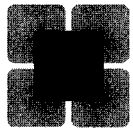

# Indications for electrophysiologic study in patients with ventricular arrhythmias

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■ Electrophysiologic study provides valuable information in the management of patients with known or suspected ventricular tachyarrhythmias. It should be used when there is uncertainty about the origin of a wide QRS tachycardia. It is useful in evaluating heart disease patients with unexplained syncope and in stratifying prognosis and guiding therapy in patients with malignant ventricular arrhythmias. All patients being considered for treatment of ventricular tachyarrhythmias with an electrical device should undergo thorough electrophysiologic study. Electrophysiologic study is also indispensable in guiding ablative therapy in patients with drug-refractory malignant ventricular arrhythmias.

□ INDEX TERMS: ARRHYTHMIA; HEART VENTRICLE ELECTROPHYSIOLOGY; HEART FUNCTION TESTS □ CLEVE CLIN J MED 1992; 59:175–185

THE ELECTROPHYSIOLOGIC study (EPS) is increasingly used in the management of patients with cardiac rhythm disorders. Because these tests require special skills to perform and interpret, they remain the province of the clinical electrophysiologist. However, internists and general cardiologists need to know the limitations of these studies, and when and when not to refer patients for EPS.

Clinical EPS involves the percutaneous insertion of one or more electrode catheters to sites in the atria, ventricles, or coronary sinus to record and stimulate at various rates. Programmed electrical stimulation (the delivery of different patterns of extrastimuli) is used to induce tachyarrhythmias. Most clinically occurring tachyarrhythmias (assumed to be of reentrant origin) can be induced and terminated by extrastimuli.[1] Intracardiac mapping—the integrated depicting of electrocardiograms (ECGs) from different sites as a function of time—is used to locate the origin and circuits of arrhythmias.[2]

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Clinically oriented EPS can be used to study the mode of initiation and termination of tachycardias, their mechanisms, site of origin, and pathways. EPS is also used to guide and evaluate therapy (drugs, devices, surgery) in patients with tachycardias; to determine an arrhythmic cause for symptoms such as palpitations, dizziness, syncope, and sudden death; and to identify individuals at risk for arrhythmic complications.[1]

Clinical EPS carries a low but tangible risk[3] and is expensive in terms of personnel and equipment; therefore, its clinical usefulness for the diagnosis and therapy of cardiac arrhythmias should be carefully considered. With that goal in mind, a joint task force of the American College of Cardiology and the American Heart Association published "Guidelines for Clinical Intracardiac Electrophysiologic Studies."[4] The guidelines divide indications for EPS into three classes. Class I conditions are those for which experts generally agree that EPS provides useful information: patients in this category should undergo EPS. Class II conditions are those in which EPS is frequently performed, but experts differ as to whether the information obtained is useful. Class III conditions are those for which experts generally agree that the EPS does not provide useful information and therefore is not warranted.

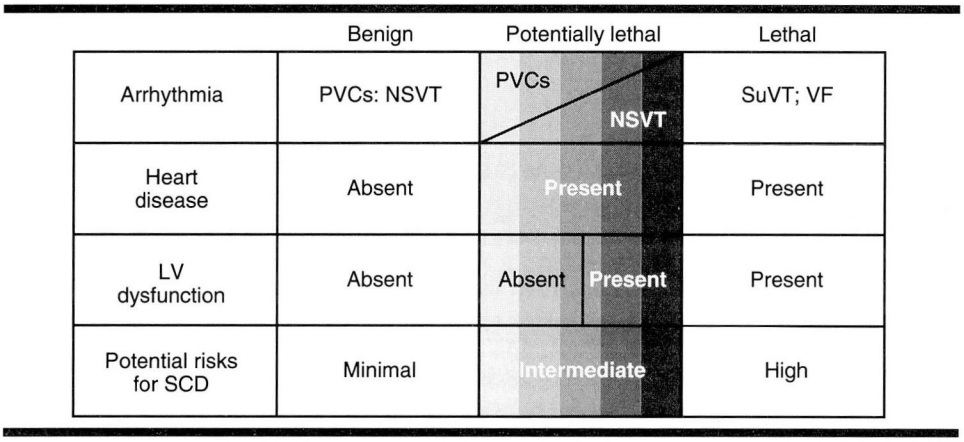


**FIGURE 1. Ventricular arrhythmias can be classified in three prognostic categories, with different therapeutic goals for each. (PVCs, premature ventricular contractions; NSVT, nonsustained ventricular tachycardia; SuVT, sustained ventricular tachycardia; VF, ventricular fibrillation; LV, left ventricular; SCD, sudden cardiac death.) Reprinted from *Am J Cardiol* (1988; 61: 102A-107A), with permission.**

