Guidelines for Clinical Intracardiac Electrophysiologic Studies

A Report of the American College of Cardiology/American Heart Association Task Force on Assessment of Diagnostic and Therapeutic Cardiovascular Procedures (Subcommittee to Assess Clinical Intracardiac Electrophysiologic Studies)

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

To this end, the American College of Cardiology and the American Heart Association in 1980 established a Task Force on Assessment of Diagnostic and Therapeutic Cardiovascular Procedures with the following charge:

The Task Force of the American College of Cardiology and the American Heart Association shall define the role of specific noninvasive and invasive procedures in the diagnosis and management of cardiovascular disease.

The Task Force shall address, when appropriate, the contribution, uniqueness, sensitivity, specificity, indications, contraindications, and cost-effectiveness of such specific procedures.

The Task Force shall include a Chairman and six members, three representatives from the American Heart Association and three representatives from the American College of Cardiology. The Task Force may select ad hoc members as needed upon the approval of the Presidents of both organizations. Recommendations of the Task Force are forwarded to the President of each organization.

The members of the Task Force are: Roman W. DeSanctis, MD, Harold T. Dodge, MD, Harriet P. Dustan, MD, J. Ward Kennedy, MD, T. Joseph Reeves, MD, Sylvan Lee Weinberg, MD, and Charles Fisch, MD, Chairman.

This document was reviewed by the officers and other responsible individuals of the two organizations and received final approval in June 1989. It is being published simultaneously in Circulation and the Journal of the American College of Cardiology. The potential impact of this document on the prac-
tice of cardiology and some of its unavoidable shortcomings are clearly set out in the introduction.
Charles Fisch, MD, FACC

I. Introduction

During the past 20 years, cardiac electrophysiologic studies have evolved into widely employed clinical tools, often indispensable in evaluating patients with specific cardiac arrhythmias. Because such studies carry a tangible, though minimal, risk and are expensive in terms of personnel and equipment, it is important that their clinical usefulness for the diagnosis and therapy of cardiac arrhythmias be carefully considered. This document presents current opinion regarding the clinical application of invasive cardiac electrophysiologic studies and is an extension of previous efforts in this area.\(^1\)\(^2\) The American College of Cardiology and the American Heart Association recognize that the ultimate judgment regarding the propriety of any specific procedure is the responsibility of the physician caring for the patient. Therefore, these guidelines should not be considered all inclusive or exclusive of other methods that may be available for the care of the individual patient. Finally, it is important to emphasize that this document may require periodic updating as the indications for electrophysiologic studies continue to evolve.

It is assumed, for the purposes of this document, that the electrophysiologic studies are performed by appropriately trained and qualified personnel in adequately equipped laboratories\(^3\) and that complete electrophysiologic studies, indicated by the patient’s clinical state and the arrhythmia in question, are performed. In general, such studies include intravenous or intraarterial placement, or both, of one or more electrode catheters at one or more sites in the atria, ventricles, or coronary sinus (occasionally in the pulmonary artery or aorta) to record or stimulate at various rates and cadences. Such studies are performed to evaluate electrophysiologic properties such as automaticity, conduction and refractoriness, to initiate and terminate tachycardias, to map sequence of activation, to evaluate patients for various forms of therapy, and to judge the response to therapy. Studies are modified according to the patient, the problem being investigated, and the site of the study, i.e., bedside, operating room, or electrophysiology laboratory. Increasingly, in the course of these studies, therapeutic interventions such as catheter ablation procedures are being performed.

The indications for electrophysiologic studies have been divided into three classes:

Class I

Conditions for which there is general agreement that the electrophysiologic study provides information that is very useful and important for patient management. Experts agree that patients with these conditions should undergo electrophysiologic studies.

Class II

Conditions for which electrophysiologic studies are frequently performed but there is less certainty regarding the usefulness of the information that is obtained. Experts are divided in their opinion as to whether patients with these conditions should undergo electrophysiologic study.

Class III

Conditions for which there is general agreement that electrophysiologic studies do not provide useful information. Experts agree that electrophysiologic studies are not warranted in patients with these conditions.

This classification is applied to three major patient groups: 1) patients who present with sinus node dysfunction, atrioventricular (AV) and intraventricular conduction delay or block, and supraventricular and ventricular tachycardias; 2) patients who present with a variety of clinical problems, including the long QT syndrome, Wolff-Parkinson-White abnormality without tachycardia, ventricular arrhythmias without tachycardia, unexplained near syncope or syncope, cardiac arrest, and unexplained episodes of palpitation; 3) patients undergoing pharmacologic, electrical, or ablative treatment of arrhythmias. Because the application of electrophysiologic studies in children on occasion differs from that in adults, that subject is dealt with in a separate section.

II. Role of Electrophysiologic Study in Evaluating Sinus Node Function

Sinus node dysfunction may be diagnosed electrocardiographically by the presence of persistent unexplained sinus bradycardia, sinus arrest, or sinoatrial exit block. Sinus node dysfunction may be related to intrinsic sinus node disease, the presence of drugs that depress sinus node function (e.g., digitalis, \(\beta\)-adrenergic blocker), autonomic imbalance (e.g., increased vagal tone), or a combination of these conditions. Evaluation of sinus node function requires, in addition to the electrocardiogram, the use of drugs such as atropine, isoproterenol and propranolol.\(^4\) Autonomic blockade may help to establish the mechanism of sinus node dysfunction in some patients. Exercise testing and long-term electrocardiographic (ECG) recording are also helpful in assessing sinus node function.\(^5\) Carotid sinus massage may identify patients with sinus node dysfunction and carotid sinus hypersensitivity.

Invasive electrophysiologic studies can be used to assess sinus node automaticity by measuring the sinus node recovery time and sinoatrial conduction time.\(^6\) The response to right atrial pacing for 30 seconds at several paced rates is used to determine the sinus node recovery time,\(^7\) which is the interval between the last paced right atrial electrogram and the first spontaneous sinus depolarization. Because the sinus node recovery time is influenced by the spontaneous sinus rate, the corrected sinus node
recovery time, i.e., the difference between the sinus node recovery time and the spontaneous sinus cycle length before pacing, has also been used to evaluate sinus node automaticity. Occasionally after cessation of pacing, the first return cycle is of normal duration but the subsequent cycles may show an abrupt prolongation, called secondary pauses.

