I. Introduction

The publication of major studies dealing with the natural history of bradyarrhythmias and tachyarrhythmias and major advances in the technology of pacemakers and implantable cardioverter-defibrillators (ICDs) has mandated this revision of the 1991 ACC/AHA Guidelines for Implantation of Pacemakers and Antiarrhythmia Devices.

This executive summary appears in the April 7, 1998 issue of Circulation. The full text of the guidelines, including the ACC/AHA Class I, II, and III recommendations, is published in the April 1998 issue of the Journal of the American College of Cardiology. Reprints of both the executive summary and the full text are available from both organizations.

Following extensive review of the medical literature and related documents previously published by the American College of Cardiology, the American Heart Association, and the North American Society for Pacing and Electrophysiology, the writing committee developed recommendations that are evidence based whenever possible.

Evidence supporting current recommendations is ranked as level A if the data were derived from multiple randomized clinical trials involving a large number of individuals. Evidence was ranked as level B when data were derived from a limited number of trials involving comparatively small numbers of patients or from well-designed data analysis of nonrandomized studies or observational data registries. Evidence was ranked as level C when consensus of expert opinion was the primary source of recommendation. The committee emphasizes that for certain conditions for which no other therapies are available, the indications for device therapies are based on years of clinical experience as well as expert consensus and are thus well supported, even though the evidence was ranked as level C.

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 implantable device can be obtained only if the device is adjusted to changing clinical conditions.

The text accompanying the list of indications should be read carefully because it includes the rationale and supporting evidence for many indications and in several instances includes a discussion of alternative acceptable therapies. Terms such as “potentially reversible,” “persistent,” “transient,” and “not expected to resolve” are frequently used. These terms are not specifically defined because the time element varies in different clinical settings. The treating physician must use appropriate clinical judgment and available data in deciding whether a condition is persistent or
when it can be expected to be transient. The statement “incidental finding at electrophysiological study” is used several times in this document and does not imply such a study is indicated. Appropriate indications for electrophysiological studies have been previously published.

The term “symptomatic bradycardia” is used frequently throughout the guidelines and is defined as a documented bradyarrhythmia that is directly responsible for the development of 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 heart failure may also result from bradycardia. These symptoms may occur at rest or with exertion. Definite correlation of symptoms with a bradyarrhythmia is a requirement to fulfill the criteria of symptomatic bradycardia. Caution should be exercised not to confuse physiological sinus bradycardia (as occurs in highly trained athletes) with pathological bradyarrhythmias.

The section on indications for ICDs has been extensively revised and enlarged to reflect emerging developments in this field and the voluminous literature attesting to the efficacy of these devices in the treatment of sudden cardiac death and malignant ventricular arrhythmias. Indications for ICDs are constantly changing and can be expected to change further as ongoing large-scale trials are reported. In these guidelines 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.

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

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.

II. Indications for Permanent Pacing

A. Pacing for Acquired Atrioventricular Block in Adults

Patients with abnormalities of atrioventricular (AV) conduction may be asymptomatic or may experience serious symptoms related to bradycardia, ventricular arrhythmias, or both. Decisions about the need for a pacemaker are necessarily influenced by the presence or absence of symptoms that are directly attributable to bradycardia.

Nonrandomized studies strongly suggest that permanent pacing improves survival in patients with third-degree AV block, particularly if syncope has occurred. It is now recognized that marked first-degree AV block can lead to symptoms even in the absence of higher degrees of AV conduction disturbance and may be associated with a “pseudopacemaker syndrome” because of close proximity of atrial systole to the preceding ventricular systole. Small uncontrolled trials have suggested some symptomatic and functional improvement with pacing in patients with PR intervals >0.30 second, especially those with left ventricular (LV) dysfunction, some of whom may benefit from dual-chamber pacing with short AV delay.

Type I second-degree AV block is unlikely to progress to advanced AV block when the delay is within the AV node. Consequently, pacing is not usually indicated in this situation. However, 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.