In this review, we interpret and comment on these guidelines as they apply to adult patients with known or suspected ventricular arrhythmias. When the indications for EPS are debatable, we present our views. Since this is an evolving field, periodic update of these recommendations will be needed. In the discussion, it is assumed that all testing indicated by the patient's clinical state is performed by appropriately trained and qualified personnel in adequately equipped laboratories.[5]

## VENTRICULAR ARRHYTHMIAS

Although initially introduced for the study of supraventricular tachycardia[6] and bradyarrhythmias,[7] EPS rapidly found its widest application in the management of patients with ventricular arrhythmias. In our laboratories, patients with known or suspected ventricular tachyarrhythmias constitute more than 75% of the case load.

Ventricular arrhythmias are heterogeneous in terms of etiology, symptomatology, mechanisms, and electrocardiographic characteristics. It is clinically useful to classify them in three prognostic categories, each having different therapeutic goals (*Figure 1*).[8]

In patients with benign ventricular arrhythmias, most therapeutic interventions have unfavorable risk-benefit ratios; therefore, management should be conservative and aimed at suppressing intolerable symptoms. On the other hand, in patients with malignant or lethal ventricular arrhythmias, antiarrhythmic therapy is always indicated, since even aggressive approaches may have a favorable risk-benefit ratio. Patients with potentially lethal or prognostically significant ventricular arrhythmias are at intermediate risk of sudden death: for these patients, prevention of sudden death is a therapeutic goal, but at present the best management strategy for these patients is uncertain.[9,10]

As with other tests used in clinical medicine, the EPS should be interpreted through probability theory.[11] The sensitivity of a test is the likelihood of a positive test result in a person with a disease (eg, in a patient who has or will have spontaneous malignant ventricular arrhythmias). The sensitivity for induction of ventricular arrhythmias during programmed stimulation is generally higher in patients with documented sustained monomorphic ventricular tachycardia (VT) than it is in patients who present with cardiac arrest. Underlying heart disease also influences the sensitivity of programmed ventricular stimulation: patients who have a sustained VT in the setting of chronic coronary artery disease are more likely to have that arrhythmia induced (90%) during EPS than are those with no heart disease or cardiomyopathy (50%).[12–14]

The specificity of a test is the likelihood of a negative test result in a patient without disease (eg, in a patient who will not have spontaneous malignant ventricular arrhythmias). However, in the case of ventricular arrhythmias, selection of a true "control" population is problematic.[12] Most ventricular arrhythmias result from interaction between an anatomical substrate (the reentrant circuit), a trigger (ventricular premature complexes), and transient modulating factors (autonomic nervous system activity, ischemia).[15] Programmed stimulation gives an artificial trigger to patients who may have a substrate, and induction of a sustained arrhythmia in patients who also experience clinical episodes of arrhythmia can be reasonably regarded as a specific response. However, if a sustained arrhythmia is induced in a patient with heart disease but without spontaneous occurrences of the arrhythmia, only the existence of the arrhythmogenic

anatomical substrate can be assumed; in the absence of other factors, spontaneous episodes may never develop. This uncertainty about the clinical significance of induced tachyarrhythmias limits the usefulness of EPS in patients without documented ventricular arrhythmias.