Sinoatrial conduction time can be estimated indirectly from the responses to programmed atrial stimulation during sinus rhythm or atrial pacing at a rate just faster than the sinus rate.8,9 It can also be measured directly with extracellular electrodes placed in the area of the sinus node.10 Direct and indirect measurements of sinoatrial conduction time seem to correlate well in patients with normal sinus node function.6 Measurement of sinus node refractoriness is a potentially important method of assessing sinus node function but needs further evaluation.11

The sensitivity of the corrected sinus node recovery time for detecting sinus node dysfunction is 54% and that of the sinoatrial conduction time is approximately 51%. The combined sensitivity of both increases to 64%. Sensitivity increases in the elderly with symptomatic sinus node dysfunction and in those showing sinoatrial exit block.12,13 The specificity of the two tests combined is about 88%. Therefore, abnormal sinus node function determined by electrophysiologic study should be viewed as supporting evidence in clinical decision making. In addition to evaluating sinus node function, it is important to test AV node and His-Purkinje function because AV conduction is frequently impaired in patients with sinus node disease.14

**Recommendations for Electrophysiologic Studies**

**Class I.** Symptomatic patients (with syncope or near syncope), in whom sinus node dysfunction is suspected as a cause of symptoms but is a causal relation between sinus bradycardia, sinus pauses, or sinus node exit block and the symptoms cannot be established by other means.

**Class II.**
1. Patients who require pacemaker insertion to treat sinus node dysfunction, when it is necessary to assess anterograde and retrograde AV conduction and vulnerability to atrial tachyarrhythmias, to determine the appropriate site and modality of pacing.
2. Patients with sinus node dysfunction, to determine the severity or mechanism, or both, of dysfunction (intrinsic versus extrinsic) and response to drugs, information that may help direct therapy.
3. Symptomatic patients (syncope and near syncope) with sinus node dysfunction, to exclude other arrhythmic mechanisms (e.g., ventricular tachycardia) as a cause of symptoms.

**Class III.**
1. Symptomatic patients with sinus node dysfunction when symptoms are clearly related to a documented bradycardia.
2. Asymptomatic patients with sinus bradyarrhythmia or sinus pauses observed during sleep.

**III. Role of Electrophysiologic Study In Patients With Acquired Atioventricular Block**

The ECG classification of AV block includes these categories: 1) First degree AV block—prolongation of the PR interval beyond 0.20 second. 2) Second degree AV block—intermittent failure to conduct a single P wave. Two types have been described. In type I second degree AV block (Wenckebach block), there is progressive prolongation of the PR interval before the blocked P wave; in type II block, PR intervals are constant before the blocked P wave. AV block with a 2:1 conduction ratio is not classified as either type I or type II. 3) Advanced AV block—some experts recommend this additional category to define a condition in which multiple consecutive P waves are blocked but complete AV block is not present. 4) Complete AV block—failure of all P waves to conduct to the ventricle, resulting in complete dissociation between P waves and QRS complexes due to block.

His bundle recordings allow delineation of three anatomic sites of AV block:15 1) Proximal (above the His bundle), representing delay or block in the AV node; 2) Intra-Hisian, representing delay or block within the His bundle; and 3) Infra-Hisian or distal to the His bundle, representing block or delay distal to the His bundle recording site either in the distal His bundle or in the bundle branches.

There are certain correlations between ECG patterns and the site of block. In type I second degree AV block with narrow QRS complexes, the block is usually at the level of the AV node; less frequently it may be within the His bundle. In type I second degree AV block with wide QRS complexes (bundle branch block) the block may be in the AV node or within or below the His bundle. Type II second degree AV block is usually within or below the His bundle and is most often seen with bundle branch block. Rarely, the block can be in the AV node. In complete AV block with an escape rhythm of narrow QRS complexes, the site of block may be in the AV node or within the His bundle. In complete AV block with an escape rhythm and wide QRS complexes, the site of block may be in the AV node or within or below the His bundle. Clinical information regarding age, gender, underlying heart disease, and the use of cardiac medication may also be helpful in predicting the site of AV block.

Prognosis of patients with AV block depends on the site of block. Chronic first degree AV block, particularly AV node block, has a good prognosis. The abnormality is frequently drug related and reversible. The clinical course of patients with second degree AV node block is usually benign and the prognosis depends on the presence and severity of underlying heart disease.16 The prognosis of patients with second degree AV block within the His bundle is uncertain. Patients frequently mani-
fest congestive heart failure and syncope. Untreated chronic second degree block below the His bundle has a poor prognosis; patients frequently proceed to higher degrees of block and become symptomatic with syncope. Patients with untreated acquired complete AV block are often symptomatic regardless of the site of the block.

**Recommendations for Electrophysiologic Studies**

**Class I.**
1. Symptomatic patients (with syncope or near syncope), in whom His-Purkinje block, suspected as a cause of symptoms, has not been established with ECG recordings.
2. Patients with second or third degree AV block treated with a pacemaker who remain symptomatic (with syncope or near syncope), in whom ventricular tachyarrhythmia is suspected as a cause of symptoms.

**Class II.**
1. Patients with second or third degree AV block, in whom knowledge of the site or mechanism of block, or both, may help direct therapy or assess prognosis.
2. Patients with concealed junctional extrasystoles suspected as a cause of second or third degree AV block pattern (i.e., pseudo AV block).

**Class III.**
1. Patients in whom the symptoms and the presence of AV block are correlated by electrocardiography.
2. Asymptomatic patients with transient AV block associated with sinus slowing (e.g., nocturnal type I second degree AV block).

**IV. Role of Electrophysiologic Study in Patients With Chronic Intraventricular Conduction Delay**

As judged from the ECG, the intraventricular conduction system appears to be trifasicular, consisting of the two fascicles of the left bundle (anterior and posterior) and the right bundle branch. The anatomic basis for the trifasicular conduction system in humans is less clearly defined. The HV interval in patients with bifascicular block is a measure of the conduction time through the remaining functioning fascicle. Patients with bifascicular block and a prolonged HV interval (>55 msec) appear to have a slightly increased risk of developing complete trifasicular block. Although the prevalence of prolonged HV interval is high, the incidence of complete trifasicular block is low (2% to 3% annually, but greater if the HV interval exceeds 100 msec) and the rate of progression in absence of an acute intervening event (drugs, electrolytes, ischemia) is slow. Thus, the HV interval has a high sensitivity (82%), but a very low specificity (63%) for predicting the development of complete trifasicular block.