Physiological AV block in the presence of supraventricular tachyarrhythmias is not an indication for pacemaker implantation except as specifically defined in the recommendations below. Similarly, neurally mediated mechanisms in young patients with AV block should be assessed before proceeding with permanent pacing. Finally, permanent pacing for AV block after valve surgery follows a variable natural history; 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 or any escape rate <40 beats per minute (bpm) in awake, symptom-free patients. (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.
   e. Postoperative AV block that is not expected to resolve. (Level of evidence: C)
   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)

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

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)

3. Asymptomatic type I second-degree AV block at intra- or infra-His levels found incidentally at electrophysiological study for other indications. (Level of evidence: B)

4. First-degree AV block with symptoms suggestive of pacemaker syndrome and documented alleviation of symptoms with temporary AV pacing. (Level of evidence: B)

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)

Class III

1. Asymptomatic first-degree AV block. (Level of evidence: B) (See “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)

3. AV block expected to resolve and unlikely to recur (eg, drug toxicity, Lyme disease). (Level of evidence: B)

B. Pacing for Chronic Bifascicular and Trifascicular Block

Symptomatic advanced AV block that develops in patients with underlying bifascicular and trifascicular block is associated with a high mortality rate and a significant incidence of sudden death. However, there is considerable evidence that the rate of progression of bifascicular block to third-degree AV block is slow. Syncope is common in patients with bifascicular block, and there is evidence that syncope in this setting is associated with an increased incidence of sudden cardiac death. Therefore, if the cause of syncope in the presence of bifascicular or trifascicular block cannot be determined with certainty, prophylactic permanent pacing is indicated.

The PR and HV intervals have been identified as possible predictors of third-degree AV block and sudden death in the presence of underlying bifascicular block. However, the prolongation is often at the level of the AV node, and frequently there is no correlation between the PR and HV intervals and progression to third-degree AV block and incidence of sudden cardiac death. Some investigators have suggested that asymptomatic patients with bifascicular block and a prolonged HV interval (≥100 milliseconds) should be considered for permanent pacing. However, the incidence of progression to third-degree AV block is low, even in the setting of prolonged HV interval. Death is often not sudden or due to advanced AV block but rather due to the underlying heart disease itself and nonarrhythmic cardiac causes.

Indications for Permanent Pacing in Chronic Bifascicular and Trifascicular Block

Class I

1. Intermittent third-degree AV block. (Level of evidence: B)

2. Type II second-degree AV block. (Level of evidence: B)

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)

2. Incidental finding at electrophysiological study of markedly prolonged HV interval (≥100 milliseconds) in asymptomatic patients. (Level of evidence: B)

3. Incidental finding at electrophysiological study of pacing-induced infra-His block that is not physiological. (Level of evidence: B)

Class IIb

None.

Class III

1. Fascicular block without AV block or symptoms. (Level of evidence: B)

2. Fascicular block with first-degree AV block without symptoms. (Level of evidence: B)

C. Pacing for Atrioventricular Block Associated With Acute Myocardial Infarction

The long-term prognosis of survivors of acute myocardial infarction (AMI) who develop AV block is related primarily to the extent of myocardial damage and the character of intraventricular conduction disturbances rather than the AV block itself. Indications for permanent pacing in this setting do not necessarily depend on the presence of symptoms. Patients with AMI who develop 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.

The decision to implant a permanent pacemaker for AV or intraventricular conduction block complicating AMI will depend on the type of conduction disturbance, location of the infarction, and relation of the electrical disturbance to infarct time. Thrombolytic therapy has decreased the incidence of high-grade AV block in AMI, but mortality remains high in this group of patients.

The impact of preexisting bundle branch block on mortality after AMI is uncertain. However, left bundle branch block combined with advanced or third-degree AV block and right bundle branch block combined with left anterior or left posterior fascicular block carry a particularly ominous prognosis.

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. (Level of evidence: B)

*These recommendations generally follow the ACC/AHA Guidelines for the Management of Patients with Acute Myocardial Infarction.
degree AV block within or below the His-Purkinje system after AMI. *(Level of evidence: B)*

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)*

3. Persistent and symptomatic second- or third-degree AV block. *(Level of evidence: B)*

**Class IIa**

None.