The various ventricular arrhythmias that can be initiated through EPS differ in their significance.[12,13] Sustained monomorphic VT is a very specific response, since it is rarely induced in patients without structural heart disease. Nonsustained VT and polymorphic sustained VT are commonly considered nonspecific responses, more common with more aggressive stimulation protocols (see below). Ventricular fibrillation (VF) can be induced during EPS in patients with normal or abnormal hearts. The number of extrastimuli required to induce VF in the normal heart tends to be higher than in the abnormal heart, but the overlap is considerable. Although some investigators consider induction of VF a nonspecific finding, we believe that under some circumstances it may have prognostic significance.[16]

Factors related to the aggressiveness of the stimulation protocol affect its sensitivity and specificity.[14] These include the number of extrastimuli delivered, the number of ventricular stimulation sites, the drive cycle lengths at which extrastimuli are introduced, the strength of the electric current, and the use of modulating interventions such as isoproterenol infusion.

Increased sensitivity can only be achieved at the cost of decreased specificity (*Figure 2*).[4] We agree that a good compromise in most clinical situations is achieved by a protocol with a maximum of three extrastimuli delivered at three different cycle lengths from two different right ventricular sites at an intensity of twice the threshold.[17]

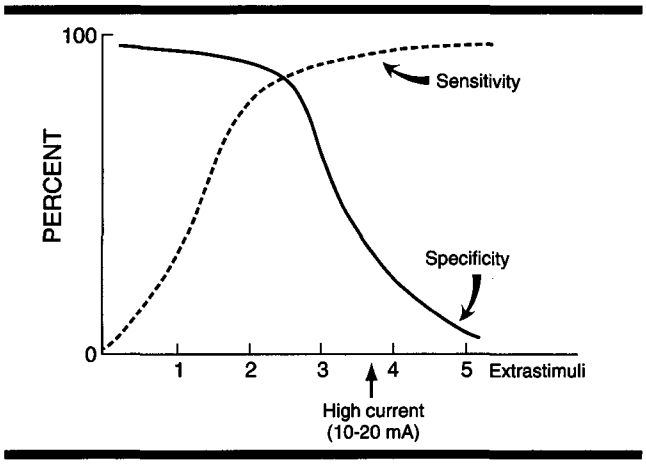


**FIGURE 2. Idealized representation of the sensitivity and specificity of programmed electrical stimulation for ventricular tachycardia. Reproduced from Gottlieb et al, with permission.[14]**

## WIDE QRS COMPLEX TACHYCARDIAS

A regular wide (≥120-millisecond) QRS complex may be present during either supraventricular tachycardia or VT, and it is important for therapeutic and prognostic reasons to make the correct diagnosis. The following possibilities must be considered in approaching the diagnosis: (1) VT; (2) supraventricular tachycardia with bundle branch block; (3) preexcited reciprocating tachycardia with anterograde conduction over an accessory pathway and retrograde conduction over the His bundle-atrioventricular node (true antidromic) or, more commonly, over a second accessory pathway[18,19]; (4) supraventricular tachycardia (eg, atrial flutter) with atrioventricular conduction over an accessory pathway, which acts as an "innocent bystander"; and (5) tachycardia with anterograde conduction over a Mahaim (nodoventricular) fiber.

A 12-lead ECG taken during tachycardia and after its termination helps to define the mechanism of the tachycardia. The relationship between ventricular and atrial rhythm and the width, axis, and morphologic characteristics of the QRS complex should be examined carefully.[20] Information from the ECG on sinus rhythm (old myocardial infarction, preexistent bundle branch block, accessory pathway) is also important. Additional clues are obtained from the patient's history (previous myocardial infarction strongly suggests VT).[21] Administering intravenous adenosine during the tachycardia may aid in the diagnosis.[22]

Analysis of the ECG has limitations. A preexisting intraventricular conduction disturbance can render the interpretation of many of the morphologic criteria of VT susceptible to error. Likewise, antiarrhythmic drugs that slow conduction may make aberrantly conducted beats seem indicative of VT. If preexcitation is latent or subtle during sinus rhythm, the diagnosis of preexcited tachycardia may be impossible when all other criteria favor VT.[23]

Although retrospective studies suggest that a correct diagnosis from the ECG can be made about 90% of the time, this may not be so in the clinical setting. Single-lead strips from a bedside or telemetric monitor, instead of a good-quality 12-lead ECG, are frequently the only recordings available. Furthermore, even highly skilled