Rapid atrial pacing has been used to improve the specificity of electrophysiologic testing in patients with bifascicular block. A positive response consists of the development of block distal to the His bundle with rapid atrial pacing during 1:1 AV node conduction. Functional distal His block due to abrupt shortening of the coupling interval (such as during the long-short cycles of Wenckebach periods or at the onset of pacing) is not considered a positive response. The sensitivity of distal His block induced by atrial pacing is relatively low, but its positive predictive value for development of complete AV block is high.

Sudden death in patients with bifascicular block cannot always be explained by the development of complete trifasicular block. There is increasing evidence that ventricular arrhythmias may play a significant role in patients with advanced heart disease and bifascicular block. For this reason, electrophysiologic evaluation of symptomatic patients with intraventricular conduction defects should also include study of sinus node function and programmed atrial and ventricular stimulation in an attempt to induce tachyarrhythmias.

**Recommendations for Electrophysiologic Studies**

**Class I.** Symptomatic patients (with syncope or near syncope) with bundle branch block whose ventricular arrhythmia is suspected to cause the symptoms; not for study of intraventricular conduction delay itself.

**Class II.** Symptomatic patients with bundle branch block in whom the knowledge of the site, severity of conduction delay, or response to drugs may help to direct therapy or assess prognosis.

**Class III.**
1. Asymptomatic patients with intraventricular conduction delay.
2. Symptomatic patients with intraventricular conduction delay whose symptoms can be causally related to ECG events.

**V. Role of Electrophysiologic Study in the Diagnosis of Patients With a Narrow QRS Tachycardia (QRS complex <0.12 seconds)**

A narrow QRS tachycardia can be caused by rapid impulse formation in the sinus node (sinus node tachycardia), the atrium (atrial tachycardia, atrial flutter, and atrial fibrillation), and the AV node–His bundle (AV junctional tachycardia), reentry within the AV node (AV node reentrant tachycardia) or a circus movement using the AV node–His pathway for anterograde AV conduction and an accessory AV or nodoventricular pathway for retrograde conduction. Rarely, rapid impulse formation high in the intraventricular conduction system can lead to a ventricular tachycardia with a narrow QRS complex.

Frequently, careful examination of the 12 lead ECG, especially when a recording during carotid sinus massage is also available, facilitates making the correct diagnosis. Proper identification of the site of origin of atrial activity, its rate, and its relation to ventricular rhythm is essential.
Atrial tachycardia typically has atrial activity preceding the QRS complex, usually creating a PR interval shorter than the RP interval. The PR interval may vary according to AV node conduction and the rate of the atrial tachycardia. In typical AV node reentrant tachycardia, the atrium and ventricle are activated simultaneously, with the P wave hidden within the QRS complex or inscribed in the terminal portion of the QRS complex. In an atypical AV reentrant tachycardia, the P wave follows the QRS complex with an RP interval longer than the PR interval. When a tachycardia circuit is present with anterograde conduction over the AV node and retrograde conduction over an accessory AV or a nodoventricular pathway, atrial activation follows the QRS complex. If the bypass fiber conducts the impulse rapidly, the P wave occurs shortly after the QRS complex so that the RP interval is shorter than the PR interval. Only a slowly conducting accessory AV pathway results in an RP interval that is longer than the next PR interval.

Rarely, AV dissociation is present during a narrow QRS tachycardia. Such tachycardias may originate in the AV node, His bundle, or fascicles, or may be due to reentry using a circuit consisting of the AV node–His bundle anterogradely and a nodoventricular fiber retrogradely, or a circuit with anterograde conduction through the His bundle and retrograde conduction through a fasciculoventricular fiber. The correct identification of the site of origin of a supraventricular tachycardia can be facilitated by use of a decision tree.25

Recommendations for Electrophysiologic Studies

Class I.

1. Patients with frequent or poorly tolerated episodes of tachycardia not adequately responding to drug therapy in whom information about site of origin, mechanism, and electrophysiologic properties of the pathways of the tachycardia is essential for choosing appropriate therapy (drugs, catheter ablation, pacing, or surgery).

2. Patients who prefer ablative therapy to pharmacologic management (see Section XV, Role of Electrophysiologic Study in Patients Who Are Candidates for Ablative Therapy of Cardiac Arrhythmias).

Class II. Patients with less symptomatic but frequent episodes of tachycardia requiring drug treatment in whom there is concern about the effects of the antiarrhythmic drug on the sinus node or on AV conduction.

Class III.

1. Patients whose 12 lead ECG gives sufficient information about the type of tachycardia to allow selection of an appropriate antiarrhythmic drug treatment.

2. Patients whose tachycardias can easily be controlled by vagal maneuvers or drug therapy even in the absence of precise information on site of origin, mechanism, or pathway of the tachycardia.

VI. Role of Electrophysiologic Study in Establishing the Diagnosis in Patients With Wide QRS Complex Tachycardias

Wide QRS complex tachycardias can be caused by supraventricular tachycardia with aberration or preexisting bundle branch block, ventricular tachycardia, and a variety of pre-excitation syndromes, including those using AV bypass tracts anterogradely (atrial tachycardia, atrial flutter, antidromic circus movement tachycardia) and, less commonly, those using nodofascicular and nodoventricular bypass tracts. Standard ECG guidelines to distinguish supraventricular tachycardia with aberration from ventricular tachycardia, particularly for right bundle branch block patterns, have been established.26–29 However, criteria to differentiate left bundle branch block type tachycardias are less well defined.30 The ECG patterns of tachycardias with ventricular activation produced by conduction over an AV bypass tract cannot be distinguished from ventricular tachycardias originating in the base of the left ventricle on the basis of QRS configuration alone. Only the presence of AV dissociation or intermittent failure of retrograde conduction during AV reciprocating tachycardia (antidromic) can exclude an AV bypass tract. Characteristic ECG configurations of ventricular tachycardias caused by foci from various regions of the heart31 or ECG patterns that cannot be explained on the basis of ventricular pre-excitation can also be used to help exclude conduction over a bypass tract. Thus, if possible, a 12 lead ECG during sinus rhythm should be used for comparison. The presence of Wolff-Parkinson-White syndrome or an intraventricular conduction disturbance during sinus rhythm can help point to aberration. A preexisting intraventricular conduction disturbance during sinus rhythm renders many of the morphologic criteria of ventricular tachycardia susceptible to error. At times, a differential between ventricular tachycardia and aberration will remain in doubt. In such instances, electrophysiologic studies may be necessary to establish a definitive diagnosis.

