

ACC/AHA/NASPE 2002 Guideline Update for Implantation of Cardiac Pacemakers and Antiarrhythmia Devices: Summary Article

A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (ACC/AHA/NASPE Committee to Update the 1998 Pacemaker Guidelines)

Committee Members

Gabriel Gregoratos, MD, FACC, FAHA, Chair; Jonathan Abrams, MD, FACC, FAHA; Andrew E. Epstein, MD, FACC, FAHA; Roger A. Freedman, MD, FACC; David L. Hayes, MD, FACC, FAHA; Mark A. Hlatky, MD, FACC, FAHA; Richard E. Kerber, MD, FACC, FAHA; Gerald V. Naccarelli, MD, FACC, FAHA; Mark H. Schoenfeld, MD, FACC, FAHA; Michael J. Silka, MD, FACC; Stephen L. Winters, MD, FACC

Task Force Members

Raymond J. Gibbons, MD, FACC, FAHA, Chair; Elliott M. Antman, MD, FACC, FAHA, Vice-Chair; Joseph S. Alpert, MD, FACC, FAHA; Gabriel Gregoratos, MD, FACC, FAHA; Loren F. Hiratzka, MD, FACC, FAHA; David P. Faxon, MD, FACC, FAHA; Alice K. Jacobs, MD, FACC, FAHA; Valentin Fuster, MD, PhD, FACC, FAHA; Sidney C. Smith, Jr, MD, FACC, FAHA

The current update of the ACC/AHA/NASPE Guidelines for Implantation of Cardiac Pacemakers and Antiarrhythmia Devices includes several significant changes in the recommendations and in the supporting narrative portion. In this summary, we list the updated recommendations along with the respective 1998 recommendations, each one accompanied by a brief comment outlining the rationale for the changes, additions, or deletions. All new or revised recommendations are listed in the second column and appear in boldface type. References that support either the 1998 recommendations that have not changed or the new or revised recommendations are noted in parentheses at the end of each recommendation. The reader is referred to the full-text

version of the guidelines posted on the American College of Cardiology (ACC), American Heart Association (AHA), and North American Society for Pacing and Electrophysiology (NASPE) World Wide Web sites for a more detailed exposition of the rationale for these changes. In addition to the recommendation changes listed here, this update includes an expanded section on the selection of pacemakers and implantable cardioverter-defibrillators (ICDs) that reflects the technical advances that have taken place since 1998. A brief expanded summary of pacemaker follow-up procedures is also new to these guidelines. For both of these expanded sections, the reader is referred to the online full-text version.

This document was approved by the American College of Cardiology Foundation Board of Trustees in September 2002, the American Heart Association Science Advisory and Coordinating Committee in August 2002, and the North American Society for Pacing and Electrophysiology in August 2002.

The ACC/AHA Task Force on Practice Guidelines makes every effort to avoid any actual or potential conflicts of interest that might arise as a result of an outside relationship or personal interest of a member of the writing panel. Specifically, all members of the writing panel are asked to provide disclosure statements of all such relationships that might be perceived as real or potential conflicts of interest. These statements are reviewed by the parent task force, reported orally to all members of the writing panel at the first meeting, and updated as changes occur. The conflict of interest information for the writing committee members is posted on the ACC, AHA, and NASPE Web sites with the full-length version of the update.

When citing this document, the ACC, the AHA, and NASPE would appreciate the following citation format: Gregoratos G, Abrams J, Epstein AE, Freedman RA, Hayes DL, Hlatky MA, Kerber RE, Naccarelli GV, Schoenfeld MH, Silka MJ, Winters SL. ACC/AHA/NASPE 2002 Guideline Update for Implantation of Cardiac Pacemakers and Antiarrhythmia Devices: Summary Article: A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (ACC/AHA/NASPE Committee to Update the 1998 Pacemaker Guidelines). *Circulation*. 2002;106:2145-2161.

Copies: This document is available on the World Wide Web sites of the ACC (www.acc.org), the AHA (www.americanheart.org), and NASPE (www.naspe.org). A single copy of the complete guidelines is available by calling 800-253-4636 (US only) or writing the American College of Cardiology Foundation, Resource Center, 9111 Old Georgetown Rd, Bethesda, MD 20814-1699 (ask for No. 71-0237). To obtain a copy of the Summary Article, ask for reprint No. 71-0236. To purchase additional reprints (specify version and reprint number): up to 999 copies, call 800-611-6083 (US only) or fax 413-665-2671; 1000 or more copies, call 410-528-4426, fax 410-528-4264, or e-mail kbradle@lww.com.

(*Circulation* 2002;106:2145-2161.)

©2002 by the American College of Cardiology Foundation and the American Heart Association, Inc.

Circulation is available at <http://www.circulationaha.org>

DOI: 10.1161/01.CIR.0000035996.46455.09

In preparing this update, the committee was guided by the following principles:

- (1) Changes in recommendations and levels of evidence were made either because of new randomized trials or because of the accumulation of new clinical evidence and the development of clinical consensus.
- (2) The committee is cognizant of the healthcare, logistic, and financial implications of recent trials and factored in these considerations in arriving at the class level of certain recommendations.
- (3) Minor wording changes were made to render some recommendations more precise.
- (4) The committee wishes to re-emphasize that the recommendations in the guideline apply to most patients but may require modification by existing situations that only the primary treating physician can evaluate properly.
- (5) All of the listed recommendations for implantation of a device presume the absence of inciting causes that may be eliminated without detriment to the patient (eg, nonessential drug therapy).
- (6) The committee endeavored to maintain consistency of recommendations in this and other previously published guidelines. In the section on atrioventricular (AV) block associated with acute myocardial infarction (AMI), the recommendations follow closely those in the ACC/AHA Guideline for the Management of Patients With Acute Myocardial Infarction.¹ However, given the rapid evolution of pacemaker/ICD science, it has not always been possible to maintain consistency with other guidelines. An example of such a discrepancy can be found in Section I-H, in which the recommendation for biventricular pacing in selected patients with

heart failure has been listed under Class IIa, whereas in the ACC/AHA Guideline for the Evaluation and Management of Chronic Heart Failure in the Adult,² biventricular pacing is cited as an investigational procedure.

The ACC/AHA classifications I, II, and III are used to summarize indications as follows:

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

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

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

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

The weight of the evidence was ranked highest (A) if the data were derived from multiple randomized clinical trials that involved large numbers of patients and intermediate (B) if the data were derived from a limited number of randomized trials that involved small numbers of patients or from careful analyses of nonrandomized studies or observational registries. A lower rank (C) was given when expert consensus was the primary basis for the recommendation.

1998 Recommendation	2002 New or Revised Recommendations	Comments
SECTION I-A: PACING FOR ACQUIRED ATRIOVENTRICULAR BLOCK IN ADULTS		
Recommendations for Permanent Pacing in Acquired Atrioventricular Block in Adults		
Class I	Class I	Class I
<p>1. Third-degree AV block at any anatomic level, associated with any one of the following conditions:</p> <p>a. Bradycardia with symptoms presumed to be due to AV block. (<i>Level of Evidence: C</i>)</p> <p>b. Arrhythmias and other medical conditions that require drugs that result in symptomatic bradycardia. (<i>Level of Evidence: C</i>)</p> <p>c. Documented periods of asystole greater than or equal to 3.0 seconds or any escape rate less than 40 beats per minute (bpm) in awake, symptom-free patients. (<i>Level of Evidence: B, C</i>)</p> <p>d. After catheter ablation of the AV junction. (<i>Level of Evidence: B, C</i>) 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.</p> <p>e. Postoperative AV block that is not expected to resolve. (<i>Level of Evidence: C</i>)</p>	<p>1. Third-degree and advanced second-degree AV block at any anatomic level, associated with any one of the following conditions:</p> <p>a. Bradycardia with symptoms (including heart failure) presumed to be due to AV block. (<i>Level of Evidence: C</i>)</p> <p>b. (No change)</p> <p>c. Documented periods of asystole greater than or equal to 3.0 seconds (3) or any escape rate less than 40 beats per minute (bpm) in awake, symptom-free patients (4,5). (<i>Levels of Evidence: B, C</i>)</p> <p>d. After catheter ablation of the AV junction. (<i>Levels of Evidence: B, C</i>) 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 (6,7).</p> <p>e. Postoperative AV block that is not</p>	<p>The changes emphasize the importance of the site of the block and introduce "advanced second-degree AV block" as a class I indication. This recommendation is based on several observational studies and is supported by a wealth of clinical experience. The narrative portion of this section also emphasizes that the site of origin of the escape rhythm in cases of advanced AV block is as important (or more important) than the escape rate itself.</p> <p>In recommendation 1a, heart failure is specifically introduced as a major symptom that merits consideration when dealing with AV block-induced bradycardia.</p> <p>In recommendation 1e, "cardiac surgery" was added to specifically define the situation(s) in which this recommendation applies.</p> <p>Recommendation 1f has been amplified to indicate that pacing therapy is recommended in patients with neuromuscular diseases and</p>