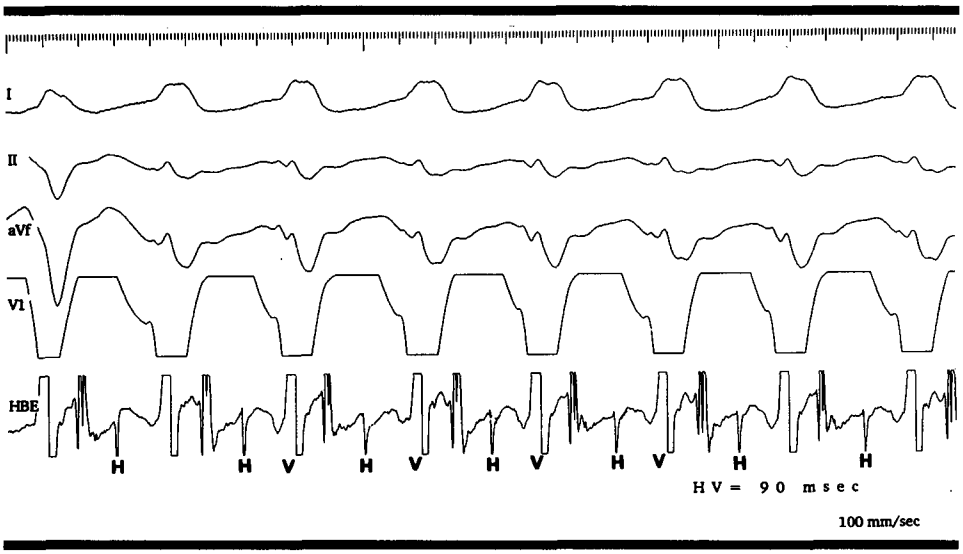


**FIGURE 3. Wide-complex tachycardia induced during electrophysiologic study. Tracings represent four surface electrocardiograph leads and a bipolar His bundle electrogram (HBE). A His deflection (H) precedes each ventricular depolarization (V). The HV interval is 90 milliseconds. The findings are typical of supraventricular tachycardia with aberrancy, in this case atrioventricular reentry using a concealed accessory pathway.**

electrocardiographers may at times disagree on the diagnosis from the 12-lead ECG.[23] In our experience, VT is often misinterpreted as supraventricular tachycardia by internists and well-trained cardiologists. The consequences of this misdiagnosis may be disastrous.[24] When the diagnosis is uncertain, it is safer to consider wide-complex tachycardias to be ventricular in origin until proven otherwise.

The EPS remains the gold standard for the differential diagnosis of wide QRS complex tachycardias. If the clinically occurring paroxysmal arrhythmia is inducible, extensive analysis of the rhythm can be performed. During aberrant conduction supraventricular impulses must travel over the His bundle and reach the ventricle; therefore, the His deflection precedes the QRS complex with a normal or prolonged HV (His bundle to ventricle) interval (*Figure 3*). But during VT the activation of the His bundle usually occurs in the retrograde direction, and the His potential is obscured within the corresponding ventricular ECG or follows the onset of the QRS (*Figure 4*). Differentiating ventricular from preexcited tachycardias requires analysis of the timing and sequence of atrial activation patterns and responses to programmed stimulation during tachycardia.

How often the EPS is needed to diagnose the origin of a wide-complex tachycardia varies according to the expertise of the attending physician and the availability of good-quality recordings. EPS should be liberally used whenever the clinical diagnosis is uncertain. When the clinical diagnosis is clear, EPS may still be needed for therapeutic purposes (*Figure 5*).