To differentiate these tachycardias, one needs to 1) record a His deflection during the tachycardia or during sinus rhythm either before the onset or after the conversion of the tachycardia to be certain that the recording electrode is in an appropriate position to record a His deflection if one is present, 2) analyze the timing and sequence of atrial activation patterns during the tachycardia, and 3) assess the response of the His deflection (if seen) and atrial activation and timing in response to ventricular stimulation during the tachycardia. For example, atrial activation can be advanced by a premature ventricular stimulus during an AV reciprocating tachycardia and at a time when the His bundle is refractory, thus indicating the presence of an accessory pathway. Supraventricular tachycardia with aberration can be excluded by demonstrating the
absence of a His deflection, dissociation of the His deflection from the QRS complex during the tachycardia, or the presence of a His deflection with an HV interval shorter than that recorded during sinus rhythm. An HV interval equal to or exceeding the HV interval recorded during sinus rhythm suggests a supraventricular tachycardia, provided the His deflection is due to anterograde and not to retrograde activation. Catheter position is obviously critical for the demonstration of these phenomena. Two unique tachycardias that may be very difficult to classify or diagnose, or both, are bundle branch reentrant tachycardia and a tachycardia using a nodofascicular bypass tract for ventricular activation. Distinguishing various pre-excitation syndromes from ventricular tachycardia may be difficult and requires careful analysis of atrial activation during the tachycardia and responses to programmed stimulation during the tachycardia. However, because all tachycardias involving AV bypass tracts anterogradey require part of the atrium, the presence of AV dissociation supports a diagnosis of ventricular tachycardia. The differential diagnosis of these complex arrhythmias requires a comprehensive electrophysiologic study as well as a detailed analysis of the ECG.

Recommendations for Electrophysiologic Studies

Class I. Patients with wide QRS complex tachycardias that are sustained or symptomatic, or both, when the correct diagnosis is unclear and is necessary for appropriate patient care.

Class II. Patients with pre-excitation syndromes with suspected antidromic tachycardia, to evaluate the possibility of multiple bypass tracts.

Class III. Patients with ventricular tachycardia or supraventricular tachycardia with aberration or pre-excitation syndromes identified by ECG criteria.

VII. Role of Electrophysiologic Study in Patients With a Prolonged QT Interval Syndrome

Prolongation of the QT interval, with associated ventricular tachyarrhythmias, may occur chronically as a part of the congenital idiopathic prolonged QT interval syndrome, or may be acquired secondarily to metabolic, toxic, or pathophysiological factors. Influence of the autonomic nervous system is strongly suspected in the congenital long QT interval syndrome.

The role of electrophysiologic studies in the diagnosis and evaluation or in guiding management of either congenital or acquired forms of prolonged QT interval syndrome at present is limited. Electrophysiologic studies in patients with congenital long QT syndrome infrequently result in initiation of ventricular arrhythmias. Such studies are of limited or no predictive value. Similarly they are of no value in acquired forms of prolonged QT interval syndrome, whether or not the syndrome is associated with spontaneous arrhythmias. Some electrophysiologists have used electrophysiologic studies for diagnostic purposes in patients who have unexplained syncope or palpitation and also have acquired prolongation of the QT interval while receiving antiarrhythmic drugs. In the future, the use of monophasic action potential recordings may prove useful.

Recommendations for Electrophysiologic Studies

Class I. None.

Class II. Identification of a proarhythmic effect of antiarrhythmic drugs in patients experiencing their first episode or episodes of sustained ventricular tachycardia or cardiac arrest while receiving an antiarrhythmic drug.

Class III.

1. Patients with congenital QT prolongation.
2. Patients with acquired prolonged QT syndrome with symptoms closely related to an identifiable cause or mechanism.

VIII. Role of Electrophysiologic Study in Patients With the Wolff-Parkinson-White Syndrome

In the Wolff-Parkinson-White syndrome, the presence of an accessory AV pathway leads to early activation of a part or the whole of the ventricle by an impulse originating in the sinus node or atrium. The second pathway between atrium and ventricle makes it possible for arrhythmias to occur. The two most frequently occurring arrhythmias are a circus movement tachycardia and atrial fibrillation.

During circus movement tachycardia, the impulse travels in a circuit consisting of atrium \( \Rightarrow \) AV node \( \Rightarrow \) His bundle \( \Rightarrow \) bundle branch \( \Rightarrow \) ventricle \( \Rightarrow \) accessory pathway \( \Rightarrow \) atrium. A circus movement tachycardia with AV conduction over the AV node and ventriculoatrial (VA) conduction over the accessory AV pathway is referred to as orthodromic. A tachycardia conducting in the reverse direction (VA conduction over the accessory pathway and VA conduction over the His-AV node) is referred to as antidromic circus movement tachycardia. Essential for the occurrence of a circus movement tachycardia is temporary or permanent unidirectional block in one of the two AV pathways. This can occur with a critically timed atrial or ventricular premature complex, by reaching a critical sinus rate, or after the administration of a drug.

Atrial fibrillation in patients with the Wolff-Parkinson-White syndrome can prove to be an extremely serious arrhythmia in the setting of an accessory pathway with a short anterograde refractory period. In that situation the occurrence of ventricular fibrillation has been documented. The electrophysiologic properties of the accessory pathway are an important determinant of the incidence and rate of a circus movement tachycardia and of the ventricular rate during atrial fibrillation. Electrophysiologic studies give information about the electrophysiologic properties of the accessory pathways and their locations that may help direct therapy.
Recommendations for Electrophysiologic Studies

Class I. Patients considered for nonpharmacologic treatment (accessory pathway interruption or antitachycardia pacing), because of life-threatening or incapacitating arrhythmias or drug intolerance.

Class II.

1. Patients with arrhythmias requiring treatment in whom information about the type of arrhythmia, localization, number, and electrophysiologic properties of one or more accessory AV pathways and the effect of antiarrhythmic drugs may influence selection of the most appropriate treatment.

2. Asymptomatic patients with ECG evidence of Wolff-Parkinson-White syndrome during sinus rhythm, in whom knowledge of electrophysiologic characteristics of the accessory pathway may help in guidance of future participation in high risk occupations or activities.

3. Patients with Wolff-Parkinson-White syndrome and a family history of premature sudden death.

4. Patients with Wolff-Parkinson-White syndrome who are undergoing cardiac surgery for other reasons.

Class III. Asymptomatic persons without arrhythmias who are not in class II-2 or II-3.

