risk of subsequent development of AV block requiring pacemaker revision. With rate-responsive atrial pacemakers, the risk of developing hemodynamically significant first-degree AV block during rate accelerations has not been extensively studied but may be significant (191).

A randomized study of DDDR versus VVIR pacing in patients with sinus node dysfunction is ongoing, with end points of total mortality, atrial fibrillation, stroke, heart failure, quality of life, and cost (83).

Pacing in Atrioventricular Block *Short-term outcomes.* A number of short-term crossover studies have compared pacing modes in patients with AV block with respect to quality of life and exercise capacity. These studies have recently been reviewed in depth (83,180). Studies comparing dual-chamber pacing with non–rate-responsive ventricular pacing have shown improved exercise capacity and symptomatology with dual-chamber pacing. Studies comparing rate-responsive ventricular pacing with non–rate-responsive ventricular pacing have shown similar advantages with rate-responsive ventricular pacing. However, studies comparing dual-chamber pacing with rate-responsive ventricular pacing have shown no significant difference in exercise capacity; with respect to symptoms, most but not all have shown an advantage of dual-chamber pacing. It is likely that the symptomatic advantage of dual-chamber pacing over rate-responsive ventricular pacing is derived from the maintenance of AV association during rest and low-level activity.

Long-term outcomes. Two *nonrandomized observational* studies comparing patients with AV block who received dual-chamber pacemakers or ventricular pacemakers have shown improved survival associated with implantation of dual-chamber pacemakers among those patients with heart failure but no difference in survival between the two pacing modes among patients without heart failure (74,192). In an ongoing study, patients with AV block are randomly assigned to receive a ventricular pacemaker or a dual-chamber pacemaker; the primary end point is total mortality (83).

Pacing in the Elderly More than 85% of pacemaker recipients are at least 64 years old (193). Elderly pacemaker patients are the rule, not the exception.

It has been suggested that elderly patients requiring pacing should be considered for less sophisticated devices, eg, single-chamber ventricular pacemakers or non–rate-responsive pacemakers. However, studies in elderly patients show improved exercise capacity and alleviated symptoms with rate-responsive ventricular pacing or dual-chamber pacing compared with non–rate-responsive ventricular pacing (75,194). A retrospective analysis of 36,312 elderly Medicare patients receiving pacemakers suggested that dual-chamber pacing is associated with improved survival compared with ventricular pacing, even after correction for confounding variables (195).

A prospective, randomized long-term comparison of rate-responsive ventricular pacing and rate-responsive dual-chamber pacing in elderly patients has recently been completed (G.A. Lamas, PACE Study, unpublished data, 1997). The primary end point of the trial was quality-of-life measures; only transient improvement in a minority of the quality-of-life measures was found to be associated with rate-responsive dual-chamber pacing compared with rate-responsive ventricular pacing.

On the basis of these studies, rate-responsive ventricular pacing and dual-chamber pacing appear to offer benefits over fixed-rate ventricular pacing with respect to quality of life in elderly patients, but there may not be any benefit of dual-chamber pacing over rate-responsive ventricular demand pacing. It does not appear appropriate to uniformly withhold use of dual-chamber or rate-responsive pacemakers in the elderly, although such a decision is appropriate in any patient who is extremely sedentary or has a limited life expectancy.

Optimizing Pacemaker Technology and Cost The cost of a pacemaker system increases with its degree of complexity and sophistication. For example, the cost of a dual-chamber pacemaker system exceeds that of a single-chamber system with respect to the cost of the generator (additional $1000), the second lead (approximately $900), additional implantation time and supplies, and additional follow-up. Similarly, the cost of a rate-responsive generator exceeds that of a non–rate-responsive generator by $500 to $1000. Against these additional costs are the potential benefits of the more sophisticated systems with respect to quality of life, morbidity, and mortality. Little is known about the cost-effectiveness of the additional features of more complex pacemaker systems. Several ongoing trials assessing the clinical benefits of dual-chamber or rate-responsive pacing include economic analyses to estimate the incremental cost-effectiveness of these features (83).

Approximately 16% of pacemaker implantations are for replacement of generators; of those, 76% are replaced because their batteries have reached end of service (193). Hardware and software (ie, programming) features of pacemaker systems that prolong useful battery longevity may improve the cost-effectiveness of pacing. For example, optimal programming of output voltages, pulse widths, and AV delays can markedly decrease battery drain; a recent study showed that expert programming of pacemaker generators can have a major impact on longevity, prolonging it by an average of 4.2 years compared with nominal settings (196). Extensive diagnostic capabilities, which typically add $500 to $1000 to the cost of a pacemaker generator, may allow for optimal programming by the experienced physician with regard to improved device longevity. Newer lead designs, such as those incorporating steroid elution or high pacing impedance, allow for less current drain; the cost of such leads is approximately $125 greater than that of conventional leads. Future generators that automatically determine whether a pacing impulse results in capture may allow for programming outputs closer to threshold values than conventional generators, and this new technology may also have a major impact on device longevity. Although all of these features arguably should prolong generator life, there are other constraints on the useful life of a pacemaker generator, including battery drain not directly related to pulse generation and the limited life expectancy of many pacemaker recipients; rigorous studies supporting the overall cost-effectiveness of advanced pacing features are lacking.