## VPC, COUPLETS, AND NONSUSTAINED VT

Ventricular ectopy, either asymptomatic or associated with minor symptoms, is a frequent finding in patients with or without organic heart disease. Complex (frequent or repetitive) ventricular arrhythmia in the context of coronary artery disease and left ventricular dysfunction is associated with increased risk of sudden death.[25] Although these arrhythmias often have been treated empirically in order to improve prognosis,[26] the available data do not support this practice. In fact, as documented by the Cardiac Arrhythmia Suppression Trial, flecainide and encainide treatment of asymptomatic ventricular arrhythmias after a myocardial infarction may be harmful.[27]

It is widely accepted to arbitrarily call induced VTs "sustained" if they last more than 30 seconds or need to be terminated sooner than that due to hemodynamic compromise. Spontaneous nonsustained VTs (during Holter monitoring) are generally short ( 3 to 10 beats). Longer runs are relatively uncommon and are more often accompanied by symptoms.[28] It is our perception that these longer runs carry a worse prognosis, especially when they are monomorphic, and should be addressed in the same way as sustained VT.

Patients with potentially lethal ventricular arrhythmias do not all have the same risk for sudden death (*Figure 1*). Both noninvasive[27] and invasive[30–34] techniques have been used in attempts to stratify these patients into prognostic groups. One approach uses EPS to distinguish patients with inducible VT (25% to 50% in different studies) from those without inducible VT. Unfortunately, most of these studies have been retrospective, the patient populations have been

heterogeneous, and definitions of inducibility have varied among the studies. Most investigators have found that patients without inducible VT have a very low risk for sudden death, even when they are not treated with antiarrhythmic drugs. However, patients with inducible VT have almost invariably received antiarrhythmic drugs, so the stratification scheme has not been entirely validated. Ethical concerns are an obstacle to validation, since it may not be desirable to assign to a control group patients who are felt to be at high-risk. Studies with random allocation of these patients to either medical treatment or defibrillator implantation would be ethical and would provide valuable information about prognosis and best treatment of this population.

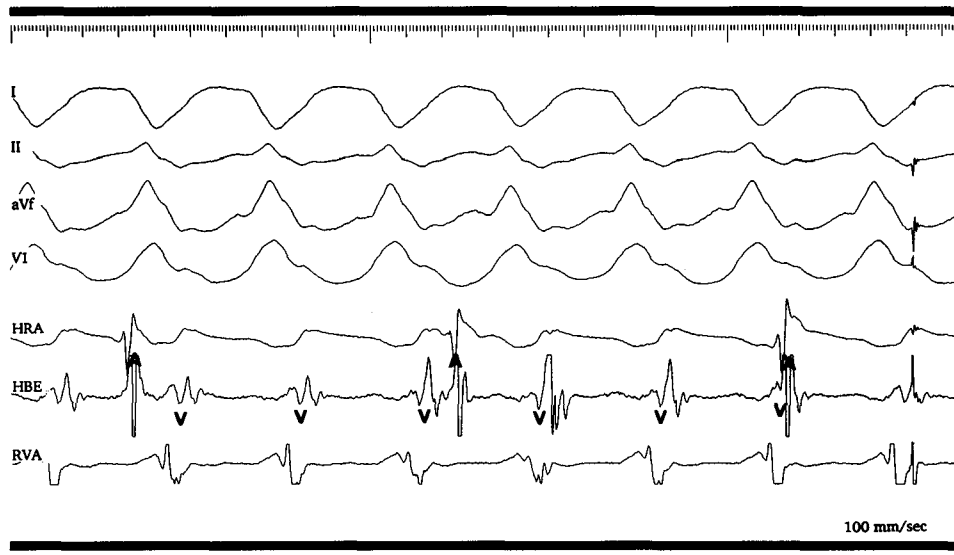


**FIGURE 4. Wide-complex tachycardia induced during electrophysiologic study. Tracings represent four surface electrocardiograph leads, and high right atrium (HRA), His bundle (HBE) and right ventricular apex (RVA) bipolar electrograms. Note the absence of His deflections and the AV dissociation. These findings are almost pathognomonic of ventricular tachycardia.**

At present, EPS can not be definitely recommended in asymptomatic patients with ventricular ectopy.[35] Available data suggest that it may be useful in patients with nonsustained VT, impaired left ventricular function, and late potentials in the signal-averaged ECG. Patients in this group without inducible VT probably do not need antiarrhythmic therapy, but it seems appropriate to attempt pharmacologic antiarrhythmic therapy if the patient has inducible VT. There are no data yet to justify the use of nonpharmacologic therapy in these patients[36] (*Figure* 6).