IX. Role of Electrophysiologic Study in Patients With Ventricular Premature Complexes and Couplets

The presence of high grade ventricular ectopic activity in patients with coronary artery disease and left ventricular dysfunction is associated with increased risk for sudden death and cardiovascular mortality. However, the causal role of ventricular ectopic activity and sudden cardiac death remains controversial. Even though the ventricular ectopic complexes in themselves do not produce serious symptoms, they are often treated with the assumption that such abnormalities represent warning arrhythmias. To date, no studies have demonstrated that suppression of ventricular premature complexes with antiarrhythmic agents reduces the incidence of sudden cardiac death, and preliminary data from the Cardiac Arrhythmia Suppression Trial suggest that treatment with some antiarrhythmic drugs after myocardial infarction may be harmful. Because not all patients with a combination of complex ventricular ectopic activity and organic heart disease have the same risk for sudden cardiac death, attempts have been made to stratify these patients into low and high risk groups. One such approach utilizes electrophysiologic studies to separate patients into groups with and without electrically inducible ventricular tachycardia and to use this response to stratify patients into high and low risk groups, respectively. However, there are no randomized studies available to support the validity of such a stratification. Therefore, the clinical value of such studies in this patient population with complex ventricular ectopic activity and organic heart disease (excluding recent myocardial infarction) remains controversial.

Recommendations for Electrophysiologic Studies

Class I. None.

Class II. Patients with premature ventricular complexes and unexplained presyncope or syncope. (See section on "Unexplained Syncope.")

Class III. Asymptomatic patients with premature ventricular complexes.

X. Role of Electrophysiologic Study in Patients With Unexplained Syncope

Syncope and near syncope are frequent medical problems. Despite a detailed history, physical examination, and neurologic study, the etiology remains obscure in about 50% of cases. Because various noninvasive methods are often ineffective in identifying the underlying etiology, electrophysiologic studies have more recently been utilized to detect arrhythmias as a possible cause. A 12 lead ECG alone is unlikely to provide a clue to the etiologic diagnosis. However, it may yield insight into the nature of underlying structural heart disease, if present. The subsequent direction of evaluation generally depends on the clues obtained during the initial diagnostic studies. Among the cardiac causes of syncope, arrhythmias are a frequent consideration. Long-term ambulatory ECG monitoring, tilt table testing or exercise testing, alone or in combination, are performed before electrophysiologic studies are instituted. Because syncope and presyncope episodes are sporadic, continuous 24 to 48 hour ambulatory records are generally unrewarding. Electrophysiologic studies can often initiate clinically important arrhythmias and are a natural extension for arrhythmia evaluation in patients with unexplained syncope.

Attempts are made to assess sinus node dysfunction, the presence of His–Purkinje block, and the induction of supraventricular as well as ventricular tachycardias. The most common abnormality induced is ventricular tachycardia. Less frequently, His–Purkinje block and sinus node dysfunction are encountered. The studies suggest that induction of sustained monomorphic ventricular tachycardia, His–Purkinje block, and evidence of sinus node dysfunction may have diagnostic value. Induction of rapid nonsustained monomorphic ventricular tachycardia and of supraventricular tachycardia when associated with hypotension may also prove clinically important. On the other hand, induction of atrial fibrillation, polymorphic ventricular tachycardia-fibrillation with an aggressive stimulation protocol may be nonspecific findings.

The results of electrophysiologic studies also suggest that, in patients with underlying structural heart disease such as prior myocardial infarction, an arrhythmia is more likely to be the cause of syncope. Conversely, in patients without structural...
heart disease and a normal ECG, the diagnostic yield of electrophysiologic studies is relatively low.44 Patients with no abnormalities noted during electrophysiologic studies have a low incidence of sudden death during follow-up, suggesting a possible prognostic value of a negative test.

Recommendations for Electrophysiologic Studies

Class I. Patients with unexplained syncope and known or suspected structural heart disease.

Class II. Patients with unexplained syncope but without structural heart disease.

Class III. Patients with known cause of syncope.

XI. Role of Electrophysiologic Study in Survivors of Cardiac Arrest

Patients resuscitated from cardiac arrest without a new Q wave myocardial infarction are at high risk for recurrent cardiac arrest and sudden death after discharge.52–56 In this group, the 1 and 2 year rate of recurrence of cardiac arrest is approximately 30% and 45%, respectively.52,54 The recurrence rate appears to be declining because of aggressive therapy of the arrhythmias and the underlying ischemic heart disease.57

In the absence of antiarrhythmic drug therapy, ventricular tachyarrhythmias can be initiated during electrophysiologic studies in 70% to 80% of patients resuscitated from cardiac arrest. Of these, sustained monomorphic ventricular tachycardia constitutes 36% to 51%. The remaining rhythms include polymorphic ventricular tachycardia degenerating to ventricular fibrillation or nonsustained ventricular tachycardia.58–63 Sustained monomorphic ventricular tachycardia or ventricular tachycardia degenerating into ventricular fibrillation is an acceptable therapeutic end point, whereas nonsustained ventricular tachycardia as a guide to therapy is a more controversial end point.

Identification of a drug effective in suppression of inducible ventricular tachycardia or fibrillation has been reported in 26% to 80% of survivors of cardiac arrest.58–63 Such a response predicts suppression of ventricular tachycardia, ventricular fibrillation, and prevention of sudden death during long-term follow-up.63 Although some patients whose arrhythmia remains inducible may not have a spontaneous recurrence, survivors of cardiac arrest with ventricular tachycardia or ventricular fibrillation still inducible at the time of hospital discharge are at significantly higher risk for recurrent cardiac arrest and sudden death (estimated risk, 23% at 1 year and 30% at 3 years). Patients whose arrhythmias have been suppressed by drugs or surgery, or both, have an estimated risk for recurrence of sudden death and cardiac arrest of 6% at 1 year and 15% at 3 years; p=0.00463 (see section XIII on Role of Electrophysiologic Study to Guide Drug Therapy). Cardiac arrest survivors whose arrhythmia remains electrically inducible despite drug treatment may be considered for nonpharmacologic treatment alterna-tives, including surgery and automatic implantable defibrillators.

Failure to induce ventricular arrhythmias during baseline electrophysiologic studies in the absence of antiarrhythmic drugs in survivors of cardiac arrest is associated with a variable prognosis. Patients with depressed left ventricular function in whom no obvious reversible cause of the arrhythmia can be identified, e.g., ischemia, remain at significant risk for recurrent cardiac arrest.58–64

Guidance of therapy by electrophysiologic studies is indicated in the majority of survivors of cardiac arrest, especially in those with infrequent spontaneous ventricular arrhythmias, and it is generally accepted as the first approach to management when available.