1998 Recommendation	2002 New or Revised Recommendations	Comments
<p>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)</p> <p>2. Second-degree AV block regardless of type or site of block, with associated symptomatic bradycardia. (Level of Evidence: B) (18)</p>	<p>expected to resolve after cardiac surgery. (Level of Evidence: C) (8–10)</p> <p>f. Neuromuscular diseases with AV block, such as myotonic muscular dystrophy, Kearns-Sayre syndrome, Erb's dystrophy (limb-girdle), and peroneal muscular atrophy, with or without symptoms, because there may be unpredictable progression of AV conduction disease. (Level of Evidence: B) (11–17)</p>	<p>third-degree AV block whether or not they are symptomatic, in view of the unpredictable progression of AV conduction in this group of diseases.</p> <p>No change</p>
<p>Class IIa</p> <p>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)</p> <p>2. Asymptomatic type II second-degree AV block. (Level of Evidence: B)</p> <p>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)</p> <p>4. First-degree AV block with symptoms suggestive of pacemaker syndrome and documented alleviation of symptoms with temporary AV pacing. (Level of Evidence: B)</p>	<p>Class IIa</p> <p>1. Asymptomatic third-degree AV block at any anatomic site with average awake ventricular rates of 40 bpm or faster especially if cardiomegaly or left ventricular (LV) dysfunction is present. (Levels of Evidence: B, C)</p> <p>2. Asymptomatic type II second-degree AV block with a narrow QRS. When type II second-degree AV block occurs with a wide QRS, pacing becomes a Class I recommendation (see next section regarding Pacing for Chronic Bifascicular and Trifascicular Block). (Level of Evidence: B) (19,20)</p> <p>3. Asymptomatic type I second-degree AV block at intra- or infra-His levels found at electrophysiological study performed for other indications. (Level of Evidence: B) (18–21)</p> <p>4. First- or second-degree AV block with symptoms similar to those of pacemaker syndrome. (Level of Evidence: B) (22,23)</p>	<p>Class IIa</p> <p>This change introduces the concept that cardiomegaly and LV dysfunction are important considerations in the decision-making process to implant a pacemaker in asymptomatic patients with third-degree AV block and otherwise "acceptable" heart rates.</p> <p>Based on reports and clinical experience, the change in this recommendation calls attention to the site of the block and emphasizes that a wide QRS complex in patients with type II second-degree AV block suggests the presence of diffuse conduction system disease and constitutes an indication for pacing therapy even in asymptomatic patients.</p> <p>Minor wording change deleting an unnecessary word (incidentally)</p> <p>Wording change to clarify that symptoms resulting from first- or second-degree AV block may be <i>similar</i> to those of the pacemaker syndrome rather than <i>suggestive of</i> this syndrome per se.</p>
<p>Class IIb</p> <p>1. Marked first-degree AV block (more than 0.30 seconds) 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) (24)</p>	<p>Class IIb</p> <p>2. Neuromuscular diseases such as myotonic muscular dystrophy, Kearns-Sayre syndrome, Erb's dystrophy (limb-girdle), and peroneal muscular atrophy with any degree of AV block (including first-degree AV block) with or without symptoms, because there may be unpredictable progression of AV conduction disease. (Level of Evidence: B) (11–17)</p>	<p>Class IIb</p> <p>No change</p> <p>New recommendation for pacemaker insertion in patients with neuromuscular diseases and second- or first-degree AV block, ie, lesser degrees of AV block than those listed under Class I recommendation 1f.</p>

1998 Recommendation	2002 New or Revised Recommendations	Comments
Class III 1. Asymptomatic first-degree AV block. (Level of Evidence: B) (25) (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) (18) 3. AV block expected to resolve and unlikely to recur (26) (eg, drug toxicity, Lyme disease). (Level of Evidence: B)	Class III 3. AV block expected to resolve and/or unlikely to recur (26) (eg, drug toxicity, Lyme disease, or during hypoxia in sleep apnea syndrome in absence of symptoms) (Level of Evidence: B)	Class III No change No change Addition of hypoxia occurring during periods of sleep apnea as a cause of transient AV block that is unlikely to recur once sleep apnea syndrome has been treated.
SECTION I-B: PACING FOR CHRONIC BIFASCICULAR AND TRIFASCICULAR BLOCK		
Recommendations for Permanent Pacing in Chronic Bifascicular and Trifascicular Block		
Class I 1. Intermittent third-degree AV block. (Level of Evidence: B) (27–33) 2. Type II second-degree AV block. (Level of Evidence: B) (34–36)	Class I 3. Alternating bundle-branch block. (Level of Evidence: C) (37)	Class I No change No change New Class I recommendation that adds alternating bundle branch block to the manifestations of fascicular block that indicate pacing therapy. This recommendation was not explicitly stated in the previous version.
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 (greater than or equal to 100 milliseconds) in asymptomatic patients. (Level of Evidence: B) (47) 3. Incidental finding at electrophysiological study of pacing-induced infra-His block that is not physiological. (Level of Evidence: B) (54)	Class IIa 1. Syncope not demonstrated to be due to AV block when other likely causes have been excluded, specifically ventricular tachycardia (VT). (Level of Evidence: B) (38–55)	Class IIa Change of "proved" to "demonstrated" because it may be very difficult to prove the cause of syncope. No change No change
Class IIb	Class IIb 1. Neuromuscular diseases such as myotonic muscular dystrophy, Kearns-Sayre syndrome, Erb's dystrophy (limb-girdle), and peroneal muscular atrophy with any degree of fascicular block with or without symptoms, because there may be unpredictable progression of AV conduction disease. (Level of Evidence: C) (11–17)	Class IIb New Class IIb recommendation for pacing therapy in patients with neuromuscular diseases and fascicular block. Clinical experience suggests that progression of AV conduction disturbance is unpredictable, and high-grade AV block can develop even in asymptomatic patients with these diseases.
Class III 1. Fascicular block without AV block or symptoms. (Level of Evidence: B) (41,43,46,47) 2. Fascicular block with first-degree AV block without symptoms. (Level of Evidence: B) (41,43,46,47)	Class III	Class III No change No change

1998 Recommendation	2002 New or Revised Recommendations	Comments
SECTION I-C: PACING FOR ATRIOVENTRICULAR BLOCK ASSOCIATED WITH ACUTE MYOCARDIAL INFARCTION		
Recommendations for Permanent Pacing After the Acute Phase of Myocardial Infarction		
Class I	Class I	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. (<i>Level of Evidence: B</i>) (36,56–60)		No change
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. (<i>Level of Evidence: B</i>) (56,57)		No change
3. Persistent and symptomatic second- or third-degree AV block. (<i>Level of Evidence: C</i>)		No change
Class IIb	Class IIb	Class IIb
1. Persistent second- or third-degree AV block at the AV node level. (<i>Level of Evidence: B</i>) (20)		No change
Class III	Class III	Class III
1. Transient AV block in the absence of intraventricular conduction defects. (<i>Level of Evidence: B</i>) (56)		No change
2. Transient AV block in the presence of isolated left anterior fascicular block. (<i>Level of Evidence: B</i>) (58)		No change
3. Acquired left anterior fascicular block in the absence of AV block. (<i>Level of Evidence: B</i>) (56)		No change
4. Persistent first-degree AV block in the presence of bundle-branch block that is old or age indeterminate. (<i>Level of Evidence: B</i>) (56)		No change
*These recommendations generally follow the ACC/AHA Guidelines for the Management of Patients With Acute Myocardial Infarction (61)		
SECTION I-D: PACING IN SINUS NODE DYSFUNCTION		
Recommendations for Permanent Pacing in Sinus Node Dysfunction		
Class I	Class I	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. (<i>Level of Evidence: C</i>) (5,62,63)		No change
2. Symptomatic chronotropic incompetence. (<i>Level of Evidence: C</i>) (5,62–65)		No change
Class IIa	Class IIa	Class IIa
1. Sinus node dysfunction occurring spontaneously or as a result of necessary drug therapy, with heart rate less than 40 bpm when a clear association between significant symptoms consistent with bradycardia and the actual presence of bradycardia has not been documented. (<i>Level of Evidence: C</i>) (4,5,62,63,66,67)		No change

1998 Recommendation	2002 New or Revised Recommendations	Comments
Class IIb	2. Syncope of unexplained origin when major abnormalities of sinus node function are discovered or provoked in electrophysiological studies (Level of Evidence: C) (68,69)	New Class IIa recommendation for pacing therapy in patients with syncope, no other demonstrable cause, and who were found to have spontaneous or provokable sinus node dysfunction at electrophysiological study.
1. In minimally symptomatic patients, chronic heart rate less than 30 bpm while awake. (Level of Evidence: C)	Class IIb	Class IIb
	1. In minimally symptomatic patients, chronic heart rate less than 40 bpm while awake. (Level of Evidence: C) (4,5,62,63,66,67)	The change of awake heart rate from 30 to 40 bpm was made on the basis of clinical experience and provides the clinician more flexibility to consider pacing in patients with suspected sinus node dysfunction, in whom a firm diagnosis cannot be made.
Class III	Class III	Class III
1. Sinus node dysfunction in asymptomatic patients, including those in whom substantial sinus bradycardia (heart rate less than 40 bpm) is a consequence of long-term drug treatment.		No change
2. Sinus node dysfunction in patients with symptoms suggestive of bradycardia that are clearly documented as not associated with a slow heart rate.		No change
3. Sinus node dysfunction with symptomatic bradycardia due to nonessential drug therapy.		No change
SECTION I-E: PREVENTION AND TERMINATION OF TACHYARRHYTHMIAS BY PACING		
Recommendations for Permanent Pacemakers That Automatically Detect and Pace to Terminate Tachycardias		
Class I	Class I	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)		This recommendation was downgraded from Class I to Class IIa. Committee consensus was that it is highly unlikely that treatment with drugs and/or ablation therapy would fail to control supraventricular tachycardia (SVT) (see below).
2. Symptomatic recurrent sustained VT as part of an automatic defibrillator system. (Level of Evidence: B)		Deleted because this indication is dealt with in the ICD section.
Class IIa	Class IIa	Class IIa
	1. Symptomatic recurrent SVT that is reproducibly terminated by pacing in the unlikely event that catheter ablation and/or drugs fail to control the arrhythmia or produce intolerable side effects. (Level of Evidence: C) (70–74)	The wording of this previously Class I recommendation is intended to convey that ablation and/or drugs are effective therapies for SVT, and it is unlikely that pacing therapy will be required.
Class IIb	Class IIb	Class IIb
1. Recurrent SVT or atrial flutter that is reproducibly terminated by pacing as an alternative to drug therapy or ablation. (Level of Evidence: C) (70–75)		No change
Class III	Class III	Class III
1. Tachycardias frequently accelerated or converted to fibrillation by pacing.		No change
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.		No change