## UNEXPLAINED SYNCOPE

Syncope and near-syncope are frequent medical problems with a myriad of etiologies. Despite detailed histories and physical examinations, the etiology remains obscure in about 50% of cases.[37]

Cardiac syncope in particular carries a serious prognosis, with a 20% incidence of sudden death in the follow-up. Sinus node dysfunction, atrioventricular block, supraventricular tachycardia, and VT are common arrhythmic causes of syncope. Their identification is important because their outcome is ominous if they are left untreated, and because potentially salutary therapy is available. Recording the arrhythmia during a symptomatic episode gives a definitive diagnosis for that spell, but because the symptoms are intermittent, 24-hour ambulatory records are generally unrewarding and provocative testing is frequently necessary. EPS can often initiate clinically important arrhythmias and so is useful in the evaluation of patients with unexplained syncope.[38]

EPS should be comprehensive and aimed at detecting a variety of bradyarrhythmias and tachyarrhythmias. VT is the most commonly induced arrhythmia in this population. The sensitivity and specificity of the stimulation protocol and the characteristics of the induced arrhythmia are critical for the interpretation of the findings. In general, induction of a sustained VT should be regarded as diagnostic.[38,39]

An arrhythmia is more likely to be the cause of syncope in patients with known or suspected heart disease. A 12-lead ECG showing old myocardial infarction, bundle branch block, ventricular premature complexes, or a signal-averaged ECG showing late potentials may be useful clues. Conversely, in patients without structural heart disease and with normal ECGs, the diagnostic yield of EPS is low.[40] Tilt-table testing is frequently diagnostic in these patients.[41] We believe EPS should be performed in patients with syn-

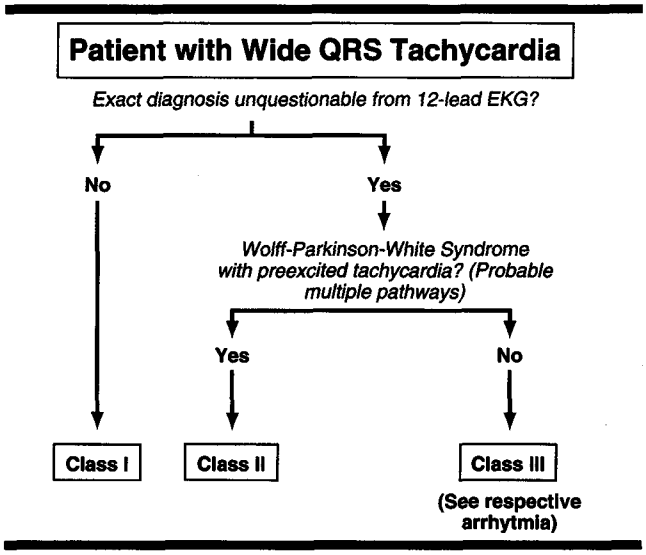


FIGURE 5. Algorithm describing indications for electrophysiologic study in patients with wide QRS tachycardias.

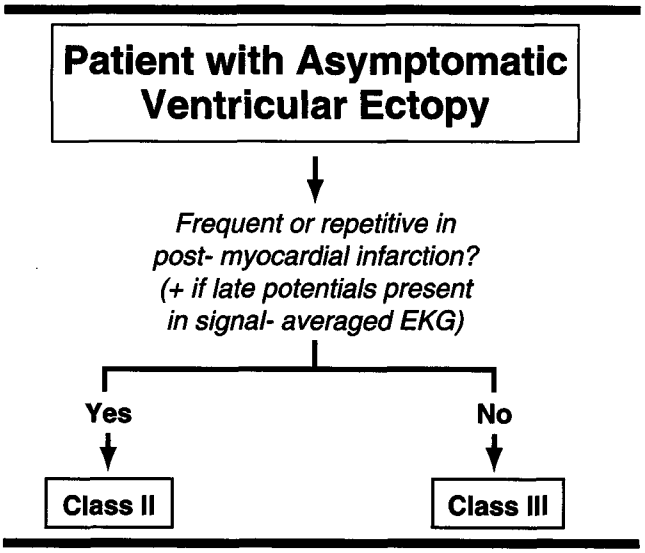


FIGURE 6. Algorithm describing indications for electrophysiologic study in patients with asymptomatic ventricular ectopy.