**Class IIb**

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

**Class III**

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

2. Transient AV block in the presence of isolated left anterior fascicular block. *(Level of evidence: B)*

3. Acquired left anterior fascicular block in the absence of AV block. *(Level of evidence: B)*

4. Persistent first-degree AV block in the presence of bundle branch block that is old or age indeterminate. *(Level of evidence: B)*

**D. Pacing in Sinus Node Dysfunction**

Correlation of symptoms with arrhythmias resulting from sinus node dysfunction (eg, sinus bradycardia, sinus arrest, paroxysmal supraventricular tachycardia alternating with periods of bradycardia or even asystole) is essential in deciding whether a permanent pacemaker is indicated. This correlation may be difficult because of the intermittent nature of the episodes. Sinus node dysfunction may also express itself as chronotropic incompetence. Rate-responsive pacemakers have clinically benefited patients by restoring physiological heart rate during physical activity in this setting.

Trained athletes may have a physiological sinus bradycardia of 40 to 50 bpm while awake and at rest and a sleeping heart rate as low as 30 bpm with sinus pauses producing asystolic intervals as long as 2.8 seconds. These findings are due to increased vagal tone and are not an indication for permanent pacing.

Temporary pacing in patients with sinus node dysfunction will frequently relieve symptoms but may not necessarily result in improved survival. Whether dual-chamber pacing improves survival compared with ventricular pacing remains controversial. Multiple prospective trials are ongoing to assess the superiority of dual-chamber versus ventricular-based pacing systems in patients with sinus node dysfunction.

**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)*

2. Symptomatic chronotropic incompetence. *(Level of evidence: C)*

**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)*

**Class IIb**

1. In minimally symptomatic patients, chronic heart rate <30 bpm while awake. *(Level of evidence: C)*

**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**

Pacing can be useful in terminating a variety of tachyarrhythmias, including atrial flutter, paroxysmal reentrant supraventricular tachycardia, and VT. A variety of pacing patterns have been used, including programmed stimulation and short bursts of rapid pacing. Antitachyarrhythmia devices may detect the tachycardia and automatically activate a pacing sequence or may respond to an external instruction (eg, application of a magnet). Similarly, prevention of tachyarrhythmias by pacing has been demonstrated in several situations (eg, patients with the long QT syndrome and recurrent pause-dependent VT). Combined therapy of pacing and β-blockade has been reported to shorten the QT interval and help prevent sudden cardiac death. Atrial synchronous ventricular pacing may prevent recurrences of reentrant supraventricular tachycardia, but this technique is rarely used today, given the availability of catheter ablation and other alternative therapies. In some patients with bradyarrhythmia-dependent atrial fibrillation, atrial pacing may also be effective in reducing the frequency of recurrence. Dual-site and biatrial pacing are actively being investigated as therapies for symptomatic drug-refractory atrial fibrillation with concomitant bradyarrhythmias.

**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)*

2. Symptomatic recurrent sustained VT as part of an automatic defibrillator system. *(Level of evidence: B)*

**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)*

**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)*

**Class IIa**

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

**Class IIb**

1. AV reentrant or AV node reentrant supraventricular tachycardia not responsive to medical or ablative therapy. *(Level of evidence: C)*

2. Prevention of symptomatic, drug-refractory, recurrent atrial fibrillation. *(Level of evidence: C)*

**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**

Hypersensitive carotid sinus syndrome is an uncommon cause of syncope or presyncope. Symptoms in this setting are mediated through both cardioinhibitory and vasodepressor reflexes. It is necessary to ascertain the relative contribution of these two components of carotid sinus stimulation before concluding that permanent pacing is clinically indicated. Patients with symptoms due entirely to the cardioinhibitory response of carotid sinus stimulation can be effectively treated with permanent pacing. However, because 10% to 20% of patients also have an important vasodepressor component in their reflex response, this component must be addressed as well.