1998 Recommendation	2002 New or Revised Recommendations	Comments
SECTION I-E: PREVENTION AND TERMINATION OF TACHYARRHYTHMIAS BY PACING		
Pacing Recommendations to Prevent Tachycardia		
Class I 1. Sustained pause-dependent VT, with or without prolonged QT, in which the efficacy of pacing is thoroughly documented. (<i>Level of Evidence: C</i>) (76,77)	Class I	Class I No change
Class IIa 1. High-risk patients with congenital long-QT syndrome. (<i>Level of Evidence: C</i>) (76,77)	Class IIa	Class IIa No change
Class IIb 1. AV re-entrant or AV node re-entrant supraventricular tachycardia not responsive to medical or ablative therapy. (<i>Level of Evidence: C</i>) (71,72,78) 2. Prevention of symptomatic, drug refractory, recurrent atrial fibrillation. (<i>Level of Evidence: C</i>)	Class IIb 2. Prevention of symptomatic, drug-refractory, recurrent atrial fibrillation in patients with coexisting sinus node dysfunction. (<i>Level of Evidence: B</i>) (79–81)	Class IIb No change This recommendation was revised and the level of evidence upgraded to “B” to reflect the available information. Several studies suggest that in some patients with recurrent atrial fibrillation and coexisting sinus node dysfunction, atrial-based pacing reduces the recurrence rate of this arrhythmia.
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.	Class III 2. Torsade de Pointes VT due to reversible causes.	Class III No change Wording change because the arrhythmia is the Torsade de Pointes VT and not the long-QT syndrome.
SECTION I-F: PACING IN HYPERSENSITIVE CAROTID SINUS AND NEUROCARDIOGENIC SYNCOPE		
Recommendations for Permanent Pacing in Hypersensitive Carotid Sinus Syndrome and Neurocardiogenic Syncope		
Class I 1. Recurrent syncope caused by carotid sinus stimulation; minimal carotid sinus pressure induces ventricular asystole of more than 3-second duration in the absence of any medication that depresses the sinus node or AV conduction. (<i>Level of Evidence: C</i>) (82,83)	Class I	Class I No change
Class IIa 1. Recurrent syncope without clear, provocative events and with a hypersensitive cardioinhibitory response. (<i>Level of Evidence: C</i>) (82,83) 2. Syncope of unexplained origin when major abnormalities of sinus node function or AV conduction are discovered or provoked in electrophysiological studies. (<i>Level of Evidence: C</i>)	Class IIa 3. Significantly symptomatic and recurrent neurocardiogenic syncope associated with bradycardia documented spontaneously or at the time of tilt-table testing. (<i>Level of Evidence: B</i>) (84–87)	Class IIa No change Deleted from this section and more appropriately placed in the Sinus Node Dysfunction section as Recommendation #2, Class IIa. This recommendation was added to reflect the results of trials that have demonstrated that pacing therapy is effective in cases of vasovagal syncope associated with episodes of spontaneous or provoked bradycardia. The level of evidence was set to “B” to reflect published trials.

1998 Recommendation	2002 New or Revised Recommendations	Comments
Class IIb 1. Neurally mediated syncope with significant bradycardia reproduced by a head-up tilt with or without isoproterenol or other provocative maneuvers. (<i>Level of Evidence: B</i>)	Class IIb	Class IIb Deleted
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, lightheadedness, or both. 3. Recurrent syncope, lightheadedness, or dizziness in the absence of a hyperactive cardioinhibitory response. (<i>Level of Evidence: C</i>) 4. Situational vasovagal syncope in which avoidance behavior is effective. (<i>Level of Evidence: C</i>)	Class III 1. A hyperactive cardioinhibitory response to carotid sinus stimulation in the absence of symptoms or in the presence of vague symptoms such as dizziness, lightheadedness, or both. (<i>Level of Evidence: C</i>)	Class III This Class III recommendation replaces the prior recommendations #1 and #2 for the sake of simplicity. Deleted This becomes #2. This becomes #3.
SECTION I-G: PACING IN CHILDREN, ADOLESCENTS, AND PATIENTS WITH CONGENITAL HEART DISEASE		
Recommendations for Permanent Pacing in Children, Adolescents, and Patients With Congenital Heart Disease		
Class I 1. Advanced second- or third-degree AV block associated with symptomatic bradycardia, congestive heart failure, or low cardiac output. (<i>Level of Evidence: C</i>) 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. (<i>Level of Evidence: B</i>) (3,5,88) 3. Postoperative advanced second- or third-degree AV block that is not expected to resolve after cardiac surgery. (<i>Level of Evidence: B, C</i>) 4. Congenital third-degree AV block with a wide QRS escape rhythm or ventricular dysfunction. (<i>Level of Evidence: B</i>)	Class I 1. Advanced second- or third-degree AV block associated with symptomatic bradycardia, ventricular dysfunction, or low cardiac output. (<i>Level of Evidence: C</i>) 3. Postoperative advanced second- or third-degree AV block that is not expected to resolve or persists at least 7 days after cardiac surgery. (<i>Level of Evidence: B, C</i>) (89,90) 4. Congenital third-degree AV block with a wide QRS escape rhythm, complex ventricular ectopy, or ventricular dysfunction. (<i>Level of Evidence: B</i>) (91–93)	Class I This recommendation was reworded, substituting "ventricular dysfunction" for "congestive heart failure" to reflect accumulating clinical experience that ventricular dysfunction adversely affects the prognosis of patients with congenital third-degree AV block even in the absence of overt heart failure. No change Reworded recommendation to specify that AV block that persists for more than 7 days after cardiac surgery is unlikely to resolve and is best treated with the implantation of a pacemaker. The change was made because of accumulating clinical experience and published studies demonstrating adverse prognosis in such patients who did not receive a permanent pacemaker for rate support. "Complex ventricular ectopy" was added to the other elements of this recommendation to reflect growing experience that in this setting, prognosis is adversely affected by such ectopy in the absence of rate support by a permanent pacemaker.

1998 Recommendation	2002 New or Revised Recommendations	Comments
5. Congenital third-degree AV block in the infant with a ventricular rate less than 50 to 55 bpm or with congenital heart disease and a ventricular rate less than 70 bpm. (<i>Level of Evidence: B, C</i>) (92,94)		No change
6. Sustained pause-dependent VT, with or without prolonged QT, in which the efficacy of pacing is thoroughly documented. (<i>Level of Evidence: B</i>) (76,77,95,96)		No change
Class IIa	Class IIa	Class IIa
1. Bradycardia-tachycardia syndrome with the need for long-term antiarrhythmic treatment other than digitalis. (<i>Level of Evidence: C</i>) (97,98)		No change
2. Congenital third-degree AV block beyond the first year of life with an average heart rate less than 50 bpm or abrupt pauses in ventricular rate that are two or three times the basic cycle length. (<i>Level of Evidence: B</i>)	2. Congenital third-degree AV block beyond the first year of life with an average heart rate less than 50 bpm, abrupt pauses in ventricular rate that are two or three times the basic cycle length, or associated with symptoms due to chronotropic incompetence. (<i>Level of Evidence: B</i>) (99)	Rewording of this recommendation to include symptoms due to chronotropic incompetence and abrupt pauses in ventricular rate in young patients with third-degree AV block after the first year of life. These events have been found to affect prognosis in patients with asymptomatic congenital third-degree AV block.
3. Long-QT syndrome with 2:1 AV or third-degree AV block. (<i>Level of Evidence: B</i>) (100,101)		No change
4. Asymptomatic sinus bradycardia in the child with complex congenital heart disease with resting heart rate less than 35 bpm or pauses in ventricular rate more than 3 seconds. (<i>Level of Evidence: C</i>)	4. Asymptomatic sinus bradycardia in the child with complex congenital heart disease with resting heart rate less than 40 bpm or pauses in ventricular rate more than 3 seconds. (<i>Level of Evidence: C</i>)	The resting heart rate was changed from 35 to 40 bpm on the basis of clinical experience and expert consensus.
	5. Patients with congenital heart disease and impaired hemodynamics due to sinus bradycardia or loss of AV synchrony. (<i>Level of Evidence: C</i>)	New recommendation for pacing in children with impaired hemodynamics as a result of sinus bradycardia or loss of AV synchrony. Clinical experience has accumulated that indicates that children with congenital heart disease and hemodynamic impairment as a result of these conditions have unfavorable prognosis if not paced.
Class IIb	Class IIb	Class IIb
1. Transient postoperative third-degree AV block that reverts to sinus rhythm with residual bifascicular block. (<i>Level of Evidence: C</i>) (102)		No change
2. Congenital third-degree AV block in the asymptomatic neonate, child, or adolescent with an acceptable rate, narrow QRS complex, and normal ventricular function. (<i>Level of Evidence: B</i>)	2. Congenital third-degree AV block in the asymptomatic infant, child, adolescent, or young adult with an acceptable rate, narrow QRS complex, and normal ventricular function. (<i>Level of Evidence: B</i>) (91,103)	Modification of this recommendation to include "young adults" with congenital third-degree AV block by clinical consensus.
3. Asymptomatic sinus bradycardia in the adolescent with congenital heart disease with resting heart rate less than 35 bpm or pauses in ventricular rate more than 3 seconds. (<i>Level of Evidence: C</i>)	3. Asymptomatic sinus bradycardia in the adolescent with congenital heart disease with resting heart rate less than 40 bpm or pauses in ventricular rate more than 3 seconds. (<i>Level of Evidence: C</i>)	Change of resting heart rate from 35 to 40 bpm as a result of clinical experience and expert consensus.
	4. Neuromuscular diseases with any degree of AV block (including first-degree AV block), with or without symptoms, because there may be unpredictable progression of AV conduction disease.	New Class IIb recommendation for pacing in children and adolescents with a neuromuscular disease and any degree of AV block. This is similar to the recommendation for pacing in this situation for adults (Section I-A).

