DURING the past decade, there has been enormous interest in the use of invasive electrophysiologic testing to evaluate patients with cardiac arrhythmias and conduction disturbances. In this editorial, we present our views on the clinical value of this procedure, ever mindful of our own biases and the knowledge that many important issues in this rapidly expanding area of cardiology remain controversial. The important contributions of invasive testing to the understanding of mechanisms of arrhythmia and drug actions remain unchallenged. Our focus is on the clinical applicability of electrophysiologic testing.

Electrophysiologic testing is used to evaluate sinus node function, atrioventricular (AV) conduction and tachyarrhythmias. The number of electrode catheters inserted depends on the nature of the study. In general, at least two catheters are required. However, for more detailed studies, up to five intracardiac catheters may be necessary. In selected patients, left ventricular stimulation may be necessary for proper evaluation.

Sinus Node Disorders

In patients with the sick sinus syndrome, an assessment of sinus node function by electrophysiologic testing is usually not necessary. The decision to implant a permanent pacemaker should be based on whether the patient has symptoms related to a bradycardia. In the majority of patients with the sick sinus syndrome, serial ambulatory electrocardiographic recordings are sufficient to determine the need for a pacemaker.

Electrophysiologic testing may be helpful in the subset of patients with electrocardiographic evidence of sinus node dysfunction who have symptoms possibly related to cerebral hypoperfusion, but never during repeated 24-hour electrocardiographic recordings. Electrophysiologic testing would be clinically valuable if it successfully predicted a positive response to permanent pacing in such patients. Measurements of sinus node recovery time (SNRT) and the sinoatrial conduction time (SACT) are used to assess sinus node function. In our experience, a markedly prolonged SNRT (> 2 seconds) may help to identify patients whose symptoms resolve with implantation of a permanent pacemaker, especially if the patient’s symptoms are replicated during postpacing pauses. Gann et al. reported that an abnormal SNRT was useful in selecting patients with chronic sinus bradycardia and dizziness or syncope for pacemaker therapy. However, the clinical significance of a mildly prolonged SNRT remains unclear. The SACT is a sensitive indicator of sinus node disease, but lacks a high degree of specificity and has been of limited value in evaluating the need for permanent pacing.

Electrophysiologic testing may be of value in patients who have asymptomatic sinus node dysfunction but who require drugs that may further depress sinus node function (e.g., antiarrhythmic drugs, sympathetic antihypertensive agents, and β-adrenergic blocking agents). If the SNRT becomes markedly prolonged (> 2 seconds) after drug administration, that drug should be used only with considerable caution or with pacemaker support.

Atrioventricular Block

Patients who have symptomatic bradyarrhythmias due to high-degree AV block should undergo implantation of a permanent pacemaker. An electrophysiologic study to evaluate the AV conduction system is not necessary in such patients. In patients with asymptomatic AV block, the decision to implant a pacemaker should depend on the level of the AV conduction system at which the block occurs. If AV block occurs at the level of the AV node, a pacemaker may not be required, whereas if the level of AV block is infranodal, a permanent pacemaker is usually indicated. The distinction between AV nodal and infranodal block can usually be made based on the morphology and rate of the escape rhythm and the response to exercise, carotid sinus massage and atropine. However, in some subgroups of patients, the level of AV block may remain unclear; for example, patients with bundle branch block who have transient episodes of AV Wenckebach conduction or high-grade AV block, patients who have AV block thought, on the basis of surface ECG recordings, to be due to concealed junctional extrasystoles, and patients with apparent Mobitz II AV block associated with narrow QRS complexes. In these subgroups of patients, electrophysiologic testing, consisting of a recording of the His bundle electrogram and evaluation of the response to atrial pacing before and after atropine, is helpful in determining the level of AV block and the need for a permanent pacemaker. In our experience, these studies often prevent the needless insertion of a permanent pacemaker.