Recommendations for Electrophysiologic Studies

Class I.

1. Patients surviving an episode of cardiac arrest without evidence of an acute Q wave myocardial infarction.

2. Patients surviving an episode of cardiac arrest occurring ≥48 hours after acute myocardial infarction.

Class II. Patients surviving a cardiac arrest due to a bradyarrhythmia.

Class III.

1. Patients with cardiac arrest occurring only within the first 48 hours of acute myocardial infarction.

2. Patients with cardiac arrest resulting from acute reversible ischemia or another clearly identifiable cause (e.g., aortic stenosis, congenital long QT syndrome).

XII. Role of Electrophysiologic Study in Patients With Unexplained Palpitations

Long-term ambulatory recording is the most useful procedure to document the cardiac rhythm associated with palpitation.65 The recording can be a continuous 24 hour recording or can be intermittent. Electrophysiologic studies are used only if repeated recording attempts fail to provide an answer. The sensitivity of electrophysiologic studies is low in patients with unexplained palpitation.

Recommendations for Electrophysiologic Studies

Class I. Patients with palpitation who have a pulse rate that has been documented by medical personnel to be inappropriately rapid (e.g., >150 beats/min) and in whom ECG recordings fail to document the cause of the symptoms.

Class II. Patients with clinically significant palpitations, suspected to be of cardiac origin, in whom the symptoms are sporadic and cannot be documented despite repeated long-term ECG or event recordings, to determine the mechanism of arrhythmias, direct the therapy or assess prognosis.

Class III. Patients with palpitation due to extra-cardiac causes (e.g., hyperthyroidism).
XIII. Role of Electrophysiologic Study to Guide Drug Therapy

Electrophysiologic studies have been used in patients with a variety of electrically inducible cardiac arrhythmias to predict antiarrhythmic drug efficacy. After inducibility is documented, the drug to be evaluated is administered, preferably orally, and electrical stimulation is repeated. In general, if the drug prevents electrical induction of the arrhythmia, a prediction of drug efficacy is made; if the arrhythmia remains inducible, the drug will most likely not prove efficacious.

Ventricular Arrhythmias

The use of electrophysiologic studies to test drug efficacy is feasible for most drugs in patients with documented, sustained, monomorphic ventricular tachycardia66 and in survivors of cardiac arrest.67 The data strongly suggest that electrophysiologic studies are the method of choice to assess drug efficacy in patients who have inducible ventricular tachyarrhythmias but insufficient spontaneous ventricular arrhythmias to allow assessment by serial long-term ECG recordings. A similar approach can be used in some patients with nonsustained ventricular tachycardia.68,69

Some patients develop their first episode of sustained ventricular tachycardia or cardiac arrest while receiving an antiarrhythmic drug. Failure to induce ventricular tachyarrhythmia after withdrawal of the antiarrhythmic drug in these patients and presence of inducibility with reintroduction of the drug suggest that the arrhythmia was exacerbated or provoked by the drug.70

Supraventricular Arrhythmias

In patients with the Wolff-Parkinson-White syndrome who have circus movement tachycardia utilizing the AV node and accessory pathway, prevention of tachycardia induction by drug administration predicts long-term efficacy. In patients with the Wolff-Parkinson-White syndrome who have atrial fibrillation with anterograde conduction over the accessory pathway, most, although not all, investigators believe that complete block of anterograde conduction over the accessory pathway, marked lengthening of the anterograde refractory period of the accessory pathway and a marked reduction in the ventricular response to induced atrial fibrillation conducted over the accessory pathway are predictive of a therapeutic success.71,72 Induction of tachycardia after isoproterenol challenge may improve prediction of drug efficacy.

Limited data suggest that drug efficacy may be predicted by response to electrophysiologic studies in selected patients with AV node reentrant tachycardia.73 Prevention of electrical induction of the atrial fibrillation may predict long-term suppression of spontaneous atrial fibrillation.74

There is no documentation of prediction of drug efficacy based on electrophysiologic studies in patients with inducible sinoatrial node reentry, intraatrial reentry and ectopic atrial tachycardias. Multifocal atrial tachycardia, isolated premature atrial complexes, and other less common atrial arrhythmias are not inducible by electrophysiologic studies.

Specific Antiarrhythmic Drugs

Programmed electrical stimulation is useful in assessing efficacy of most drugs approved for use in the United States. The utility of electrophysiologic studies in assessing efficacy of amiodarone is controversial.75–78 Though prevention of ventricular tachyarrhythmia induction by amiodarone is a highly accurate predictor of long-term efficacy, many patients whose ventricular tachyarrhythmia continues to be inducible despite therapy with amiodarone may remain asymptomatic and free of arrhythmias. The likely prevention of sudden death by amiodarone in face of continuing arrhythmia inducibility can be anticipated when the induced arrhythmia is associated with a slower rate and absence of hemodynamic deterioration.75 Each new antiarrhythmic drug must be evaluated individually to determine whether electrophysiologic studies can be used to predict its efficacy in suppression of spontaneous arrhythmias.

Recommendations for Electrophysiologic Studies

Class I

1. Sustained ventricular tachycardia or cardiac arrest due to ventricular tachycardia or ventricular fibrillation not associated with the long QT syndrome or appearing within 48 hours of an acute myocardial infarction, especially if spontaneous premature ventricular complexes are too infrequent to permit use of long-term ECG recordings.

2. Wolff-Parkinson-White syndrome with atrial fibrillation associated with a rapid ventricular response, a short bypass anterograde refractory period, or cardiac arrest; or with recurrent, symptomatic circus movement tachycardia unresponsive to empiric antiarrhythmic therapy.

3. AV node reentrant tachycardia unresponsive to empiric antiarrhythmic therapy.

Class II

1. Recurrent, symptomatic paroxysmal atrial fibrillation not prevented by empiric antiarrhythmic therapy.

2. Recurrent, symptomatic, inducible sinoatrial node reentry, intraatrial reentry and ectopic atrial tachycardia unresponsive to empiric therapy.

3. Recurrent, nonsustained ventricular tachycardia not associated with acute myocardial infarction or the long QT syndrome.

4. Identification of a proarrhythmic effect of antiarrhythmic drugs in patients experiencing their first episode or episodes of sustained ventricular tachycardia or cardiac arrest while receiving an antiarrhythmic drug.
5. Risk stratification and consideration for therapy of the postmyocardial infarction patient with reduced left ventricular function, frequent premature ventricular complexes or episodes of nonsustained ventricular tachycardia, or both, especially if the signal-averaged ECG shows the presence of late potentials.