1998 Recommendation	2002 New or Revised Recommendations	Comments
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 with longest RR interval less than 3 seconds and minimum heart rate more than 40 bpm. (Level of Evidence: C) (104)	Class III 1. Transient postoperative AV block with return of normal AV conduction. (Level of Evidence: B) (90,102)	Class III Rewording of this Class III recommendation to eliminate the 7-day window. There is clinical evidence that patients with postoperative AV block who regain normal AV conduction at any time have generally favorable prognosis without pacing. No change No change No change
SECTION I-H: PACING IN SPECIFIC CONDITIONS		
1. HYPERTROPHIC OBSTRUCTIVE CARDIOMYOPATHY		
Pacing Recommendations for Hypertrophic Cardiomyopathy		
Class I 1. Class I indications for sinus node dysfunction or AV block as previously described. (Level of Evidence: C)	Class I	Class I No change
Class IIb 1. Medically refractory, symptomatic hypertrophic cardiomyopathy with significant resting or provoked LV outflow obstruction. (Level of Evidence: C)	Class IIb 1. Medically refractory, symptomatic hypertrophic cardiomyopathy with significant resting or provoked LV outflow obstruction. (Level of Evidence: A) (105–110)	Class IIb No change in recommendation class. Level of evidence raised from “C” to “A” on the basis of published trials.
Class III 1. Patients who are asymptomatic or medically controlled. 2. Symptomatic patients without evidence of LV outflow obstruction.	Class III	Class III No change No change
SECTION I-H: PACING IN SPECIFIC CONDITIONS (continued)		
2. IDIOPATHIC DILATED CARDIOMYOPATHY		
Pacing Recommendations for Dilated Cardiomyopathy		
Class I 1. Class I indications for sinus node dysfunction or AV block as previously described. (Level of Evidence: C)	Class I	Class I No change
Class IIa	Class IIa 1. Biventricular pacing in medically refractory, symptomatic New York Heart Association (NYHA) class III or IV patients with idiopathic dilated or ischemic cardiomyopathy, prolonged QRS interval (greater than or equal to 130 ms), LV end-diastolic diameter greater than or equal to 55 mm and ejection fraction less than or equal to 35%. (Level of Evidence: A) (111,112)	Class IIa New recommendation for <i>biventricular</i> pacing in patients with advanced heart failure, specific indices of LV dysfunction, and prolonged QRS duration. Multiple trials have demonstrated clinical and structural cardiac improvement with this form of therapy.

1998 Recommendation	2002 New or Revised Recommendations	Comments
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)	Class IIb	Class IIb Deleted
Class III 1. Asymptomatic dilated cardiomyopathy. 2. Symptomatic dilated cardiomyopathy when patients are rendered asymptomatic by drug therapy. 3. Symptomatic ischemic cardiomyopathy.	Class III 3. Symptomatic ischemic cardiomyopathy when the ischemia is amenable to intervention.	Class III No change No change Modification of this recommendation to clarify that pacing therapy is not indicated in symptomatic ischemic cardiomyopathy when the patient can be treated with revascularization therapy.

SECTION I-H: PACING IN SPECIFIC CONDITIONS (continued)**3. CARDIAC TRANSPLANTATION****Pacing Recommendations 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 I	Class I No change
Class IIb 1. Symptomatic bradyarrhythmias/chronotropic incompetence that, although transient, may persist for months and require intervention. (Level of Evidence: C)	Class IIb	Class IIb No change
Class III 1. Asymptomatic bradyarrhythmias after cardiac transplantation.	Class III	Class III No change

SECTION II: INDICATIONS FOR IMPLANTABLE CARDIOVERTER-DEFIBRILLATOR THERAPY**Recommendations for ICD Therapy**

Class I 1. Cardiac arrest due to ventricular fibrillation (VF) or VT not due to a transient or reversible cause. (Level of Evidence: A) (113–134) 2. Spontaneous sustained VT. (Level of Evidence: B)	Class I 2. Spontaneous sustained VT in association with structural heart disease. (Level of Evidence: B) (113–127)	Class I No change This recommendation for ICD implantation was modified with the addition of the requirement for structural heart disease to be present. This change was made because ICD therapy is most efficacious in patients with impaired LV performance. Conversely, VT arising in structurally normal hearts can usually be treated pharmacologically or with catheter ablation.
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) (127,133,135–140)		No change

1998 Recommendation	2002 New or Revised Recommendations	Comments
<p>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. (<i>Level of Evidence: B</i>)</p>	<p>4. Nonsustained VT in patients with coronary disease, prior myocardial infarction (MI), LV dysfunction, and inducible VF or sustained VT at electrophysiological study that is not suppressible by a Class I antiarrhythmic drug. (<i>Level of Evidence: A</i>) (141–143)</p>	<p>No substantive change. Level of evidence raised from “B” to “A” as a result of newly published studies.</p>
Class IIa	<p>5. Spontaneous sustained VT in patients who do not have structural heart disease that is not amenable to other treatments. (<i>Level of Evidence: C</i>)</p>	<p>New recommendation for ICD implantation in patients with sustained VT and structurally normal hearts when alternative treatments have failed (See #2 above)</p>
Class IIa	<p>Class IIa</p> <p>Patients with LV ejection fraction of less than or equal to 30%, at least one month post myocardial infarction and three months post coronary artery revascularization surgery. (<i>Level of Evidence: B</i>) (159)</p>	<p>Class IIa</p> <p>New recommendation for implantation of an ICD prophylactically in the defined population. This recommendation is promulgated as a result of a randomized trial that demonstrated a 5.6% absolute risk reduction and a 31% relative risk reduction for death in the patient group receiving an ICD. The committee consensus was that further risk stratification of the referenced population might better define the benefit of an ICD in such patients. The reader should review the discussion regarding this recommendation in the full-text guideline on the ACC, AHA, and NASPE web sites.</p>
Class IIb	Class IIb	Class IIb
<p>1. Cardiac arrest presumed to be due to VF when electrophysiological testing is precluded by other medical conditions. (<i>Level of Evidence: C</i>) (124,131,144,145)</p>	<p>2. Severe symptoms (eg, syncope) attributable to ventricular tachyarrhythmias in patients awaiting cardiac transplantation. (<i>Level of Evidence: C</i>) (146,147)</p>	<p>No substantive change. Syncope was added as an example of “severe symptoms.”</p>
<p>2. Severe symptoms attributable to sustained ventricular tachyarrhythmias while awaiting cardiac transplantation. (<i>Level of Evidence: C</i>)</p>	<p>No change</p>	<p>No change</p>
<p>3. Familial or inherited conditions with a high risk for life-threatening ventricular tachyarrhythmias such as long-QT syndrome or hypertrophic cardiomyopathy. (<i>Level of Evidence: B</i>) (27,39,148–154)</p>	<p>No change</p>	<p>No change</p>
<p>4. Nonsustained VT with coronary artery disease, prior MI, LV dysfunction, and inducible sustained VT or VF at electrophysiological study. (<i>Level of Evidence: B</i>) (113,118,126,141,142,155,156)</p>	<p>6. Syncope of unexplained etiology or family history of unexplained sudden cardiac death in association with typical or atypical right bundle-branch block and ST-segment elevations (Brugada syndrome). (<i>Level of Evidence: C</i>) (157,158)</p>	<p>New recommendation for ICD implantation in patients with the Brugada syndrome and syncope or family history of sudden cardiac death. Several reports suggest that ICD therapy in patients with this syndrome is effective in preventing sudden death.</p>
<p>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. (<i>Level of Evidence: C</i>)</p>	<p>No change</p>	<p>No change</p>

1998 Recommendation	2002 New or Revised Recommendations	Comments
	7. Syncope in patients with advanced structural heart disease in which thorough invasive and noninvasive investigation has failed to define a cause. (Level of Evidence: C)	New recommendation based on clinical experience and expert consensus. Patients with advanced structural heart disease and syncope of undetermined etiology despite thorough investigation are likely to have an arrhythmic cause of the syncope and thus may benefit from ICD insertion.
Class III	Class III	Class III
1. Syncope of undetermined cause in a patient without inducible ventricular tachyarrhythmias. (Level of Evidence: C)	1. Syncope of undetermined cause in a patient without inducible ventricular tachyarrhythmias and without structural heart disease. (Level of Evidence: C)	Modification of this recommendation to exclude patients with structural heart disease who fall under #7, Class IIb, above.
2. Incessant VT or VF. (Level of Evidence: C)		No change
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) (130,160–163)		No change
4. Ventricular tachyarrhythmias due to a transient or reversible disorder (eg, AMI, electrolyte imbalance, drugs, trauma). (Level of Evidence: C) (164)	4. Ventricular tachyarrhythmias due to a transient or reversible disorder (eg, AMI, electrolyte imbalance, drugs, or trauma) when correction of the disorder is considered feasible and likely to substantially reduce the risk of recurrent arrhythmia. (Level of Evidence: B) (165–167)	Changed to address the issue of many patients with structural heart disease who experience cardiac arrest in the setting of abnormal electrolytes. Such patients may still be at risk for recurrent arrhythmic events and may still benefit from ICD therapy.
5. Significant psychiatric illnesses that may be aggravated by device implantation or may preclude systematic follow-up. (Level of Evidence: C) (168,169)		No change
6. Terminal illnesses with projected life expectancy less than six months. (Level of Evidence: C)		No change
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) (170)		No change
8. NYHA Class IV drug-refractory congestive heart failure in patients who are not candidates for cardiac transplantation. (Level of Evidence: C)		No change