The cost of pacemaker implantation may vary between different locations within a hospital (eg, cardiac catheterization laboratory versus operating room); costs can be minimized by selecting the most economical site for implantation that preserves excellent patient outcome. There has been a trend to shorter hospital stays for pacemaker implantations, and some implantations are now being performed on an outpatient basis. Reuse of explanted pacemakers, not currently performed to any extent in the United States, may eventually add significantly to the cost-effectiveness of cardiac pacing (197).

J. Pacemaker Follow-up

After implantation of a pacemaker, careful follow-up and continuity of care are required. The committee considered the advisability of extending the scope of these guidelines to include recommendations for follow-up and device replacement. In general, follow-up is dictated by the patient's disease substrate, the device used, and evolving technology. The North American Society of Pacing and Electrophysiology has published a comprehensive series of reports on antibradycardia pacemaker follow-up (198–200). In addition, the Health Care Financing Administration has established guidelines for monitoring of patients covered by Medicare who have antibradycardia pacemakers (201). These documents are endorsed by this writing group.

Many of the same considerations are relevant to both pacemaker and ICD follow-up. Programming undertaken at implantation should be reviewed before discharge and changed accordingly at subsequent follow-up visits as indicated by interrogation and testing. With careful attention to programming pacing amplitude, pulse width, and diagnostic functions, battery life can be significantly enhanced without compromising patient safety. The frequency of follow-up is dictated by multiple factors, including other cardiovascular or medical problems managed by the physician involved, the age of the pacemaker, and the results of transtelephonic testing. In patients who are pacemaker dependent, clinical evaluation may be more frequent than for those who are not pacemaker dependent. In general, follow-up usually includes assessment of battery status, pacing threshold and pulse width, sensing function, and lead integrity.

Because the indications for device implantation are evolving and some of the original indications for a particular patient may have been controversial, future replacement decisions may be more or less certain and must be individualized.

II. Indications for Implantable Cardioverter-Defibrillator Therapy

A. Background

ICDs were originally developed and have been most frequently used for prevention of sudden cardiac death in patients with life-threatening ventricular arrhythmias such as sustained VT or VF (202–205). Epidemiological studies report high rates of recurrence of these life-threatening arrhythmias (30% to 50% in 2 years) during follow-up. Early *observational* reports documenting efficacy in reversion of sustained VT and VF (103–105,202,203,205–215) have now been supplemented by large *prospective* and sometimes *randomized* single-center and multicenter studies with long-term outcome data (204,216–221). Enrollment in these trials has included patients with coronary and noncoronary heart diseases with a wide range of ventricular function and coexisting disorders.

These studies uniformly document sudden cardiac death recurrence rates that average 1% to 2% annually after device implantation in these populations. Simultaneously, rapid technological evolution of ICD systems has occurred. The ICD has evolved from a short-lived nonprogrammable device requiring a thoracotomy for lead insertion into a multiprogrammable antiarrhythmia device inserted almost exclusively without thoracotomy, now capable of treating bradycardia, VT, and VF (222–224). Clinical studies have recorded major improvements in implant risk, system longevity, symptoms associated with arrhythmia recurrences, quality of life, and diagnosis and management of inappropriate device therapy (103,216–218,225–229). Implantation, follow-up, and replacement of these devices is a complex process requiring familiarity with device capabilities, adequate case volume, continuing education, and skill in the management of ventricular arrhythmias, therefore mandating involvement of a trained electrophysiologist (230) to provide an optimal personnel team for patient safety and device management. A substantial new body of information has emerged regarding the clinical outcome of patients with VT or VF treated with currently available antiarrhythmic therapies. There are currently three major therapeutic options to reduce or prevent VT or VF in patients at risk for these arrhythmias. These are

1. Antiarrhythmic drug therapy selected by electrophysiological study or ambulatory monitoring or prescribed empirically.
2. Ablative techniques applied at cardiac surgery or percutaneously using catheter techniques.
3. Implantation of a cardioverter-defibrillator device system.

A combination of ICD therapy with drugs or ablation is also frequently used. Currently the largest clinical experience is with combined antiarrhythmic drug and ICD therapy.

B. Clinical Efficacy of ICD Therapy

ICD devices have been extensively evaluated in *prospective* clinical trials, and clinical experience now exceeds 100,000 implants worldwide since the inception of this therapy (103–105,202–221). ICDs have been clearly documented to revert sustained ventricular tachyarrhythmias, including pace termination of sustained VT and shock reversion of VF. Large series have shown nonthoracotomy systems can be implanted with an average procedural mortality of 0.5% (217) to 0.8% (216). The ICD has been shown to terminate VF successfully in 98.8% (217) and 98.6% (216) of episodes. VT has been converted with antitachycardia pacing in 89.4% (216) to 91.2% (217) of episodes, with further successful conversions (98%) using shock therapy. Inappropriate therapy, typically for atrial fibrillation, with a rapid ventricular response, has been noted in 5% to 11% of patients.