Ten to forty percent of syncopal episodes are due to a variety of neurally mediated syndromes, the most common being vasovagal syncope. Considerable controversy exists concerning the role of permanent pacing in refractory neurally mediated syncope associated with significant bradycardia or asystole because approximately 25% of patients have a predominant vasodepressor reaction without significant bradycardia. There is conflicting evidence in the literature regarding the efficacy of permanent pacing in neurally mediated syncope, although a recent randomized trial in highly symptomatic patients with bradycardia demonstrated that permanent pacing increased the time to the first syncopal event.

**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)*

**Class IIa**

1. Recurrent syncope without clear, provocative events and with a hypersensitive cardioinhibitory response. *(Level of evidence: C)*

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)*

**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**

Permanent pacing in children or adolescents is generally indicated in (1) symptomatic sinus bradycardia, (2) recurrent bradycardia-tachycardia syndromes, (3) congenital AV block,
and (4) advanced second- or third-degree surgically induced or acquired AV block. Important differences between indications for permanent pacing in children and adults include (1) age dependency of physiological heart rate and (2) impact of residual ventricular dysfunction and abnormal circulatory physiology after surgical palliation of complex congenital cardiac defects.

Symptomatic bradycardia is an indication for pacemaker implantation, provided other causes of symptoms have been excluded. The bradycardia-tachycardia syndrome is an increasingly frequent problem in young patients after surgery for congenital heart disease. Recurrent or chronic atrial flutter is responsible for substantial morbidity and mortality in young patients, and long-term atrial pacing at physiological rates and atrial antitachycardia pacing have been used in this setting. However, the results of permanent pacing to date have been equivocal and the source of considerable controversy.

The indications for permanent pacing in congenital third-degree AV block have evolved with some studies suggesting improved long-term survival and prevention of syncopal episodes in asymptomatic patients with congenital complete heart block who meet specific criteria. High-grade second- or third-degree AV block persisting for 7 to 14 days after cardiac surgery is an indication for permanent pacing. The need for permanent pacing in patients with transient postoperative AV block and residual bifascicular block has not been established, whereas patients with AV conduction that returns to normal have a favorable prognosis.

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)

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)

4. Congenital third-degree AV block with a wide QRS escape rhythm or ventricular dysfunction. (Level of evidence: B)

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)

6. Sustained pause-dependent VT, with or without prolonged QT, in which the efficacy of pacing is thoroughly documented. (Level of evidence: B)

Class IIa

1. Bradycardia-tachycardia syndrome with the need for long-term antiarrhythmic treatment other than digoxin. (Level of evidence: C)

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)

3. Long QT syndrome with 2:1 AV or third-degree AV block. (Level of evidence: B)

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)

2. Congenital third-degree AV block in the asymmetric neonate, child, or adolescent with an acceptable rate, narrow QRS complex, and normal ventricular function. (Level of evidence: B)

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)

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 when the longest RR interval is <3 seconds and the minimum heart rate is ≥40 bpm. (Level of evidence: C)

H. Pacing in Specific Conditions

In patients with severely symptomatic hypertrophic cardiomyopathy, early nonrandomized studies demonstrated that implantation of a dual-chamber pacemaker with a short AV delay decreased the magnitude of LV outflow obstruction and improved symptoms. However, recent observational studies suggest that a decrease in LV outflow gradient produced by a temporary dual chamber may adversely affect ventricular filling and cardiac output. Recent randomized trials have yielded variable results: one study demonstrated that DDD pacing reduced outflow tract gradient and improved New York Heart Association (NYHA) functional class, whereas another randomized double-blind study demonstrated no significant subjective or exercise capacity improvement in the paced versus nonpaced patient group at 2 to 3 months of follow-up, despite a significant decrease in LV outflow gradient. As a result, pacing indications for hypertrophic cardiomyopathy remain controversial.