References

- Ryan TJ, Antman EM, Brooks NH, et al. 1999 update: ACC/AHA guidelines for the management of patients with acute myocardial infarction: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Management of Acute Myocardial Infarction). *J Am Coll Cardiol*. 1999;34:890–911.
- Hunt SA, Baker DW, Chin MH, et al. ACC/AHA guidelines for the evaluation and management of chronic heart failure in the adult: executive summary. a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Revise the 1995 Guidelines for the Evaluation and Management of Heart Failure) developed in collaboration with the International Society for Heart and Lung Transplantation endorsed by the Heart Failure Society of America. *J Am Coll Cardiol*. 2001;38:2101–2113.
- Ector H, Rolies L, De Geest H. Dynamic electrocardiography and ventricular pauses of 3 seconds and more: etiology and therapeutic implications. *Pacing Clin Electrophysiol*. 1983;6:548–551.
- Shaw DB, Holman RR, Gowers JI. Survival in sinoatrial disorder (sick-sinus syndrome). *BMJ*. 1980;280:139–141.
- Kay R, Estioko M, Wiener I. Primary sick sinus syndrome as an indication for chronic pacemaker therapy in young adults: incidence, clinical features, and long-term evaluation. *Am Heart J*. 1982;103:338–342.
- Gallagher JJ, Svenson RH, Kasell JH, et al. Catheter technique for closed-chest ablation of the atrioventricular conduction system. *N Engl J Med*. 1982;306:194–200.
- Langberg JJ, Chin MC, Rosenqvist M, et al. Catheter ablation of the atrioventricular junction with radiofrequency energy. *Circulation*. 1989;80:1527–1535.
- Kastor JA. Atrioventricular block (first of two parts). *N Engl J Med*. 1975;292:462–465.
- Glikson M, Dearani JA, Hyberger LK, Schaff HV, Hammill SC, Hayes DL. Indications, effectiveness, and long-term dependency in permanent pacing after cardiac surgery. *Am J Cardiol*. 1997;80:1309–1313.
- Kim MH, Deeb GM, Eagle KA, et al. Complete atrioventricular block after valvular heart surgery and the timing of pacemaker implantation. *Am J Cardiol*. 2001;87:649–651, A10.

11. Perloff JK, Stevenson WG, Roberts NK, Cabeen W, Weiss J. Cardiac involvement in myotonic muscular dystrophy (Steinert's disease): a prospective study of 25 patients. *Am J Cardiol*. 1984;54:1074-1081.
12. Hiromasa S, Ikeda T, Kubota K, et al. Myotonic dystrophy: ambulatory electrocardiogram, electrophysiologic study, and echocardiographic evaluation. *Am Heart J*. 1987;113:1482-1488.
13. Stevenson WG, Perloff JK, Weiss JN, Anderson TL. Facioscapulo-humeral muscular dystrophy: evidence for selective, genetic electrophysiologic cardiac involvement. *J Am Coll Cardiol*. 1990;15:292-299.
14. James TN, Fisch C. Observations on the cardiovascular involvement in Friedreich's ataxia. *Am Heart J*. 1963;66:164-175.
15. Roberts NK, Perloff JK, Kark RA. Cardiac conduction in the Kearns-Sayre syndrome (a neuromuscular disorder associated with progressive external ophthalmoplegia and pigmentary retinopathy): report of 2 cases and review of 17 published cases. *Am J Cardiol*. 1979;44:1396-1400.
16. Charles R, Holt S, Kay JM, Epstein EJ, Rees JR. Myocardial ultrastructure and the development of atrioventricular block in Kearns-Sayre syndrome. *Circulation*. 1981;63:214-219.
17. James TN. Observations on the cardiovascular involvement, including the conduction system, in progressive muscular dystrophy. *Am Heart J*. 1962;63:48-56.
18. Strasberg B, Amat YL, Dhingra R, et al. Natural history of chronic second-degree atrioventricular nodal block. *Circulation*. 1981;63:1043-1049.
19. Recommendations for pacemaker prescription for symptomatic bradycardia: report of a working party of the British Pacing and Electrophysiology Group. *Br Heart J*. 1991;66:185-191.
20. Shaw DB, Kekwick CA, Veale D, Gowers J, Whistance T. Survival in second degree atrioventricular block. *Br Heart J*. 1985;53:587-593.
21. Connelly DT, Steinhaus DM. Mobitz type I atrioventricular block: an indication for permanent pacing? *Pacing Clin Electrophysiol*. 1996;19:261-264.
22. Barold SS. Indications for permanent cardiac pacing in first-degree AV block: class I, II, or III? *Pacing Clin Electrophysiol*. 1996;19:747-751.
23. Kim YH, O'Nunain S, Trouto T, et al. Pseudo-pacemaker syndrome following inadvertent fast pathway ablation for atrioventricular nodal reentrant tachycardia. *J Cardiovasc Electrophysiol*. 1993;4:178-182.
24. Brecker SJ, Xiao HB, Sparrow J, Gibson DG. Effects of dual-chamber pacing with short atrioventricular delay in dilated cardiomyopathy. *Lancet*. 1992;340:1308-1312.
25. Mymin D, Mathewson FA, Tate RB, Manfreda J. The natural history of primary first-degree atrioventricular heart block. *N Engl J Med*. 1986;315:1183-1187.
26. McAlister HF, Klementowicz PT, Andrews C, Fisher JD, Feld M, Furman S. Lyme carditis: an important cause of reversible heart block. *Ann Intern Med*. 1989;110:339-345.
27. Freidberg CK, Donoso E, Stein WG. Nonsurgical acquired heart block. *Ann N Y Acad Sci*. 1964;111:835-847.
28. Gadboys HL, Wisoff BG, Litwak RS. Surgical treatment of complete heart block: an analysis of 36 cases. *JAMA*. 1964;189:97-102.
29. Johansson BW. Complete heart block: a clinical, hemodynamic and pharmacological study in patients with and without an artificial pacemaker. *Acta Med Scand Suppl*. 1966;451:1-127.
30. Hindman MC, Wagner GS, JaRo M, et al. The clinical significance of bundle branch block complicating acute myocardial infarction, 2: indications for temporary and permanent pacemaker insertion. *Circulation*. 1978;58:689-699.
31. Donmoyer TL, DeSanctis RW, Austen WG. Experience with implantable pacemakers using myocardial electrodes in the management of heart block. *Ann Thorac Surg*. 1967;3:218-227.
32. Edhag O, Swahn A. Prognosis of patients with complete heart block or arrhythmic syncope who were not treated with artificial pacemakers: a long-term follow-up study of 101 patients. *Acta Med Scand*. 1976;200:457-463.
33. Penton GB, Miller H, Levine SA. Some clinical features of complete heart block. *Circulation*. 1956;13:801-824.
34. Dhingra RC, Denes P, Wu D, Chuquimia R, Rosen KM. The significance of second degree atrioventricular block and bundle branch block: observations regarding site and type of block. *Circulation*. 1974;49:638-646.
35. Donoso E, Adler LN, Friedberg CK. Unusual forms of second-degree atrioventricular block, including Mobitz type-II block, associated with the Morgagni-Adams-Stokes syndrome. *Am Heart J*. 1964;67:150-157.
36. Ranganathan N, Dhurandhar R, Phillips JH, Wigle ED. His Bundle electrogram in bundle-branch block. *Circulation*. 1972;45:282-294.
37. Josephson ME. Clinical cardiac electrophysiology: techniques and interpretations. 2nd ed. Philadelphia: Lea & Febiger, 1993:145.
38. Kulbertus H, Collignon P. Association of right bundle-branch block with left superior or inferior intraventricular block: its relation to complete heart block and Adams-Stokes syndrome. *Br Heart J*. 1969;31:435-440.
39. DePasquale NP, Bruno MS. Natural history of combined right bundle branch block and left anterior hemiblock (bilateral bundle branch block). *Am J Med*. 1973;54:297-303.
40. Dhingra RC, Denes P, Wu D, et al. Syncope in patients with chronic bifascicular block: significance, causative mechanisms, and clinical implications. *Ann Intern Med*. 1974;81:302-306.
41. Scheinman MM, Peters RW, Modin G, Brennan M, Mies C, O'Young J. Prognostic value of infranodal conduction time in patients with chronic bundle branch block. *Circulation*. 1977;56:240-244.
42. Denes P, Dhingra RC, Wu D, Wyndham CR, Leon F, Rosen KM. Sudden death in patients with chronic bifascicular block. *Arch Intern Med*. 1977;137:1005-1010.
43. McAnulty JH, Kauffman S, Murphy E, Kassebaum DG, Rahimtoola SH. Survival in patients with intraventricular conduction defects. *Arch Intern Med*. 1978;138:30-35.
44. Peters RW, Scheinman MM, Modin C, O'Young J, Somelofski CA, Mies C. Prophylactic permanent pacemakers for patients with chronic bundle branch block. *Am J Med*. 1979;66:978-985.
45. Fisch GR, Zipes DP, Fisch C. Bundle branch block and sudden death. *Prog Cardiovasc Dis*. 1980;23:187-224.
46. McAnulty JH, Rahimtoola SH, Murphy, et al. Natural history of "high-risk" bundle-branch block: final report of a prospective study. *N Engl J Med*. 1982;307:137-143.
47. Scheinman MM, Peters RW, Suave MJ, et al. Value of the H-Q interval in patients with bundle branch block and the role of prophylactic permanent pacing. *Am J Cardiol*. 1982;50:1316-1322.
48. Morady F, Higgins J, Peters RW, et al. Electrophysiologic testing in bundle branch block and unexplained syncope. *Am J Cardiol*. 1984;54:587-591.
49. Click RL, Gersh BJ, Sugrue DD, et al. Role of invasive electrophysiologic testing in patients with symptomatic bundle branch block. *Am J Cardiol*. 1987;59:817-823.
50. Ezri M, Lerman BB, Marchlinski FE, Buxton AE, Josephson ME. Electrophysiologic evaluation of syncope in patients with bifascicular block. *Am Heart J*. 1983;106:693-697.
51. Twidale N, Heddle WF, Ayres BF, Tonkin AM. Clinical implications of electrophysiology study findings in patients with chronic bifascicular block and syncope. *Aust N Z J Med*. 1988;18:841-847.
52. Englund A, Bergfeldt L, Rehnqvist N, Astrom H, Rosenqvist M. Diagnostic value of programmed ventricular stimulation in patients with bifascicular block: a prospective study of patients with and without syncope. *J Am Coll Cardiol*. 1995;26:1508-1515.
53. Probst P, Pachinger O, Akbar MA, Leisch F, Kaindl F. The HQ time in congestive cardiomyopathies. *Am Heart J*. 1979;97:436-441.
54. Dhingra RC, Wyndham C, Bauernfeind R, et al. Significance of block distal to the His bundle induced by atrial pacing in patients with chronic bifascicular block. *Circulation*. 1979;60:1455-1464.
55. Cheng TO. Atrial pacing: its diagnostic and therapeutic applications. *Prog Cardiovasc Dis*. 1971;14:230-247.
56. Col JJ, Weinberg SL. The incidence and mortality of intraventricular conduction defects in acute myocardial infarction. *Am J Cardiol*. 1972;29:344-350.
57. Ritter WS, Atkins JM, Blomqvist CG, Mullins CB. Permanent pacing in patients with transient trifascicular block during acute myocardial infarction. *Am J Cardiol*. 1976;38:205-208.
58. Ginks WR, Sutton R, Oh W, Leatham A. Long-term prognosis after acute anterior infarction with atrioventricular block. *Br Heart J*. 1977;39:186-189.
59. Domenighetti G, Perret C. Intraventricular conduction disturbances in acute myocardial infarction: short- and long-term prognosis. *Eur J Cardiol*. 1980;11:51-59.
60. Lamas GA, Muller JE, Turi ZG, et al. A simplified method to predict occurrence of complete heart block during acute myocardial infarction. *Am J Cardiol*. 1986;57:1213-1219.
61. Ryan TJ, Anderson JL, Antman EM, et al. ACC/AHA guidelines for the management of patients with acute myocardial infarction: a report of the American College of Cardiology/American Heart Association Task