Early *retrospective reports* showed significant improvements in survival with the defibrillator (205,208,231). The study design tended to overestimate benefits by using device therapies (antitachycardia pacing and shocks) as surrogate mortality events. In a large body of subsequent experience, the sudden death rate reported in virtually all series ranges from 1% to 2% per year with a cumulative incidence of ≤10% at 5 years (105,205–207,210,211,213,215–217,232). Higher sudden death rates have been reported in patients with severe LV dysfunction (233,234). Dilution of the survival benefit conferred by sudden death reduction in ICD patients by non-arrhythmic mortality and its impact on overall survival is patient population–dependent (232,233).

Device therapy delivery cannot be used as a surrogate mortality end point because arrhythmias other than VT/VF can activate the device, and recurrent VT is not invariably fatal. Symptomatic ICD activations alone underestimate antiarrhythmic benefits of ICD therapy. More recently, firmer estimates of benefits from ICD therapy using devices with event memory capabilities have become possible in the absence of placebo-controlled studies (225,227,235,236). In these studies ICD patients had successful reversion (>98%) of VT with circulatory collapse or VF, with a significant projected survival benefit compared with untreated populations (235). This benefit is incremental and continues to increase over longer periods (3 to 4 years). A similar benefit exists in patients with sustained VT (236).

There has been controversy about the appropriate end point for evaluation of ICD efficacy. Many studies have used sudden death, but classification of the cause of death is often difficult and imprecise. Consequently, a consensus has emerged that total mortality is the appropriate primary end point for judging ICD efficacy (237). Rates of sudden death and ICD discharges provide useful information, but they should be considered as secondary end points. Total mortality varies significantly between reports due to differences in the disease status of the population under study and LV function. The presence of concomitant cardiac disease is a major determinant of survival (233,238,239). Survival of ICD recipients is influenced by LV function. Patients with LV ejection fractions ≤30% have reduced survival compared with those with higher ejection fractions at 3 years (233,234). However, both populations appear to derive a significant survival benefit (221).

C. Alternatives to ICD Therapy

Pharmacological options for guided antiarrhythmic therapy include drugs in Classes I, II, and III. Therapy can be guided by Holter monitoring or serial electrophysiological testing. High arrhythmia recurrence rates and moderate sudden death rates are observed with Class I agents (240). By contrast, Class III agents are associated with significantly lower arrhythmia recurrences, sudden death, and total mortality (240–243).

β-Blocking agents have also demonstrated efficacy in reducing mortality after AMI (244,245). However, their value in a population of patients with sustained ventricular tachyarrhythmias is not well established. Suppression of inducible VT as well as control of spontaneous VT is often not achieved (246). Although the overall survival of cardiac arrest patients treated empirically with β-blockers and Class I agents may be comparable, patients given Class I agents on serial electrophysiological testing have a better outcome than those treated with empiric β-blocker therapy (247). Current data do not support a significant role for monotherapy with β-blockers in this condition.

In the post–myocardial infarction patient, empiric amiodarone can reduce arrhythmic mortality, but benefit with respect to total mortality in such patients with ventricular dysfunction is less clear (248–251). In cardiac arrest survivors treated empirically with amiodarone, patients with a reduced ejection fraction (<40%) continue to exhibit high arrhythmia recurrence and sudden death rates (252). Similarly, patients with congestive heart failure may show little to no mortality reduction with empiric amiodarone therapy (253,254).

Long-term maintenance of effective antiarrhythmic drug therapy remains problematic. Discontinuation for drug intolerance is high for Class I agents and sotalol at initiation of therapy and during long-term administration (240). Amiodarone therapy is also frequently discontinued for adverse effects on long-term administration (243).

Ablative therapy has been most often used for patients with sustained monomorphic VT induced at cardiac surgery or electrophysiological study and mapped to a specific ventricular site(s). Intraoperative ablation is accomplished mechanically or with physical energy sources (cryothermia or laser), whereas catheter-based energy delivery (direct-current shock, radiofrequency, microwaves, laser) is used during electrophysiological procedures (255–258). These methods are applicable to a select population of patients with malignant ventricular tachyarrhythmias that have reproducibly inducible monomorphic VT suitable for cardiac mapping. Surgical experience is more extensive and favorable in patients with coronary heart disease than noncoronary disease. Perioperative mortality is now lower and averages <5% in more recent experience, particularly when preoperative LV systolic function is preserved. Intraoperative map-guided ablation is associated with low arrhythmia recurrence (<10% at 2 years) and minimal sudden death rates (256–258) during long-term follow-up, making it an important therapeutic alternative in this subgroup.