Bundle Branch Block

Patients with chronic bundle branch block (BBB) are at increased risk of developing complete AV block. However, the overall incidence of progression to high-grade or complete AV block is quite low, especially in
young, asymptomatic patients without organic cardiac disease. Therefore, invasive studies or permanent pacemaker implantation are not indicated in these patients. In patients with BBB and transient neurologic symptoms, the symptoms may be related to episodic high-grade AV block or ventricular tachycardia. A sound clinical approach in these patients is to obtain repeated 24-hour ECGs to document whether symptoms are related to a bradyarrhythmia. Invasive studies may be of value in patients whose symptoms do not occur during monitoring. Two large prospective studies have shown that an abnormal HQ interval in patients with BBB is associated with an increased risk of progression to high-grade AV block. In our experience, although an HQ interval greater than 70 msec is associated with an increased risk of progression to high-degree AV block, this risk is still small. However, when the HQ interval is greater than 100 msec, there is a higher risk of progression (24% over 22 months). In addition, the occurrence of infranodal AV block during atrial pacing in patients with BBB is associated with a high risk of progression to high-degree AV block. Therefore, in the patient with BBB who has symptoms possibly due to cerebral hypoperfusion that is otherwise unexplained, if the HQ interval is significantly prolonged (> 100 msec), or if atrial pacing results in infranodal AV block, implantation of a permanent pacemaker may be recommended. An HQ interval of < 70 msec or the absence of infranodal block during atrial pacing may not exclude intermittent high-degree AV block as the cause of the patient’s symptoms, but does make this possibility less likely.

There is no evidence that prophylactic pacemaker insertion in symptomatic patients with prolonged infranodal conduction prolongs life or reduces the incidence of sudden death. On the contrary, the existing information from larger prospective trials suggests that the cause of sudden death in patients with BBB is related to malignant ventricular arrhythmias. Furthermore, ventricular stimulation studies in patients with syncope and BBB demonstrate a high incidence of inducible ventricular tachycardia (VT). Therefore, the clinical rule that a permanent pacemaker is indicated in patients with BBB and recurrent syncope of unknown cause is clearly untenable. These patients require detailed, complete electrophysiologic evaluation to best determine the cause of syncope.

**Supraventricular Tachycardia**

Invasive electrophysiologic testing has provided impressive new insights into the mechanisms of paroxysmal supraventricular tachycardia (PSVT). Patients with rare episodes of PSVT or those with PSVT associated with minimal symptoms may be treated using empiric drug trials. If such an approach is used, the clinician should be mindful that a significant proportion of these patients may have an accessory pathway capable of antegrade conduction during atrial fibrillation. Therefore, digitalis and i.v. verapamil should be avoided in these empiric trials because they might increase the ventricular rate if atrial fibrillation occurs. Electrophysiologic testing is indicated in the following subgroups of patients with PSVT: (1) Patients in whom tachycardia is associated with severe symptoms (e.g., angina, pulmonary edema, syncope) or marked hypotension. In these patients, electrophysiologic testing allows for rapid identification of an effective drug regimen. (2) Patients with the Wolff-Parkinson-White syndrome complicated by atrial fibrillation and a very rapid ventricular rate; after appropriate management of the acute episode, further electrophysiologic testing studies are required to assure appropriate medical or surgical treatment. (3) Patients with recurrent symptomatic bouts of PSVT who prove refractory or intolerant to drug treatment. Such patients require detailed evaluation to assess the safety and efficacy of chronic antitachycardia pacing. If cardiac electrosurgery is contemplated, detailed studies are required to define the structures that are necessary links in the tachycardia circuit.

Still controversial is the role of invasive testing in asymptomatic persons with ventricular preexcitation, especially in those who engage in vigorous sports. These people may be at increased risk of morbidity or mortality if a tachyarrhythmia occurs during strenuous exertion. A low-risk subgroup of patients with accessory pathways that have a long refractory period can be identified by abrupt normalization of QRS complexes in response to exercise testing or to a type I antiarrhythmic drug. However, available noninvasive techniques cannot detect the subgroup at high risk for development of a malignant arrhythmia.