- Force on Practice Guidelines (Committee on Management of Acute Myocardial Infarction). *J Am Coll Cardiol*. 1996;28:1328–1428.
62. Kusumoto FM, Goldschlager N. Cardiac pacing. *N Engl J Med*. 1996;334:89–97.
 63. Rasmussen K. Chronic sinus node disease: natural course and indications for pacing. *Eur Heart J*. 1981;2:455–459.
 64. Linde-Edelstam C, Nordlander R, Pehrsson SK, Ryden L. A double-blind study of submaximal exercise tolerance and variation in paced rate in atrial synchronous compared to activity sensor modulated ventricular pacing. *Pacing Clin Electrophysiol*. 1992;15:905–915.
 65. Gammage M, Schofield S, Rankin I, Bennett M, Coles P, Pentecost B. Benefit of single setting rate responsive ventricular pacing compared with fixed rate demand pacing in elderly patients. *Pacing Clin Electrophysiol*. 1991;14:174–180.
 66. Dreifus LS, Michelson EL, Kaplinsky E, Bradyarrhythmias. clinical significance and management. *J Am Coll Cardiol*. 1983;1:327–338.
 67. Rubenstein JJ, Schulman CL, Yurchak PM, DeSanctis RW. Clinical spectrum of the sick sinus syndrome. *Circulation*. 1972;46:5–13.
 68. Fisher JD. Role of electrophysiologic testing in the diagnosis and treatment of patients with known and suspected bradycardias and tachycardias. *Prog Cardiovasc Dis*. 1981;24:25–90.
 69. Reiffel JA, Kuehnert MJ. Electrophysiological testing of sinus node function: diagnostic and prognostic application-including updated information from sinus node electrograms. *Pacing Clin Electrophysiol*. 1994;17:349–365.
 70. Peters RW, Scheinman MM, Morady F, Jacobson L. Long-term management of recurrent paroxysmal tachycardia by cardiac burst pacing. *Pacing Clin Electrophysiol*. 1985;8:35–44.
 71. Fisher JD, Johnston DR, Furman S, Mercado AD, Kim SG. Long-term efficacy of antitachycardia pacing for supraventricular and ventricular tachycardias. *Am J Cardiol*. 1987;60:1311–1316.
 72. den Dulk K, Bertholet M, Brugada P, et al. Clinical experience with implantable devices for control of tachyarrhythmias. *Pacing Clin Electrophysiol*. 1984;7:548–556.
 73. Saksena S, Pantopoulos D, Parsonnet V, Rothbart ST, Hussain SM, Gielchinsky I. Usefulness of an implantable antitachycardia pacemaker system for supraventricular or ventricular tachycardia. *Am J Cardiol*. 1986;58:70–74.
 74. Barold SS, Wyndham CR, Kappenberger LL, Abinader EG, Griffin JC, Falkoff MD. Implanted atrial pacemakers for paroxysmal atrial flutter: long-term efficacy. *Ann Intern Med*. 1987;107:144–149.
 75. Spurrell RA, Nathan AW, Camm AJ. Clinical experience with implantable scanning tachycardia reversion pacemakers. *Pacing Clin Electrophysiol*. 1984;7:1296–300.
 76. Eldar M, Griffin JC, Abbott JA, et al. Permanent cardiac pacing in patients with the long QT syndrome. *J Am Coll Cardiol*. 1987;10:600–607.
 77. Eldar M, Griffin JC, Van Hare GF, et al. Combined use of beta-adrenergic blocking agents and long-term cardiac pacing for patients with the long QT syndrome. *J Am Coll Cardiol*. 1992;20:830–837.
 78. Attuel P, Pellerin D, Mugica J, Coumel P. DDD pacing: an effective treatment modality for recurrent atrial arrhythmias. *Pacing Clin Electrophysiol*. 1988;11:1647–1654.
 79. Saksena S, Prakash A, Hill M, et al. Prevention of recurrent atrial fibrillation with chronic dual-site right atrial pacing. *J Am Coll Cardiol*. 1996;28:687–694.
 80. Saksena S, Delfaut P, Prakash A, Kaushik RR, Krol RB. Multisite electrode pacing for prevention of atrial fibrillation. *J Cardiovasc Electrophysiol*. 1998;9:S155–S162.
 81. Lamas GA, Lee KL, Sweeney MO, et al. Ventricular pacing or dual-chamber pacing for sinus-node dysfunction. *N Engl J Med*. 2002;346:1854–1862.
 82. Peretz DI, Gerein AN, Miyagishima RT. Permanent demand pacing for hypersensitive carotid sinus syndrome. *Can Med Assoc J*. 1973;108:1131–1134.
 83. Brignole M, Menozzi C, Gianfranchi L, Oddone D, Lolli G, Bertulla A. Neurally mediated syncope detected by carotid sinus massage and head-up tilt test in sick sinus syndrome. *Am J Cardiol*. 1991;68:1032–1036.
 84. Sutton R, Brignole M, Menozzi C, et al, for the Vasovagal Syncope International Study (VASIS) Investigators. Dual-chamber pacing in the treatment of neurally mediated tilt-positive cardioinhibitory syncope: pacemaker versus no therapy: a multicenter randomized study. *Circulation*. 2000;102:294–299.
 85. Connolly SJ, Sheldon R, Roberts RS, Gent M. The North American Vasovagal Pacemaker Study (VPS): a randomized trial of permanent cardiac pacing for the prevention of vasovagal syncope. *J Am Coll Cardiol*. 1999;33:16–20.
 86. Sheldon R, Koshman ML, Wilson W, Kieser T, Rose S. Effect of dual-chamber pacing with automatic rate-drop sensing on recurrent neurally mediated syncope. *Am J Cardiol*. 1998;81:158–162.
 87. Ammirati F, Colivicchi F, Santini M. Permanent cardiac pacing versus medical treatment for the prevention of recurrent vasovagal syncope: a multicenter, randomized, controlled trial. *Circulation*. 2001;104:52–57.
 88. Mackintosh AF. Sinusatrial disease in young people. *Br Heart J*. 1981;45:62–66.
 89. Lillehei CW, Sellers RD, Bonnanbeau RC, Eliot RS. Chronic post-surgical complete heart block with particular reference to prognosis, management, and a new P-wave pacemaker. *J Thorac Cardiovasc Surg*. 1963;46:436–456.
 90. Kertesz N, McQuinn T, Collins E, Friedman R. Surgical Atrioventricular block in 888 congenital heart operations: new implications for early implantation of a permanent pacemaker [abstract]. *PACE*. 1996;19:613.
 91. Michaelsson M, Jonzon A, Riesenfeld T. Isolated congenital complete atrioventricular block in adult life: a prospective study. *Circulation*. 1995;92:442–449.
 92. Pinsky WW, Gillette PC, Garson A Jr, McNamara DG. Diagnosis, management, and long-term results of patients with congenital complete atrioventricular block. *Pediatrics*. 1982;69:728–733.
 93. Moak JP, Barron KS, Hougen TJ, et al. Congenital heart block: development of late-onset cardiomyopathy, a previously underappreciated sequela. *J Am Coll Cardiol*. 2001;37:238–242.
 94. Michaelsson M, Engle MA. Congenital complete heart block: an international study of the natural history. *Cardiovasc Clin*. 1972;4:85–101.
 95. Moss AJ, Liu JE, Gottlieb S, Locati EH, Schwartz PJ, Robinson JL. Efficacy of permanent pacing in the management of high-risk patients with long QT syndrome. *Circulation*. 1991;84:1524–1529.
 96. Viskin S, Alla SR, Barron HV, et al. Mode of onset of torsade de pointes in congenital long QT syndrome. *J Am Coll Cardiol*. 1996;28:1262–1268.
 97. Gillette PC, Zeigler VL, Case CL, Harold M, Buckles DS. Atrial antitachycardia pacing in children and young adults. *Am Heart J*. 1991;122:844–849.
 98. Rhodes LA, Walsh EP, Gamble WJ, Triedman JK, Saul JP. Benefits and potential risks of atrial antitachycardia pacing after repair of congenital heart disease. *Pacing Clin Electrophysiol*. 1995;18:1005–1016.
 99. Dewey RC, Capeless MA, Levy AM. Use of ambulatory electrocardiographic monitoring to identify high-risk patients with congenital complete heart block. *N Engl J Med*. 1987;316:835–839.
 100. Trippel DL, Parsons MK, Gillette PC. Infants with long-QT syndrome and 2:1 atrioventricular block. *Am Heart J*. 1995;130:1130–1144.
 101. Solti F, Szatmary L, Vecsey T, Renyi-Vamos F Jr, Bodor E. Congenital complete heart block associated with QT prolongation. *Eur Heart J*. 1992;13:1080–1083.
 102. Krongrad E. Prognosis for patients with congenital heart disease and postoperative intraventricular conduction defects. *Circulation*. 1978;57:867–870.
 103. Sholler GF, Walsh EP. Congenital complete heart block in patients without anatomic cardiac defects. *Am Heart J*. 1989;118:1193–1198.
 104. Greenspan AM, Kay HR, Berger BC, Greenberg RM, Greenspon AJ, Gaughan MJ. Incidence of unwarranted implantation of permanent cardiac pacemakers in a large medical population. *N Engl J Med*. 1988;318:158–163.
 105. Fananapazir L, Epstein ND, Curiel RV, Panza JA, Tripodi D, McAreavey D. Long-term results of dual-chamber (DDD) pacing in obstructive hypertrophic cardiomyopathy: evidence for progressive symptomatic and hemodynamic improvement and reduction of left ventricular hypertrophy. *Circulation*. 1994;90:2731–2742.
 106. Nishimura RA, Hayes DL, Ilstrup DM, Holmes DR Jr, Tajik AJ. Effect of dual-chamber pacing on systolic and diastolic function in patients with hypertrophic cardiomyopathy: acute Doppler echocardiographic and catheterization hemodynamic study. *J Am Coll Cardiol*. 1996;27:421–430.
 107. Nishimura RA, Symanski JD, Hurrell DG, Trusty JM, Hayes DL, Tajik AJ. Dual-chamber pacing for cardiomyopathies: a 1996 clinical perspective. *Mayo Clin Proc*. 1996;71:1077–1087.
 108. Kappenberger L, Linde C, Daubert C, et al, for the PIC Study Group. Pacing in hypertrophic obstructive cardiomyopathy: a randomized crossover study. *Eur Heart J*. 1997;18:1249–1256.