Catheter ablation approaches are still in technological evolution (259,260). Hemodynamically stable VT is required for mapping, and radiofrequency energy is currently used for ablation (261,262). Procedural complication rates are moderate with modest arrhythmia control (261,262), often in conjunction with previously ineffective drug therapy in patients with coronary artery disease. Higher efficacy rates are observed in patients with right ventricular outflow tract tachycardia, idiopathic left septal VT, and bundle branch reentrant VT in whom ablation may be the preferred therapy (263–265). Multiple VT morphologies, polymorphic VT, and progressive cardiomyopathy, when present, are less amenable to a favorable result with ablative intervention (255,256).

D. Comparison of Drug and Device Therapy for Secondary Prevention of Cardiac Arrest and Sustained Ventricular Tachycardia

A significant body of information now exists comparing these two therapeutic options. Direct comparison of drug and device therapy has been performed in several *retrospective nonrandomized* reports and fewer *prospective randomized* studies. In comparison with concurrently medically treated but nonrandomized populations receiving amiodarone, a significant mortality benefit was noted in the patients with ICDs over the first 3 years of follow-up (206,209). This benefit may dissipate with follow-up beyond 5 years in some reports (209). In similar *nonrandomized* comparisons in sudden death survivors discharged either on electrophysiologically guided antiarrhythmic therapy using Class I or III drugs or on an ICD-based regimen, the survival of the ICD patients was superior, both in patients with early or advanced LV dysfunction (210). In such analyses, the use of an ICD in the treatment regimen was the strongest predictor of long-term survival. ICD recipients also show improved survival in such comparisons with patients receiving guided sotalol therapy (266).

Information from *randomized* trials comparing drug and device therapy also suggests survival benefits with the ICD in this population when compared with electrophysiologically guided drug therapy using Class I agents, propafenone, or sotalol (267,268). A large *prospective randomized* comparative study comparing ICD therapy with Class III antiarrhythmic drug therapy, predominantly empiric amiodarone, has been recently reported (221). In survivors of cardiac arrest and hemodynamically unstable VT, survival was greater with ICD therapy. Unadjusted survival estimates for the ICD and drug therapy were 89.3% versus 82.3%, respectively, at 1 year, 81.6% versus 74.7% at 2 years, and 75.4% versus 64.1% at 3 years. Estimated relative risk reduction with ICD therapy was 39% at 1 year and 31% at 3 years.

Implementation of ICD therapy has been directly compared for safety with antiarrhythmic drug therapy in large systematic trials. *Prospective observational* data demonstrate a low perioperative mortality (0.4% to 1.8%) for primary nonthoracotomy implants (105,216–218). Similar mortality estimates in large prospective antiarrhythmic drug trials range from 3.2% to 13.0% (221,240,243). However, these populations may not be directly comparable. During long-term therapy, drug discontinuation rates have ranged from 7% to 32%, the lowest being with sotalol in reported data (240). In a large *prospective* trial, 98% of randomly assigned patients could be maintained on ICD therapy, with 25.4% requiring the addition of drug therapy by 2 years (221). Withdrawal of device therapy is infrequent and rarely exceeds 2% of implants (216–218). The addition of an antiarrhythmic drug in selected patients with ICDs may improve quality of life by reducing arrhythmia recurrences and the need for shock therapy (266,269).

E. Specific Disease States and Secondary Prevention of Cardiac Arrest or Sustained Ventricular Tachycardia

Prior guidelines do not relate the decision to implant an ICD device to the underlying cardiac disease (270). Recent information suggests that the underlying disease state may have an important impact on patient prognosis and will influence the decision to implant an ICD earlier or later in the treatment algorithm.

Coronary Artery Disease Patients with coronary artery disease represent the majority of patients receiving devices in most reports. Device implantation is widely accepted as improving the outcome of these patients. Patients with reduced LV function may experience greater benefit with ICD therapy than with drug therapy (208,210,267). To limit patient risk during defibrillation efficacy testing (270,271), assessment for the presence of active ischemia should precede implementation of device therapy. Furthermore, optimal anti-ischemic therapy (including, where possible, a β-blocker) will further enhance survival. Measurement of ventricular function is recommended, although poor function is not necessarily a contraindication to device implantation. Abbreviated defibrillation threshold testing, however, may be desirable in patients with elevated pulmonary capillary wedge pressures or severely compromised cardiac output (271).

Idiopathic Dilated Cardiomyopathy Dilated cardiomyopathy is associated with a high mortality within 2 years of diagnosis, with a minority of patients surviving 5 years (272). Approximately one half of these deaths are sudden and unexpected (273). The combination of poor LV function and frequent episodes of nonsustained VT in these patients is associated with an increased risk of sudden death (274). Moreover, unlike in ischemic heart disease, the value of electrophysiological studies is limited (275). The efficacy of drug therapy is low in the presence of impaired LV function and difficult to predict on the basis of invasive or noninvasive testing. ICD implantation may be preferred for treatment of symptomatic VT/VF patients with this condition. In one large prospective study, this population represented 10% of the study group and showed survival benefits with ICD rather than empiric amiodarone therapy similar to the entire study cohort (221).