Class III
1. Isolated premature atrial or ventricular complexes.
2. Multifocal atrial tachycardia.
3. Ventricular tachycardia or cardiac arrest occurring only in the acute phase of myocardial infarction (≤48 hours).
4. Asymptomatic, nonrecurrent, or drug-responsive supraventricular or nonsustained ventricular tachyarrhythmias.
5. Ventricular arrhythmias associated with the congenital long QT interval syndrome.

XIV. Role of Electrophysiologic Study in Patients Who Are Candidates for Implantable Electrical Devices

Application
Use of electrical devices to manage patients with tachycardia includes provision of rate support when symptomatic bradycardia results from therapy for tachycardia, e.g., sinus bradycardia as a side effect of drug administration or heart block after His bundle ablation; prevention of tachycardia onset, e.g., pacing at normal rates to treat complete AV heart block or other bradyarrhythmias that appear to cause ventricular tachyarrhythmia, pacing to suppress selected tachyarrhythmias such as torsade de pointes, pacing at short AV intervals to prevent AV node or AV reentrant tachycardias, and rapid atrial pacing to initiate an atrial tachyarrhythmia with a high degree of AV block to suppress a slower atrial tachycardia that produced a more rapid ventricular rate; and termination of tachycardia by pacing, cardioversion, or defibrillation.79

Devices
Available devices to treat patients with symptomatic bradycardias encompass the complete range of single and dual chamber pacemakers, including rate-adapting units. Pacemakers used to prevent tachycardias depend on the mechanism of the arrhythmia and range from single chamber units that deliver stimuli at normal or fast rates to more complex pacemakers that can pace the atrium and ventricle simultaneously or at short AV intervals.

Electrical devices that terminate tachycardias by pacing are capable of delivering stimuli at multiple rates and cadences, depending on the tachycardia and its rate.80,81 Because pacing for ventricular tachycardia may accelerate the tachycardia or produce ventricular flutter or ventricular fibrillation, and because the primary arrhythmia may be a rapid ventricular tachycardia that cannot be terminated by pacing, or may be ventricular flutter or ventricular fibrillation, devices must be able to both cardiovert and defibrillate.

Although a transvenous device capable of cardioverting ventricular tachycardia has been tested in humans,82 the device most widely used is the automatic implantable cardioverter-defibrillator.83 It detects arrhythmia by ventricular rate or a probability density function. Cardioversion or defibrillation is achieved by a 25 to 35 J shock usually delivered over two epicardial patches. The device does not have pacing capabilities. Although important complications have been noted,84 the arrhythmic mortality rates are very satisfactory.85 Efforts to reduce defibrillation thresholds, such as using sequential shocks, may permit implantation of smaller devices.86 Subsequent devices will combine bradycardia and competitive antitachycardia pacing, low energy synchronous cardioversion, and high energy defibrillation capabilities in one unit. Devices will be fully programmable and will deliver programmed therapy in response to the patient’s tachyarrhythmia.

Patient Evaluation
All patients being considered for treatment of a tachycardia with an electrical device must undergo thorough electrophysiologic studies preceding device implantation to determine 1) a specific mechanism and rate of the tachycardia; 2) the most appropriate site for antibradycardic stimulation; 3) the most effective stimulation sequence for termination; 4) the reproducible safety and efficacy of the therapy; 5) the potential for acceleration or induction of atrial or of ventricular tachyarrhythmias; 6) sensing and pacing characteristics during tachycardia; and 7) accurate automatic arrhythmia detection. Capability of the device to cardiovert ventricular tachycardia and defibrillate ventricular fibrillation must be demonstrated in patients with these tachyarrhythmias. Defibrillation testing must be performed in all patients with ventricular tachyarrhythmias, even in those who have never experienced an episode of spontaneous ventricular fibrillation, because electrical therapy of ventricular tachycardia may provoke ventricular fibrillation that the device must then be able to terminate. An exercise test is useful to determine the rate of the patient’s sinus tachycardia that might inappropriately trigger the device.

Recommendations for Electrophysiologic Studies

Class I
1. All patients who are candidates for implantation of an electrical device to treat arrhythmias.
2. All patients in whom such an electrical device is already implanted and in whom changes in therapy are contemplated that may influence the safety or efficacy of the device.

Class II
1. Patients with an antitachycardia device to confirm acceptable device function during follow-up.
2. Patients who receive antiarrhythmic devices to test for the most appropriate pacing site or sites and the capability of atrioventricular and ventriculoatrial conduction.

Class III. Patients who are not candidates for implantable electrical devices.

XV. Role of Electrophysiologic Study in Patients Who Are Candidates for Ablative Therapy of Cardiac Arrhythmias

Surgical management of tachycardias may be considered a reasonable option to long-term antiarrhythmic drugs (e.g., young patients with Wolff-Parkinson-White syndrome) and in some instances the best option for long-term survival (e.g., recurrent ventricular tachycardia early after acute infarction). These surgical procedures may be divided into: 1) direct procedures that involve resection or ablation of the specific sites in the heart that are responsible for the genesis of the tachyarrhythmia or constitute a critical component of the tachycardia pathway; 2) indirect procedures such as ablation of the AV node or His bundle to control the ventricular response to a variety of atrial arrhythmias; and 3) specific procedures that isolate or exclude all or part of the cardiac chamber of arrhythmia origin from the rest of the heart.87,88

The recent development of catheter ablation of the AV junction has eliminated the need for intraoperative surgical ablation of the AV junction in 85% or more of cases. Surgical resection and ablation procedures have been used to disrupt AV bypass tracts in the Wolff-Parkinson-White syndrome for almost 20 years. With use of either an epicardial or an endocardial approach to the bypass tract, a success rate in excess of 90% and close to zero mortality is expected.87–90

Electrophysiologically directed surgical procedures for drug-refractory recurrent sustained ventricular tachycardia in the setting of chronic ischemic heart disease have also evolved, resulting in freedom from recurrent tachycardias in 70% to 90% of surgical survivors with (30%) or without (70%) antiarrhythmic drugs.87,88,91–95 A more limited experience has been reported in patients with drug-resistant atrial tachycardias, AV node reentrant supraventricular tachycardia,96 atrial flutter and fibrillation, and ventricular tachycardia unrelated to ischemic heart disease, particularly that after repair of tetralogy of Fallot.87,88