109. Nishimura RA, Trusty JM, Hayes DL, et al. Dual-chamber pacing for hypertrophic cardiomyopathy: a randomized, double-blind, crossover trial. *J Am Coll Cardiol*. 1997;29:435–441.
110. Maron BJ, Nishimura RA, McKenna WJ, Rakowski H, Josephson ME, Kieval RS. Assessment of permanent dual-chamber pacing as a treatment for drug-refractory symptomatic patients with obstructive hypertrophic cardiomyopathy: a randomized, double-blind, crossover study (M-PATHY). *Circulation*. 1999;99:2927–2933.
111. Cazeau S, Leclercq C, Lavergne T, et al. Effects of multisite biventricular pacing in patients with heart failure and intraventricular conduction delay. *N Engl J Med*. 2001;344:873–880.
112. Abraham WT, et al. Randomized controlled trial of cardiac resynchronization in chronic heart failure. *N Engl J Med*. 2002;346:1845–1853.
113. Mehta D, Saksena S, Krol RB, Makhija V. Comparison of clinical benefits and outcome in patients with programmable and nonprogrammable implantable cardioverter defibrillators. *Pacing Clin Electrophysiol*. 1992;15:1279–1290.
114. Saksena S, Poczebott-Johanos M, Castle LW, et al, for the Guardian Multicenter Investigators Group. Long-term multicenter experience with a second-generation implantable pacemaker-defibrillator in patients with malignant ventricular tachyarrhythmias. *J Am Coll Cardiol*. 1992;19:490–499.
115. Bardy GH, Troutman C, Poole JE, et al. Clinical experience with a tiered-therapy, multiprogrammable antiarrhythmia device. *Circulation*. 1992;85:1689–1698.
116. Mirowski M, Reid PR, Mower MM, et al. Termination of malignant ventricular arrhythmias with an implanted automatic defibrillator in human beings. *N Engl J Med*. 1980;303:322–324.
117. Lehmann MH, Steinman RT, Schuger CD, Jackson K. The automatic implantable cardioverter defibrillator as antiarrhythmic treatment modality of choice for survivors of cardiac arrest unrelated to acute myocardial infarction. *Am J Cardiol*. 1988;62:803–805.
118. Tchou PJ, Kadri N, Anderson J, Caceres JA, Jazayeri M, Akhtar M. Automatic implantable cardioverter defibrillators and survival of patients with left ventricular dysfunction and malignant ventricular arrhythmias. *Ann Intern Med*. 1988;109:529–534.
119. Fogoros RN, Fiedler SB, Elson JJ. The automatic implantable cardioverter-defibrillator in drug-refractory ventricular tachyarrhythmias. *Ann Intern Med*. 1987;107:635–641.
120. Winkle RA, Mead RH, Ruder MA, et al. Long-term outcome with the automatic implantable cardioverter-defibrillator. *J Am Coll Cardiol*. 1989;13:1353–1561.
121. Fogoros RN, Elson JJ, Bonnet CA, Fiedler SB, Burkholder JA. Efficacy of the automatic implantable cardioverter-defibrillator in prolonging survival in patients with severe underlying cardiac disease. *J Am Coll Cardiol*. 1990;16:381–386.
122. Newman D, Sauve MJ, Herre J, et al. Survival after implantation of the cardioverter defibrillator. *Am J Cardiol*. 1992;69:899–903.
123. Powell AC, Fuchs T, Finkelstein DM, et al. Influence of implantable cardioverter-defibrillators on the long-term prognosis of survivors of out-of-hospital cardiac arrest. *Circulation*. 1993;88:1083–1092.
124. Crandall BG, Morris CD, Cutler JE, et al. Implantable cardioverter-defibrillator therapy in survivors of out-of-hospital sudden cardiac death without inducible arrhythmias. *J Am Coll Cardiol*. 1993;21:1186–1192.
125. PCD Investigator Group. Clinical outcome of patients with malignant ventricular tachyarrhythmias and a multiprogrammable implantable cardioverter-defibrillator implanted with or without thoracotomy: an international multicenter study. *J Am Coll Cardiol*. 1994;23:1521–1530.
126. Zipes DP, Roberts D, for the Pacemaker-Cardioverter-Defibrillator Investigators. Results of the international study of the implantable pacemaker cardioverter-defibrillator: a comparison of epicardial and endocardial lead systems. *Circulation*. 1995;92:59–65.
127. Wever EF, Hauer RN, Schrijvers G, et al. Cost-effectiveness of implantable defibrillator as first-choice therapy versus electrophysiologically guided, tiered strategy in postinfarct sudden death survivors: a randomized study. *Circulation*. 1996;93:489–496.
128. The Antiarrhythmics versus Implantable Defibrillators (AVID) Investigators. A comparison of antiarrhythmic-drug therapy with implantable defibrillators in patients resuscitated from near-fatal ventricular arrhythmias. *N Engl J Med*. 1997;337:1576–1583.
129. Borggrefe M, Chen X, Martinez-Rubio A, et al. The role of implantable cardioverter defibrillators in dilated cardiomyopathy. *Am Heart J*. 1994;127:1145–1150.
130. Morady F, Harvey M, Kalbfleisch SJ, et al. Radiofrequency catheter ablation of ventricular tachycardia in patients with coronary artery disease. *Circulation*. 1993;87:363–372.
131. Wever EF, Hauer RN, van Capelle FL, et al. Randomized study of implantable defibrillator as first-choice therapy versus conventional strategy in postinfarct sudden death survivors. *Circulation*. 1995;91:2195–2203.
132. Krol RB, Saksena S. Clinical trials of antiarrhythmic drugs in recipients of implantable cardioverter-defibrillators. In: Saksena S, Luderitz B, eds. *Interventional electrophysiology*. Armonk: Futura Publishing Co, 1996:365–375.
133. Connolly SJ, Gent M, Roberts RS, et al. Canadian implantable defibrillator study (CIDS): a randomized trial of the implantable cardioverter defibrillator against amiodarone. *Circulation*. 2000;101:1297–1302.
134. Kuck KH, Cappato R, Siebels J, Ruppel R. Randomized comparison of antiarrhythmic drug therapy with implantable defibrillators in patients resuscitated from cardiac arrest: the Cardiac Arrest Study Hamburg (CASH). *Circulation*. 2000;102:748–754.
135. Saksena S, Breithardt G, Dorian P, Greene HL, Madan N, Block M. Nonpharmacological therapy for malignant ventricular arrhythmias: implantable defibrillator trials. *Prog Cardiovasc Dis*. 1996;38:429–444.
136. Nisam S, Kaye SA, Mower MM, Hull M. AICD automatic cardioverter defibrillator clinical update: 14 years experience in over 34,000 patients. *Pacing Clin Electrophysiol*. 1995;18:142–147.
137. Axtell K, Tchou P, Akhtar M. Survival in patients with depressed left ventricular function treated by implantable cardioverter defibrillator. *Pacing Clin Electrophysiol*. 1991;14:291–296.
138. Hook BG, Marchlinski FE. Value of ventricular electrogram recordings in the diagnosis of arrhythmias precipitating electrical device shock therapy. *J Am Coll Cardiol*. 1991;17:985–990.
139. Leitch JW, Gillis AM, Wyse DG, et al. Reduction in defibrillator shocks with an implantable device combining antitachycardia pacing and shock therapy. *J Am Coll Cardiol*. 1991;18:145–151.
140. Bocker D, Haverkamp W, Block M, Borggrefe M, Hammel D, Breithardt G. Comparison of d,l-sotalol and implantable defibrillators for treatment of sustained ventricular tachycardia or fibrillation in patients with coronary artery disease. *Circulation*. 1996;94:151–157.
141. Moss AJ, Hall WJ, Cannom DS, et al, Multicenter Automatic Defibrillator Implantation Trial Investigators. Improved survival with an implanted defibrillator in patients with coronary disease at high risk for ventricular arrhythmia. *N Engl J Med*. 1996;335:1933–1940.
142. Saksena S, Moss AJ, Gorgeberidze I, et al. Factors associated with shock delivery in the Multicenter Automatic Defibrillator Implantation Trial [MADIT] [abstract]. *J Am Coll Cardiol*. 1997;29(Suppl A):79A.
143. Buxton AE, Lee KL, Fisher JD, Josephson ME, Prystowsky EN, Hafley G, for the Multicenter Unsustained Tachycardia Trial Investigators. A randomized study of the prevention of sudden death in patients with coronary artery disease. *N Engl J Med*. 1999;341:1882–1890.
144. Bardy GH, Yee R, Jung W, for the Active Can Investigators. Multicenter experience with a pectoral unipolar implantable cardioverter-defibrillator. *J Am Coll Cardiol*. 1996;28:400–410.
145. Groh WJ, Silka MJ, Oliver RP, Halperin BD, McAnulty JH, Kron J. Use of implantable cardioverter-defibrillators in the congenital long QT syndrome. *Am J Cardiol*. 1996;78:703–706.
146. Grimm M, Wieselthaler G, Avanesian R, et al. The impact of implantable cardioverter-defibrillators on mortality among patients on the waiting list for heart transplantation. *J Thorac Cardiovasc Surg*. 1995;110:532–539.
147. Sweeney MO, Ruskin JN, Garan H, et al. Influence of the implantable cardioverter/defibrillator on sudden death and total mortality in patients evaluated for cardiac transplantation. *Circulation*. 1995;92:3273–3281.
148. Garson A Jr, Dick M, Fournier A, et al. The long QT syndrome in children: an international study of 287 patients. *Circulation*. 1993;87:1866–1872.
149. McKenna WJ, Franklin RC, Nihoyannopoulos P, Robinson KC, Deanfield JE. Arrhythmia and prognosis in infants, children and adolescents with hypertrophic cardiomyopathy. *J Am Coll Cardiol*. 1988;11:147–153.
150. Fananapazir L, Epstein SE. Hemodynamic and electrophysiologic evaluation of patients with hypertrophic cardiomyopathy surviving cardiac arrest. *Am J Cardiol*. 1991;67:280–287.
151. Wichter T, Block M, Bocker D, Borggrefe G, Breithardt G. Cardioverter-defibrillator therapy in a high-risk subgroup of patients