Long QT Syndrome The long QT syndromes represent a spectrum of electrophysiological disorders characterized by a propensity for development of malignant ventricular arrhythmias, especially polymorphic VT (239,276–278). Because this is a primary electrical disorder, usually with no evidence of structural heart disease or LV dysfunction, the long-term prognosis is excellent if arrhythmia is controlled. Long-term treatment with β-blockers, permanent pacing, or left cervicothoracic sympathectomy is frequently effective (277). ICD implantation is recommended for selected patients in whom recurrent syncope, sustained ventricular arrhythmias, or sudden cardiac death occur despite drug therapy (276). Furthermore, use of the ICD as primary therapy should be considered in certain patients, such as those in whom aborted sudden cardiac death is the initial presentation of the long QT syndrome, where there is a strong family history of sudden cardiac death, or when compliance or intolerance to drugs is a concern (276).

Idiopathic Ventricular Fibrillation It has been estimated that in 10% of young patients resuscitated from cardiac arrest, an etiology of VF is not determined despite extensive evaluation (279,280). Electrophysiological testing in these patients with “idiopathic VF” usually reveals polymorphic VT or VF that is often suppressible with Class IA drugs (279). However, the long-term efficacy of drug therapy remains unknown. Given the guarded prognosis even with effective drug therapy (the annual rate of sudden cardiac death is estimated to be as high as 11%), the limited clinical data available appear to support the use of ICDs in such patients (279–281). Catheter ablation should be considered before ICD insertion in patients with idiopathic right or left VT (263).

Hypertrophic Cardiomyopathy Hypertrophic cardiomyopathy should be suspected and is often identified as the cause of sudden death in young athletes (239,282). Ventricular tachyarrhythmias are a mechanism of sudden death in this condition (283). Sudden death may also be the first manifestation of the disease in a previously asymptomatic individual. Criteria to risk-stratify these patients are not well defined. In contrast with other cardiomyopathies, electrophysiological testing may be of prognostic value because inducible sustained ventricular arrhythmias appear to be associated with cardiac arrest and syncope in some studies (284). Studies of patients resuscitated from cardiac arrest indicate that many patients will experience another event. Pharmacological therapy in the form of β-blockers or calcium channel antagonists has frequently been used, but efficacy in sudden death prevention is not definitively established. Empiric use of amiodarone has been reported to be associated with improved survival (282). However, prediction of drug efficacy remains difficult and controversial. Sudden death survivors should be considered for ICD therapy in preference to or in conjunction with drug therapy (285). Because these patients are often young, drug compliance is frequently an issue. Long-term protection for these patients may be better afforded by treatment with an ICD.

Arrhythmogenic Right Ventricular Dysplasia Arrhythmogenic right ventricular dysplasia can be an important cause of congestive heart failure and ventricular arrhythmias in some patients (286). Drug therapy is often used as primary therapy but is often ineffective. Nonpharmacological options for treatment of significant arrhythmias include catheter ablation of the sites of tachycardia, surgical disarticulation of the right ventricle, and ICDs. In patients with drug-refractory malignant arrhythmias, the ICD provides prophylaxis against syncope due to hemodynamically unstable VT and sudden death (287,288).

Syncope With Inducible Sustained Ventricular Tachycardia Patients with syncope of undetermined etiology in whom clinically relevant VT/VF is induced at electrophysiological study may be candidates for ICD therapy. In these patients, the induced arrhythmia is presumed to be the cause for syncope (289–291). Cardiovascular mortality averages 20% annually, with a large proportion of it sudden. In some patients, antiarrhythmic treatment is limited by inefficacy, intolerance, or noncompliance. ICD therapy is often used with results comparable to sustained VT populations (292). In patients with hemodynamically significant and symptomatic inducible sustained VT, ICD therapy can be a primary treatment option.

F. Pediatric Patients

Pediatric experience with ICDs represents less than 1% of all implantations (239,293). Special considerations such as the need for lifelong pharmacological therapy with its associated problems of noncompliance and side effects make the ICD an important treatment option for young patients.

Sudden cardiac death is uncommon in childhood but is associated with three principal forms of cardiovascular disease: (1) congenital heart disease, (2) cardiomyopathy, and (3) primary electrical disease (239,294). Patients with preexisting heart disease are more likely to experience ventricular tachyarrhythmias as the immediate cause of sudden death compared with those with normal hearts (295). However, a lower percentage of children undergoing resuscitation survive to hospital discharge compared with adults (296).

Indications for ICD therapy for pediatric patients are similar to those for arrhythmias in adults. However, the data used for risk stratification in adults with coronary artery disease may have less positive predictive value in pediatric patients with a variety of underlying diseases (297). Because the risk of unexpected sudden death may be greatest in young patients with diseases such as hypertrophic cardiomyopathy or long QT syndrome, a family history of sudden death may influence the decision to use an ICD in a pediatric patient (277,282).

In patients with congenital heart disease, sudden death has been estimated to occur in 1.5% to 2.5% of patients per decade after repair of tetralogy of Fallot (298). An even higher risk has been identified for patients with transposition of the great arteries and aortic stenosis, with most cases presumed to be due to a malignant ventricular arrhythmia associated with ischemia, ventricular dysfunction, or a rapid response to atrial flutter (120,299). An ischemic substrate for arrhythmias leading to sudden cardiac death also exists in congenital coronary anomalies or after Kawasaki disease.