**Wide-complex Tachycardia of Uncertain Origin**

Clues from the surface ECG are often helpful in deciding whether a wide-complex tachycardia is ventricular or supraventricular in origin. However, the surface ECG does not always provide enough information for this important distinction to be made. In such situations, electrophysiologic testing is extremely useful in clarifying the mechanism of a paroxysmal tachycardia. A majority of both recurrent ventricular and supraventricular tachycardias are probably due to continuous propagation of an impulse within a reentry circuit. Provocation of an arrhythmia by atrial or ventricular stimulation strongly suggests a reentrant mechanism. So-called triggered rhythms can also be induced by cardiac stimulation. In the majority of patients who have a spontaneous episode of VT or supraventricular tachycardia (SVT), the tachycardia can be reproduced in the electrophysiology laboratory. VT can be distinguished from SVT with aberrant conduction by noting the relationship of the His bundle and atrial electrograms to the ventricular depolarizations and by assessing the response to atrial and ventricular pacing during the tachycardia.

**Ventricular Tachycardia**

Electropharmacologic testing is a reliable technique for establishing a drug regimen effective in the long-term treatment of many patients with recurrent sustained VT. Available data indicate that VT can be
induced by programmed stimulation in approximately 90% of patients who have spontaneous episodes of sustained VT and that sustained VT (unimorphic and of relatively constant cycle length) is rarely, if ever, inducible in patients who have not had VT. In general, the results of drug testing in the electrophysiology laboratory predict the long-term response to therapy; a drug regimen that suppresses the induction of VT will usually suppress spontaneous episodes of VT, whereas drug regimens that do not suppress the induction of VT usually do not suppress spontaneous episodes of VT.

Although usually helpful in designing an effective drug regimen, electropharmacologic testing does have limitations. In the case of some drugs (e.g., amiodarone), the results of electropharmacologic testing may not predict the clinical responses. This technique is not applicable in patients who do not have inducible VT. The issue of what stimulation protocol is the most predictive of clinical response to drug therapy is unresolved. Ventricular stimulation studies expose patients to the risks of serious ventricular arrhythmias and to the potential complications of an invasive catheter study (1–2%).

In patients with recurrent sustained VT refractory to drug therapy, invasive electrophysiologic testing is indicated when antichagycardia pacing or cardiac electrosurgery is being considered for treatment.

The clinical value of electrophysiologic testing in patients with nonsustained VT (six beats to 30 seconds of VT) remains unclear. Because nonsustained VT is often a nonspecific response to programmed ventricular stimulation, especially when three extrastimuli are used (personal observations), electropharmacologic testing with an aggressive stimulation protocol in patients with nonsustained VT may not be as helpful as in patients with sustained VT. It remains to be determined whether a less aggressive stimulation protocol will be of value in formulating drug therapy in patients with nonsustained VT.

Survivors of Out-of-hospital Cardiac Arrest

Patients who survive a cardiac arrest have a high risk of sudden death. Electrophysiologic testing in these patients has demonstrated that VT or ventricular fibrillation (VF) can be induced in 29–76%, depending on the aggressiveness of the stimulation protocol. Drug therapy based on the results of electropharmacologic testing may be effective in preventing sudden death in patients who have inducible VT or VF in the control state but not while receiving specific drug therapy. Furthermore, the lack of inducible VT or VF may identify a subset of patients who do not require antiarrhythmic drug therapy and who may do well with therapy directed at the underlying heart disease, usually coronary artery disease. However, one preliminary report indicates that patients who do not have inducible VT may die suddenly despite treatment directed at their underlying heart disease. Some of the patients with spontaneous VT or VF may not have ventricular arrhythmias inducible in the laboratory. The role of reversible factors, such as ischemia, in the pathogenesis of cardiac arrest remains unclear in patients with and without inducible VT.

Survivors of a cardiac arrest should undergo a complete cardiac evaluation, including exercise testing, cardiac catheterization with coronary angiography and electrophysiologic testing. Therapy should be guided by the results of these studies. We perform electropharmacologic testing in patients who have inducible VT or VF. In our experience, using a stimulation protocol that includes right and left ventricular stimulation with triple extrastimuli, only 26% of such patients have responded to a conventional antiarrhythmic drug; therefore, the majority of patients require treatment with either experimental drugs or cardiac electrosurgery.