Localization and Mapping of Arrhythmias

All patients undergoing surgery for recurrent arrhythmias require some form of electrophysiologic studies determined by the type of arrhythmia and the method of surgical correction. The purpose of the electrophysiologic studies is to determine the probable mechanism of the arrhythmia, the components of the circuit, and the localization of the substrate. Attempts at localization should take place both during preoperative electrophysiologic studies and at the time of surgery.87,95,97,98 Localization by catheter techniques in the electrophysiologic laboratory are necessary because 1) the arrhythmia or arrhythmias may not be inducible in the operating room, 2) only nonmappable arrhythmias may be induced intraoperatively (e.g., polymorphic ventricular tachycardia or ventricular fibrillation), 3) not all arrhythmias observed spontaneously or induced by preoperative electrophysiologic studies are induced in the operating room, 4) rapid interpretation of intraoperative data may be difficult, and 5) intraoperative catastrophes may preclude induction or mapping, or both, of arrhythmias. Intraoperative localization procedures are important to define more precisely the arrhythmogenic substrate to be approached surgically and to localize additional pathways in patients with Wolff-Parkinson-White syndrome or multiple tachycardias in patients with ventricular tachycardia.87,93,99–102

Mapping the site of origin of ventricular tachycardia may be difficult. Mapping during sinus rhythm to identify potentially arrhythmogenic areas by finding abnormal fractionated or late potentials, or both, may be inaccurate.87,93,103–105 Pace-mapping may be useful.102,106,107 Catheter and intraoperative mapping are highly accurate in identifying the site of bypass tracts in the Wolff-Parkinson-White syndrome.

Because of the limitations in accuracy and reproducibility of mapping in patients with ventricular tachycardia, different surgical techniques are utilized that do not specifically localize and resect the arrhythmogenic area but use visible anatomic landmarks to provide guidance for a more widespread surgical procedure. Such techniques include endocardial or subendocardial encircling ventriculotomy, encircling endocardial cryoablation, and total subendocardial resection.87,88,90,108 These latter techniques theoretically do not need map guiding and can be successful in selected patients. However, if possible, mapping and localization of the tachycardia are preferred because 1) approximately 15% of tachycardias do not originate from areas with fractionated electrograms found during sinus rhythm or from visible scar, 2) resection or destruction of unnecessary tissue (e.g., papillary muscles) may be avoided, and 3) anatomic landmarks to guide surgery may not be present, for example, in patients with recent myocardial infarction.

Atrial tachycardias require both catheter and intraoperative mapping. Should the tachycardia be not inducible, and therefore not mappable, one should initially consider AV junctional ablation as an alternative.

It is commonly agreed that all patients who undergo surgical therapy for arrhythmias should have postoperative electrophysiologic studies to evaluate the results of surgery.

Recommendations for Electrophysiologic Studies

Class I

1. Patients in whom antiarrhythmic surgery or other ablative procedures are contemplated.
2. Patients who have had antiarrhythmic surgery or other ablative procedures to assess efficacy of the procedure and need for further therapy.

Class II. Patients with symptoms compatible with arrhythmias having a known anatomic substrate suitable for surgical or other ablative procedures (e.g., aneurysms, postoperative tetralogy of Fallot, Wolff-Parkinson-White syndrome).

Class III. Patients with arrhythmias who are not candidates for surgical or other ablative procedures.

XVI. Role of Electrophysiologic Study in Children: Differences From Adults

The performance and interpretation of intracardiac electrophysiologic studies in children are generally similar to those in adults. The indications for electrophysiologic studies in children are also similar to the general categories in adults. However, there are areas in which there are some differences.

Need for Sedation

Small children, and even adolescents, require sedation. The electrophysiologic effects of sedation can be vagolytic (meperidine and promethazine) or sympathomimetic (ketamine). The "physiologic" conditions may change throughout a study in a child with the addition of different types of sedation as well as a different state of wakefulness. For this reason, the tests of sinus node and AV conduction as well as refractory periods of accessory connections may be less reproducible in children than in adults.

Prognostic Testing in “High Risk” Groups

Some children, such as those who have had repair of congenital heart disease, are thought to be at high risk for arrhythmic death. No randomized studies have been carried out to answer this question. Some pediatric cardiologists have advocated the use of electrophysiologic studies in an attempt to identify patients who may be at higher risk for sudden death. Additionally, children believed to have an otherwise normal heart who have ventricular tachycardia induced in the electrophysiology laboratory may also be at risk for arrhythmic death. The use of electrophysiologic studies in such patients remains controversial.

Incessant Supraventricular Tachycardia

Incessant supraventricular tachycardias may lead to cardiomyopathy that occasionally is severe enough to require cardiac transplantation. The major causes are atrial automatic tachycardia and the permanent form of junctional reciprocating tachycardia. These are relatively rare in adults but more common in children and may be confused with sinus tachycardia. In a child with congestive cardiomyopathy believed to have "sinus tachycardia," it may be important to perform electrophysiologic mapping to distinguish chronic atrial tachycardia from sinus tachycardia. Electrophysiologic studies mapping followed by surgical ablation has resulted in return of normal cardiac ablation.

Complete AV Block

Congenital complete AV block most often occurs with a narrow QRS escape rhythm. Electrophysiologic studies have not been demonstrated to be clinically useful in this situation. However, if congenital complete AV block occurs with a wide QRS escape rhythm, such studies could provide data to determine the site of block and presence of infranodal disease. Acquired complete AV block in children is considered an indication for a permanent pacemaker, and electrophysiologic studies are not necessary. Electrophysiologic studies have not been demonstrated to be beneficial in predicting prognosis in asymptomatic patients with surgically acquired bifascicular block.

Recommendations for Electrophysiologic Studies

Class I.

1. Patients similar to those described in the sections on adults.

2. Patients with an undiagnosed "narrow QRS" tachycardia that cannot be distinguished from sinus tachycardia.

Class II.

1. Patients similar to those described in the sections on adults.

2. Asymptomatic patients possibly at "high risk" for sudden arrhythmic death such as those with postoperative congenital heart disease or a normal heart with complex ventricular arrhythmias.

3. Patients with congenital complete AV block and wide QRS escape rhythm.

Class III.

1. Patients similar to those described in the sections on adults.

2. Patients with congenital complete AV block and narrow QRS escape rhythm.

3. Patients with acquired complete AV block.

4. Asymptomatic patients with surgically induced bifascicular block.

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