ICD therapy may be preferable to antiarrhythmic drugs in patients with dilated cardiomyopathy or other causes of impaired ventricular function who experience sustained ventricular arrhythmias because of concern about drug-induced proarrhythmia and myocardial depression. ICDs may also be considered as a bridge to orthotopic heart transplantation in pediatric patients with ventricular arrhythmias whose anomalies are not amenable to surgical correction, particularly given the longer times to donor procurement in younger patients (300). Young patients with hypertrophic cardiomyopathy have a higher annual sudden cardiac death event rate than adults (282,301). A limited experience with ICDs implanted in this population after resuscitation has been encouraging (285, 293,302).

G. Primary Prevention of Sudden Cardiac Death

Coronary Artery Disease Nonsustained VT in patients with prior MI and LV dysfunction is associated with a 2-year mortality estimated at 30% (303,304). Approximately one half of this is believed to be arrhythmic in origin. Antiarrhythmic drug therapy has been widely prescribed in patients after MI with and without ventricular arrhythmia, but evidence of improved survival with this approach is not forthcoming. Increased mortality in coronary disease patients with and without nonsustained VT has actually been noted with specific Class I agents (305). Empiric amiodarone therapy has shown inconsistent survival benefit in large *prospective randomized* trials (250,251), although quantitative overviews (meta-analyses) suggest total mortality may be reduced compared with other medical therapies (241,306). In this population electrophysiological testing has identified a subgroup with inducible sustained ventricular tachyarrhythmias that is at high risk for sudden death (307). While arrhythmia-related symptoms and repeated MIs may help identify such patients, asymptomatic persons post MI may also be at high risk (304,307,308). Survival of patients treated with drugs that suppressed induced arrhythmias improved in comparison with historically untreated or drug-refractory patients (307). In a recent prospective randomized trial, improved survival was documented after implantation of ICDs in patients with inducible and nonsuppressible ventricular tachyarrhythmias when compared with conventional drug therapy, including amiodarone (220).

After Coronary Artery Bypass Surgery Routine ICD insertion does not improve survival in patients with coronary artery disease undergoing bypass surgery who are believed to be at high risk of sudden death based on QRS duration and severe LV dysfunction. In one *randomized* study, no benefit was noted over placebo (309) in patients with ejection fractions <35% and a positive signal-averaged ECG.

As a Bridge to Heart Transplantation Orthotopic heart transplantation has emerged as an acceptable therapeutic alternative for selected patients with congestive heart failure caused by severe ventricular dysfunction. About 20% of patients requiring transplantation die awaiting a donor organ, with a significant incidence of sudden death. ICDs effectively prevent sudden death in these patients (310,311). This benefit is diluted by mortality due to heart failure in some patients (310–312).

Other Populations Other high-risk populations under study for similar benefits include asymptomatic patients with dilated cardiomyopathy and ventricular dysfunction or symptomatic congestive heart failure (313–315), but no recommendations can yet be made with respect to these patients.

H. Contraindications to ICD Therapy

ICD therapy is not recommended for the conditions listed below. The first major group, which can be identified by invasive and noninvasive preimplantation testing, includes those patients in whom a reversible triggering factor for VT/VF can be identified, such as ventricular tachyarrhythmias in evolving AMI or electrolyte abnormalities. Another population is coronary disease patients without inducible or spontaneous VT undergoing routine coronary bypass surgery (309). Similarly, patients with Wolff-Parkinson-White syndrome presenting with VF secondary to atrial fibrillation should undergo catheter or surgical ablation if their accessory pathways are amenable to such treatment.

Patients with terminal illnesses, NYHA Class IV drug-refractory congestive heart failure who are not candidates for cardiac transplantation, or with a life expectancy not exceeding 6 months are likely to obtain limited benefit from ICD therapy. Significant behavioral disorders, including anxiety, device dependence, or social withdrawal have been described (316,317). A history of psychiatric disorders, including uncontrolled depression and substance abuse that interfere with the meticulous care and follow-up needed by these patients, is a relative contraindication to device therapy.

Patients who have frequent tachyarrhythmias that may trigger shock therapy, such as sustained VT not responsive to antitachycardia pacing or pharmacological therapy, are not suitable candidates for a device because these events would cause frequent device activation and multiple shocks. Alternative therapies, such as combining drugs or ablation with ICD insertion, should be considered.

I. Cost-Effectiveness of ICD Therapy

Several studies have addressed the cost-effectiveness of ICD therapy. The cost-effectiveness ratio compares the total cost of ICD therapy with the total cost of an alternative management strategy such as amiodarone or guided serial drug testing. The overall costs of the ICD have been reduced as the result of nonthoracotomy implantation methods and improvements in ICD reliability and longevity that reduce cost of device replacement and modification. Significant reductions in initial costs have been realized, with newer treatment algorithms eliminating prolonged drug testing (318,319).