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. \(\text{Level of evidence: C}\)

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

Several nonrandomized trials of patients with symptomatic dilated cardiomyopathy refractory to medical therapy have reported limited improvement of symptoms with dual-chamber pacing with a short AV delay. However, at this time no long-term data are available, and there is no consensus of opinion for this indication. It has been hypothesized that a well-timed atrial contraction primes the ventricles and decreases mitral regurgitation, thus augmenting stroke volume and arterial pressure. However, one randomized controlled trial showed no significant benefit of DDD pacing through a range of PR intervals despite the presence of both tricuspid and mitral regurgitation. Preliminary data suggest that simultaneous biventricular pacing may improve cardiac hemodynamics and lead to subjective and objective symptom improvement. This technique must still be considered investigational at this time.

Pacing Indications for Dilated Cardiomyopathy

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

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. \(\text{Level of evidence: C}\)

Class III
1. Asymptomatic dilated cardiomyopathy.
2. Symptomatic dilated cardiomyopathy when patients are rendered asymptomatic by drug therapy.

Bradyarrhythmias after cardiac transplantation are common, occurring in 8% to 23% of patients with transplantation and are usually associated with sinus node dysfunction. Although some investigators have recommended more liberal use of cardiac pacing for persistent postoperative bradycardia, approximately 50% of these patients demonstrate significant improvement within 6 to 12 months after transplantation, and therefore long-term pacing is often unnecessary. However, patients with irreversible sinus node dysfunction or AV block with previously stated Class I indications should have permanent pacing.

Pacing Indications After Cardiac Transplantation

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

Class IIa
None.

Class IIb
1. Symptomatic bradyarrhythmias/chronotropic incompetence that, although transient, may persist for months and require intervention. \(\text{Level of evidence: C}\)

Class III
1. Asymptomatic bradyarrhythmias after cardiac transplantation.

I. Selection and Follow-up of Pacemaker Devices

Once a decision has been reached to implant a pacemaker, the clinician may choose from a large number of pacemaker generators and leads. Generator choices include single- versus dual-chamber devices, unipolar versus bipolar configuration, presence of rate responsiveness and type of sensor used, advanced features such as automatic mode switching, generator size, battery capacity, and cost. Lead choices include polarity, type of insulation material, active versus passive fixation mechanism, presence of steroid elution, and typical pacing impedance. Other factors that frequently influence the choice of a pacemaker system include the capabilities of the pacemaker programmer and local availability of technical support. Current single-chamber pacemakers incorporate a number of programming features such as pacing mode, lower rate, pulse width and amplitude, sensitivity, and refractory period. Additional features of current dual-chamber pacemakers include maximum tracking rate and AV delays. 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 individualized for each patient. Many of these considerations are beyond the scope of this document. The Table presents brief guidelines for selecting the appropriate pacemaker for the most commonly encountered indications for pacing. Fig 1 depicts a decision tree for selecting a pacing system for a patient with AV block. Fig 2 depicts a decision tree for selecting a pacing system for a patient with sinus node dysfunction.

It has been suggested that less sophisticated devices, eg, single-chamber ventricular pacemakers or non–rate-responsive pacemakers, should be considered for elderly patients who require pacing. However, a large retrospective analysis of elderly Medicare patients suggested that dual-chamber pacing is associated with improved survival compared with ventricular pacing even after correction for
confounding variables. On the basis of results of recently published randomized and nonrandomized trials, 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. However, there may be no benefit of dual-chamber pacing over rate-responsive ventricular demand pacing.

The cost of a pacemaker increases with its degree of complexity and sophistication. At this time little is known about the cost-effectiveness of the additional features of the more complex pacemakers. Several ongoing trials assessing the clinical benefits of dual-chamber or rate-responsive pacing include economic analysis to estimate the incremental cost-effectiveness of these features. Optimal programming of

Figure 1. Selection of pacemaker systems for patients with atrioventricular block. AV indicates atrioventricular.
output voltages, pulse widths, and AV delays can markedly decrease battery drain and prolong generator life. It has been shown that expert programming of pacemaker generators may prolong their longevity by an average of 4.2 years compared with nominal settings.