Electrophysiologic testing is necessary to evaluate patients with out-of-hospital cardiac arrest who are candidates for an automatic implanted defibrillator or cardiac electrosurgery. There is an alternative approach to the long-term treatment of patients with recurrent VT or VF, based on the principle that the risk of VT or VF is related to the occurrence of frequent and complex forms of ventricular ectopy. In this approach, the end point for drug testing is a decrease in the frequency of ventricular premature depolarizations (VPDs) and suppression of complex forms of VPDs. This approach has several limitations: (1) In some patients, there is no relationship between the occurrence of complex VPDs and the risk of VT, i.e., a drug may suppress VT without suppressing complex VPDs; (2) some patients with recurrent VT have very rare or no VPDs between episodes of VT; and (3) 2–3 weeks of hospitalization are often required to arrive at an effective drug regimen.

Because no prospective studies have compared the efficacy of drug therapy based on electropharmacologic testing with therapy based on suppression of VPDs, no conclusions can be drawn as to which technique is more efficacious. In our view, the limitations of the latter approach outweigh those of the former, and we therefore favor electropharmacologic testing in patients with sustained VT and in survivors of out-of-hospital cardiac arrest. The available data suggest that patients who fail to respond to a single type I agent (procainamide) will rarely (7%) respond to other conventional drugs. In addition, combinations of conventional drugs are similarly usually ineffective if the patient is unresponsive to large doses of procainamide. In view of the above, it appears reasonable to perform a single electrophysiologic study to define the subset of patients who respond to conventional drugs and avoid the potential toxicity of experimental agents. If the patient fails to respond to testing with conventional drugs, alternative therapy includes use of experimental drugs or cardiac electrosurgery. Patients in the latter category require detailed endocardial mapping studies before surgery.

Syncope

Invasive electrophysiologic studies appear to be helpful in evaluating some patients with recurrent un-
Table 1. Clinical Usefulness of Electrophysiologic Testing

<table>
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<tr>
<th>Abnormality</th>
<th>Indications for electrophysiologic testing</th>
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<td><strong>Electrophysiologic testing usually helpful</strong></td>
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<td>Wide complex tachycardia</td>
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<td>Evaluation of antitachycardia pacemaker</td>
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<tr>
<td><strong>Electrophysiologic testing sometimes helpful</strong></td>
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<tr>
<td>PSVT</td>
<td>Severe arrhythmia-related symptoms</td>
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<td>No cause found with neurologic and noninvasive cardiac evaluation</td>
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<td>both present, but cause of symptoms unclear</td>
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<td>Evaluation of drugs that may aggravate sinus node dysfunction</td>
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Abbreviations: AV = atrioventricular; PSVT = paroxysmal supraventricular tachycardia; VT = ventricular tachycardia; WPW = Wolff-Parkinson-White syndrome.

explained syncope. The available data suggest that electrophysiologic abnormalities may be found in up to 56–68% of these patients and that patients treated on the basis of these abnormalities (i.e., pacemaker or specific drug therapy) often respond with resolution of symptoms. Subjects without organic heart disease are less likely to have an abnormality demonstrated by electrophysiologic testing.

**General Comments**

Invasive electrophysiologic testing clearly adds an important new dimension in the clinical evaluation and treatment of selected patients with cardiac arrhythmias, cardiac conduction disturbances, or recurrent syncope (table 1). Several points bear emphasis. Invasive electrophysiologic testing is associated with risk, albeit small, and should be performed only by adequately trained physicians. Physicians who have completed a standard cardiology training program are usually not equipped to supervise these studies. Occasional studies performed by undertrained physicians are an invitation to disaster. The physicians performing the electrophysiologic testing must be fully cognizant of the clinical problem in order to choose the appropriate testing protocol. For appropriate decision making, the results of testing must be integrated into the patient’s total clinical picture. Finally, physicians must be aware of the limitations of electrophysiologic testing in the evaluation of various clinical problems. Only in these ways can invasive electrophysiologic testing be meaningfully applied in clinical practice.

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