The early studies of ICD cost-effectiveness were based on mathematical models and relied on nonrandomized studies to estimate clinical efficacy and cost. These studies found cost-effectiveness ratios of $17,000 (320), $18,100 (321), and $29,200 per year of life saved (322). A more recent model incorporated costs of nonthoracotomy ICDs and efficacy estimates based on *randomized* trials and found ICD cost-effectiveness was between $27,300 and $54,000 per life-year gained, corresponding to risk reduction of 40% and 20%, respectively (323).

Several recently completed and ongoing *randomized* clinical trials have measured cost as well as clinical outcomes and thus can directly estimate ICD cost-effectiveness. A preliminary analysis of the MADIT (324) trial found the ICD to have a cost-effectiveness ratio of $27,000 per life-year gained. All studies suggest that ICD implantation in appropriately selected patients has a cost-effectiveness ratio comparable to other cardiovascular therapies as well as widely accepted noncardiac therapies such as renal dialysis ($30,000 to $50,000 per year of life saved). The cost-effectiveness of the ICD is more favorable in patients with high risk of arrhythmic death but low risk of other causes of death. Cost-effectiveness of the ICD would be improved by lowering the cost of the device itself and further improving its reliability and longevity.

J. Selection of ICD Generators

All ICDs currently marketed in the United States incorporate a number of advanced features, including multiple tachycardia zones, with rate detection criteria and tiered therapy (including low-energy cardioversion and high-energy defibrillation shocks) independently programmable for each zone. Furthermore, all devices incorporate programmable ventricular demand pacing and extensive diagnostics, including stored electrograms of rhythms immediately before and after tachycardia detection and therapy. The principal feature distinguishing some ICDs from others is the availability of antitachycardia pacing as a programmable therapy option. The addition of antitachycardia pacing increases the cost of the device by 5% to 10% compared with similar ICDs without this feature. The vast majority of devices used for new implants are small enough for pectoral implants. Larger devices suitable for abdominal implants are available primarily as replacement generators in patients with preexisting lead systems; these larger devices are available at a cost savings of approximately 10% to 25% compared with the smaller devices.

Antitachycardia pacing appears to be a useful feature in the majority of patients receiving ICDs. In one study (325), antitachycardia pacing was activated in 68% of patients receiving ICDs with such a capability, despite the fact that the efficacy of antitachycardia pacing was tested with the device in only 53% of the patients in whom it was activated; in the remainder, antitachycardia pacing algorithms were programmed empirically. In the patients with activated antitachycardia pacing, 96% of all detected episodes of ventricular tachyarrhythmias were terminated with pacing (325). Acceleration of VT by antitachycardia pacing remains a concern, with most series reporting an incidence of antitachycardia pacing acceleration of an episode of VT ranging from 3% to 6% (326). Patients whose only clinical arrhythmia detected before ICD implantation was VF have a lower likelihood of having VT subsequently detected by the ICD than do patients with a prior history of VT (327). However, the incidence of subsequent VT in those with a history of only VF before device implantation is not inconsiderable (18% during 14 months of follow-up in one study [327]), so it is reasonable to select a device with antitachycardia pacing even in such patients.

Defibrillators incorporating an atrial lead have recently become available. Such devices not only provide dual-chamber pacing but also use the pattern of sensed atrial depolarization to distinguish supraventricular from ventricular arrhythmias. A dual-chamber pacemaker–ventricular defibrillator device is an appropriate choice for an ICD candidate who has a concomitant need for dual-chamber pacing or a patient with supraventricular tachycardia thought likely to lead to inappropriate ICD therapies.

K. ICD Follow-up Program

All patients with ICDs require periodic and meticulous follow-up to ensure safety and optimal device performance. The goals of ICD follow-up include monitoring of device system function; optimizing performance for maximal clinical effectiveness and system longevity; minimizing complications; anticipating replacement of system components; ensuring timely intervention for clinical problems; patient tracking, education, and support; and maintenance of ICD system records. The need for device surveillance and management should be discussed *a priori* with patients before insertion of an ICD. Compliance with device follow-up is an important element in evaluating appropriate candidates for device therapy and obtaining the best long-term result. ICD follow-up is best achieved in an organized program analogous to pacemaker follow-up at outpatient clinics (198).

Institutions performing implantation of these devices should also maintain these facilities for inpatient and outpatient use. Such facilities should obtain and maintain implantation and follow-up support devices for all ICDs used at that facility. The facility should be staffed or supported by a fully trained clinical cardiac electrophysiologist (328) who may work in conjunction with trained associated professionals (198,328,329). Access to these services should be available as far as is feasible on both a regularly scheduled and emergent 24-hour-per-day basis. The implantation and/or follow-up facility should be able to locate and track patients who have received ICDs or who have entered the follow-up program.