After implantation of a pacemaker, careful follow-up and continuity of care are absolute requirements. Programming at implantation must be reviewed before the patient is discharged and further refined at subsequent follow-up visits as indicated by interrogation and testing. 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. Patients who are pacemaker dependent require more frequent clinical evaluations than those who are not. Follow-up evaluation usually includes assessment of battery status, pacing threshold and pulse width, sensing function, and lead integrity. The North American Society of Pacing and Electrophysiology and the Health Care Financing Administration have published reports on antibradycardia pacemaker follow-up and guidelines for monitoring of patients with antibradycardia pacemakers, respectively.

### III. Indications for Implantable Cardioverter-Defibrillator Therapy

Three major therapeutic options are currently available to reduce or prevent VT or ventricular fibrillation (VF) in patients at risk for these arrhythmias: (1) antiarrhythmic drug therapy selected by electrophysiological study or ambulatory monitoring or prescribed empirically; (2) ablative techniques used in cardiac surgery or percutaneously with catheter techniques; and (3) implantation of an ICD. A combination of ICD therapy with drugs or ablation is also frequently used. Both early observational reports and more recent prospective and sometimes randomized single-center and multicenter trials with long-term outcome data uniformly document sudden cardiac death recurrence rates of 1% to 2% annually after device implantation compared with recurrences of 15% to 25% without device therapy. Recent studies have recorded major improvements in implantation risk, system longevity, symptoms associated with arrhythmia recurrences, quality of life, and diagnosis and management of delivery of inappropriate device therapy. Furthermore, the ICD has rapidly evolved from a short-lived nonprogrammable device requiring thoracotomy for a lead insertion into a multiprogrammable antiarrhythmia device inserted almost exclusively without thoracotomy and now capable of treating bradycardia, VT, and VF.

ICDs have been extensively evaluated in prospective clinical trials and clearly documented to revert sustained VTs, including pace termination of sustained VT and shock reversion of VF. Early retrospective reports documented significant improvements in patient survival with the use of an ICD. However, these studies tended to overestimate benefits by using device therapies (antiarrhythmic pacing and shocks) as surrogate mortality events. It has recently become apparent that delivery of device therapy 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. Considerable controversy exists about the appropriate end point for evaluation of ICD efficacy, with many studies using sudden death. However, classification of the cause of

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![Figure 2. Selection of pacemaker systems for patients with sinus node dysfunction. AV indicates atrioventricular.](http://circ.ahajournals.org/)

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death is often difficult and imprecise; therefore, a consensus has emerged that “all-cause” mortality is the appropriate primary end point for judging ICD efficacy. Total mortality has varied significantly between reports due to differences in disease status of the population under study and LV function. Survival of ICD recipients is greatly influenced by LV function. Patients with an LV ejection fraction $\leq 30\%$ have reduced survival rates compared with those with higher ejection fractions. However, both populations appear to derive a significant survival benefit from ICD implantation.

A significant body of information is now available comparing the efficacy of antiarrhythmic drug therapy and ICDs for the secondary prevention of cardiac arrest and sustained VT. Evidence from both early retrospective nonrandomized reports and more recent prospective randomized studies comparing ICD therapy with Class III antiarrhythmic drug therapy indicates a significant relative risk reduction with ICD therapy at 1 and 3 years of follow-up. In a recently reported large prospective trial, 98% of randomly selected patients could be maintained on ICD therapy, with 25.4% requiring the addition of drug therapy by 2 years. Therefore, the addition of an antiarrhythmic drug for selected patients with ICDs may improve their quality of life by reducing recurrence of arrhythmias and the need for defibrillation.

Patients with coronary artery disease constitute the majority of patients receiving devices in most reports. Device implantation is widely accepted today as improving the outcome of these patients. It has been reported that patients with impaired LV function may obtain greater benefit with ICDs than with drug therapy. Optimal anti-ischemic therapy including (when possible) a $\beta$-blocker should be used concomitantly with an ICD. In patients with marked elevation of LV filling pressures, abbreviated defibrillation threshold testing is desirable.