- with arrhythmogenic right ventricular disease [abstract]. *J Am Coll Cardiol*. 1993;21:127A.
152. Evans RW, Manninen DL, Dong FB, Frist WH, Kirklin JK. The medical and surgical determinants of heart transplantation outcomes: the results of a consensus survey in the United States. *J Heart Lung Transplant*. 1993;12:42–45.
 153. Maron BJ, Fananapazir L. Sudden cardiac death in hypertrophic cardiomyopathy. *Circulation*. 1992;85:1-57–I-63.
 154. Kaminer SJ, Pickoff AS, Dunnigan A, Sterba R, Wolff GS. Cardiomyopathy and the use of implanted cardio-defibrillators in children. *Pacing Clin Electrophysiol*. 1990;13:593–597.
 155. Mehta D, Saksena S, Krol RB, et al. Device use patterns and clinical outcome of implantable cardioverter defibrillator patients with moderate and severe impairment of left ventricular function. *Pacing Clin Electrophysiol*. 1993;16:179–185.
 156. Wilber DJ, Olshansky B, Moran JF, Scanlon PJ. Electrophysiological testing and nonsustained ventricular tachycardia: use and limitations in patients with coronary artery disease and impaired ventricular function. *Circulation*. 1990;82:350–358.
 157. Brugada J, Brugada R, Brugada P. Pharmacological and device approach to therapy of inherited cardiac diseases associated with cardiac arrhythmias and sudden death. *J Electrocardiol*. 2000;33(Suppl):41–47.
 158. Priori SG, Napolitano C, Gasparini M, et al. Natural history of Brugada syndrome: insights for risk stratification and management. *Circulation*. 2002;105:1342–1347.
 159. Moss AJ, Zareba W, Hall WJ, et al. Prophylactic implantation of a defibrillator in patients with myocardial infarction and reduced ejection fraction. *N Engl J Med*. 2002;346:877–883.
 160. Stevenson WG, Khan H, Sager P, et al. Identification of reentry circuit sites during catheter mapping and radiofrequency ablation of ventricular tachycardia late after myocardial infarction. *Circulation*. 1993;88:1647–1670.
 161. Gonska BD, Cao K, Schaumann A, Dorszewski A, von zur MF, Kreuzer H. Catheter ablation of ventricular tachycardia in 136 patients with coronary artery disease: results and long-term follow-up. *J Am Coll Cardiol*. 1994;24:1506–1514.
 162. Hindricks G, for the Multicentre European Radiofrequency Survey (MERFS) investigators of the Working Group on Arrhythmias of the European Society of Cardiology. The Multicentre European Radiofrequency Survey (MERFS): complications of radiofrequency catheter ablation of arrhythmias. *Eur Heart J*. 1993;14:1644–1653.
 163. Klein LS, Shih HT, Hackett FK, Zipes DP, Miles WM. Radiofrequency catheter ablation of ventricular tachycardia in patients without structural heart disease. *Circulation*. 1992;85:1666–1674.
 164. Maron BJ, Poliac LC, Kaplan JA, Mueller FO. Blunt impact to the chest leading to sudden death from cardiac arrest during sports activities. *N Engl J Med*. 1995;333:337–342.
 165. Michaud GF, Sticherling C, Tada H, et al. Relationship between serum potassium concentration and risk of recurrent ventricular tachycardia or ventricular fibrillation. *J Cardiovasc Electrophysiol*. 2001;12:1109–1112.
 166. Michaud GF, Strickberger SA. Should an abnormal serum potassium concentration be considered a correctable cause of cardiac arrest? *J Am Coll Cardiol* 2001;38:1224–1225.
 167. Anderson JL, Hallstrom AP, Epstein AE, et al, for the AVID Investigators. Design and results of the Antiarrhythmics Vs Implantable Defibrillators (AVID) registry. *Circulation*. 1999;99:1692–1699.
 168. Vlay SC, Olson LC, Fricchione GL, Friedman R. Anxiety and anger in patients with ventricular tachyarrhythmias: responses after automatic internal cardioverter defibrillator implantation. *Pacing Clin Electrophysiol*. 1989;12:366–373.
 169. Luderitz B, Jung W, Deister A, Marneros A, Manz M. Patient acceptance of the implantable cardioverter defibrillator in ventricular tachyarrhythmias. *Pacing Clin Electrophysiol*. 1993;16:1815–1821.
 170. Bigger JT Jr, for the Coronary Artery Bypass Graft (CABG) Patch Trial Investigators. Prophylactic use of implanted cardiac defibrillators in patients at high risk for ventricular arrhythmias after coronary-artery bypass graft surgery. *N Engl J Med*. 1997;337:1569–1575.

KEY WORDS: ACC/AHA Scientific Statements ■ pacemakers ■ arrhythmia ■ defibrillation ■ syncope

ACC/AHA/NASPE 2002 Guideline Update for Implantation of Cardiac Pacemakers and Antiarrhythmia Devices: Summary Article: A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (ACC/AHA/NASPE Committee to Update the 1998 Pacemaker Guidelines)

Committee Members, Gabriel Gregoratos, Jonathan Abrams, Andrew E. Epstein, Roger A. Freedman, David L. Hayes, Mark A. Hlatky, Richard E. Kerber, Gerald V. Naccarelli, Mark H. Schoenfeld, Michael J. Silka, Stephen L. Winters, Raymond J. Gibbons, Elliott M. Antman, Joseph S. Alpert, Gabriel Gregoratos, Loren F. Hiratzka, David P. Faxon, Alice K. Jacobs, Valentin Fuster and Sidney C. Smith, Jr

Circulation. 2002;106:2145-2161

doi: 10.1161/01.CIR.0000035996.46455.09

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231

Copyright © 2002 American Heart Association, Inc. All rights reserved.

Print ISSN: 0009-7322. Online ISSN: 1524-4539

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

<http://circ.ahajournals.org/content/106/16/2145>

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in *Circulation* can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the [Permissions and Rights Question and Answer](#) document.

Reprints: Information about reprints can be found online at:
<http://www.lww.com/reprints>

Subscriptions: Information about subscribing to *Circulation* is online at:
<http://circ.ahajournals.org/subscriptions/>