Elements of ICD Follow-up The follow-up of an ICD patient must be individualized in accordance with the patient's clinical status and conducted by a fully trained clinical cardiac electrophysiologist. In general, device programming is initiated at implantation and should be reviewed at predischarge and/or subsequent postoperative electrophysiological testing. Devices should be followed at 1- to 4-month intervals, depending on the device model and the patient's clinical status. Manufacturers' guidelines for device follow-up vary with individual models and should be available. Transtelephonic follow-up should always be supplemented by clinic visits at a minimum of 3-month intervals for patient and device evaluation (330).

It is often necessary to reprogram the initially selected parameters either in the outpatient clinic or by electrophysiological testing. When device function or concomitant antiarrhythmic therapy is modified, electrophysiological testing can be and often is required to evaluate sensing, pacing, or defibrillation functions of the device. Particular attention should be given to review of sensing parameters, programmed defibrillation and pacing therapies, device activation, and event logs. Technical elements requiring review include battery status, lead system parameters, and elective replacement indicators. Intervening evaluation of device function is often necessary. In general, in patients experiencing device activation, with or without therapy, delivery should be evaluated shortly after the event until a regular acceptable pattern of patient symptomatology and tolerance for such events is established and device behavior is deemed reliable, safe, and effective.

After insertion of a device, its performance should be reviewed, limitations on the patient's specific physical activities established, and registration accomplished. Recent policies on driving advise the patient with an ICD to avoid operating a motor vehicle for a minimum of 3 months and preferably 6 months after the last symptomatic arrhythmic event to determine the pattern of recurrent VT/VF (331,332). Interactions with electromagnetic interference sources, impact on employment, and prophylaxis for device infections should be discussed. ICD recipients should be encouraged to carry proper identification and information about their device at all times. Patients receiving these devices can experience transient or sustained emotional disturbances. Education and psychological support before, during, and after ICD insertion are highly desirable and can improve the patient's quality of life (316,317).

Indications for ICD Therapy

Class I

1. **Cardiac arrest due to VF or VT not due to a transient or reversible cause.** ***(Level of evidence: A)*** **(103–105,202,203, 205–211,216,217,219,221,238,260,267,269)**
2. **Spontaneous sustained VT.** ***(Level of evidence: B)*** **(103–105,202,203,205–211,216,217,219)**
3. **Syncope of undetermined origin with clinically relevant, hemodynamically significant sustained VT or VF induced at electrophysiological study when drug therapy is ineffective, not tolerated, or not preferred.** ***(Level of evidence: B)*** **(204,213,215,219,227,228,266)**
4. **Nonsustained VT with coronary disease, prior MI, LV dysfunction, and inducible VF or sustained VT at electrophysiological study that is not suppressible by a Class I antiarrhythmic drug.** ***(Level of evidence: B)*** **(220,308)**

Class IIa

None.

Class IIb

1. **Cardiac arrest presumed to be due to VF when electrophysiological testing is precluded by other medical conditions.** ***(Level of evidence: C)*** **(211,218,267,276)**
2. **Severe symptoms attributable to sustained ventricular tachyarrhythmias while awaiting cardiac transplantation.** ***(Level of evidence: C)*** **(310,311)**
3. **Familial or inherited conditions with a high risk for life-threatening ventricular tachyarrhythmias such as long QT syndrome or hypertrophic cardiomyopathy.** ***(Level of evidence: B)*** **(8,41,277,282,284,288,300–302)**
4. **Nonsustained VT with coronary artery disease, prior MI, and LV dysfunction, and inducible sustained VT or VF at electrophysiological study.** ***(Level of evidence: B)*** **(103,205,212,217,220,307,308)**
5. **Recurrent syncope of undetermined etiology in the presence of ventricular dysfunction and inducible ventricular arrhythmias at electrophysiological study when other causes of syncope have been excluded.** ***(Level of evidence: C)***

Class III

1. **Syncope of undetermined cause in a patient without inducible ventricular tachyarrhythmias.** ***(Level of evidence: C)***
2. **Incessant VT or VF.** ***(Level of evidence: C)***
3. **VF or VT resulting from arrhythmias amenable to surgical or catheter ablation; for example, atrial arrhythmias associated with the Wolff-Parkinson-White syndrome, right ventricular outflow tract VT, idiopathic left ventricular tachycardia, or fascicular VT.** ***(Level of evidence: C)*** **(259–263)**
4. **Ventricular tachyarrhythmias due to a transient or reversible disorder (eg, AMI, electrolyte imbalance, drugs, trauma).** ***(Level of evidence: C)*** **(333)**
5. **Significant psychiatric illnesses that may be aggravated by device implantation or may preclude systematic follow-up.** ***(Level of evidence: C)*** **(316,317)**
6. **Terminal illnesses with projected life expectancy $\leq$6 months.** ***(Level of evidence: C)***
7. **Patients with coronary artery disease with LV dysfunction and prolonged QRS duration in the absence of spontaneous or inducible sustained or nonsustained VT who are undergoing coronary bypass surgery.** ***(Level of evidence: B)*** **(309)**
8. **NYHA Class IV drug-refractory congestive heart failure in patients who are not candidates for cardiac transplantation.** ***(Level of evidence: C)***

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