Patients with idiopathic dilated cardiomyopathy have a high mortality rate within 2 years of diagnosis. Approximately half die suddenly and unexpectedly, and it has been shown that the combination of poor LV function and frequent episodes of nonsustained VT is associated with an increased risk of sudden death. In a recently published large prospective trial, patients with idiopathic dilated cardiomyopathy constituted 10% of the study group and showed similar survival benefits with ICD therapy compared with empiric amiodarone therapy as the entire cohort. Similarly, ICD therapy has been shown to confer a significant survival benefit in selected patients with the long QT syndrome, hypertrophic cardiomyopathy, arrhythmogenic right ventricular dysplasia, idiopathic VF, and syncope with inducible sustained VT.

Pediatric patients represent $\leq 1\%$ of persons with ICDs. Nevertheless, ICD therapy is an important treatment option for young patients, given the problems of noncompliance and drug-induced side effects with lifelong pharmacological treatment. Sudden cardiac death is uncommon in childhood and is mainly associated with three forms of heart disease: (1) congenital heart disease, (2) cardiomyopathy, and (3) primary electrical disease. It is noteworthy that a lower percentage of children who undergo resuscitation survive to hospital discharge compared with adults.

Sudden death has been estimated to occur in 1.5% to 2.5% of pediatric patients after repair of tetralogy of Fallot, and the risk is even higher for patients with transposition of the great arteries and aortic stenosis. Most cases are presumed to be due to a malignant ventricular arrhythmia associated either with ischemia, ventricular dysfunction, or rapid ventricular response to atrial flutter. The risk of sudden cardiac death may be greatest in young patients with diseases such as hypertrophic cardiomyopathy or the long QT syndrome. Family history of sudden cardiac death may be an important indication for implantation of an ICD in a pediatric patient with these conditions. Limited experience with ICDs in young patients with hypertrophic cardiomyopathy after resuscitation has been encouraging.

ICD therapy is also used in patients with coronary artery disease for the “primary prevention” of sudden cardiac death: nonsustained VT in patients with prior MI and LV dysfunction carries a 2-year mortality estimate of 30%. Approximately half of this mortality is thought to be due to malignant ventricular arrhythmias. In general, improved patient survival with conventional antiarrhythmic drug therapy has not been shown in this setting. Empiric amiodarone therapy has also shown inconsistent survival benefit, although a recent meta-analysis suggests that total mortality may be reduced when amiodarone is compared with other medical therapies. A recent prospective randomized trial has documented improved survival of patients with inducible and nonsuppressible ventricular tachyarrhythmias treated with ICDs when compared with antiarrhythmic drug therapy, including amiodarone. However, routine insertion of ICDs in patients thought to be at high risk of sudden death who are undergoing aortic coronary bypass graft surgery has not improved survival.

ICDs are not recommended for a number of patients, including those with ventricular tachyarrhythmias in evolving AMI or with electrophysiologically abnormal hearts, those without inducible or spontaneous VT undergoing coronary bypass surgery, and those with preexcitation syndrome presenting with VT as a result of atrial fibrillation. Similarly, patients with terminal illnesses or drug-refractory NYHA Class IV congestive heart failure who are not candidates for cardiac transplantation are likely to obtain limited benefit from ICD therapy. A history of psychiatric disorders, including severe depression and substance abuse, that interfere with the meticulous care and follow-up needed is also a relative contraindication to device therapy.

In appropriately selected patients, ICDs have been found to be cost-effective and comparable to other widely accepted noncardiac therapies such as hemodialysis. A preliminary analysis of a recent randomized clinical trial indicates that in this group of patients, ICDs had a cost-effectiveness ratio of $\leq 27,000$ per life-year gained.

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)

2. Spontaneous sustained VT. (Level of evidence: B)
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)*

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)*

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)*

2. Severe symptoms attributable to sustained ventricular tachyarrhythmias while awaiting cardiac transplantation. *(Level of evidence: C)*

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)*

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)*

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)*

4. Ventricular tachyarrhythmias due to a transient or reversible disorder (eg, AMI, electrolyte imbalance, drugs, trauma). *(Level of evidence: C)*

5. Significant psychiatric illnesses that may be aggravated by device implantation or may preclude systematic follow-up. *(Level of evidence: C)*

6. Terminal illnesses with projected life expectancy ≤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)*

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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