cope and without structural heart disease only after nondiagnostic tilt-table testing (*Figure 7*). Patients with no abnormalities noted during EPS have a low incidence of sudden death during follow-up; this suggests that a negative test result may have prognostic value.[38]

## CARDIAC ARREST SURVIVORS

Although ventricular tachyarrhythmia is generally the final cause of out-of-hospital cardiac arrest, its mechanisms vary greatly.[15,42,43] Most patients who are resuscitated from such episodes have severe coronary artery disease but not an acute Q-wave myocardial infarction. For these patients, the 1- to 2-year recurrence for cardiac arrest is 20% to 30% in the absence of specific therapy.[44]

EPS is useful in stratifying prognosis and guiding therapy in these patients[45] (*Figure 8*). In about 50% of patients a sustained monomorphic VT is induced, reflecting the existence of a fixed arrhythmogenic substrate and the need for specific antiarrhythmic therapy. Serial electrophysiologic testing is the preferred mode to guide therapy in these patients (see below). Induction of sustained polymorphic VT or VF in these patients (20%) may also be a specific finding, but the role of serial testing in guiding therapy is not so clear in those cases.

In patients with no inducible ventricular tachyarrhythmias, the prognosis varies according to left ventricular function.[46,47] If left ventricular function is preserved, the prognosis is excellent when therapy is directed solely to the underlying cause (eg, myocardial revascularization). On the other hand, patients with no inducible ventricular tachyarrhythmias and depressed ventricular function (for instance, those with cardiomyopathy) have a significant risk of recurrent cardiac arrest. In the absence of an acceptable end point to guide therapy, implantable cardioverter-defibrillators may be the best alternative for this population.

In many patients, the first episode of cardiac arrest or sustained VT occurs while they are receiving antiarrhythmic drug therapy to suppress ventricular ectopy or atrial tachyarrhythmias. In these cases, cardiac arrest could have resulted either from failure of the drug therapy or because of proarrhythmic response to the drug. The distinction can seldom be made on noninvasive grounds. EPS may be helpful in these situations. In many patients, ventricular tachyarrhythmias are induced in the absence of antiarrhythmic drugs,[48] suggesting a spontaneous tendency to ventricular arrhythmias and a probable need for antiarrhythmic therapy. Failure to induce ventricular tachyarrhythmias after withdrawal of the antiarrhythmic drug and inducibility with its reintroduction suggest that the clinical arrhythmia was exacerbated or provoked by the drug.[49]

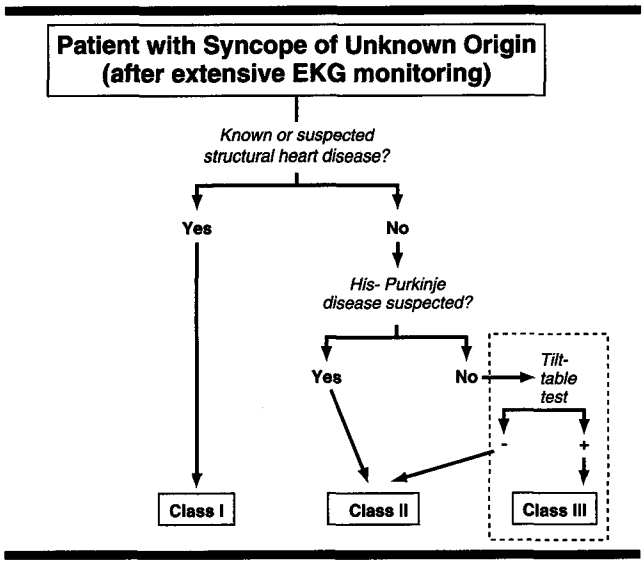


FIGURE 7. Algorithm describing indications for electrophysiologic study in patients with syncope of unknown origin. Dashed insert shows our preferred approach to patients without evidence of structural heart disease.

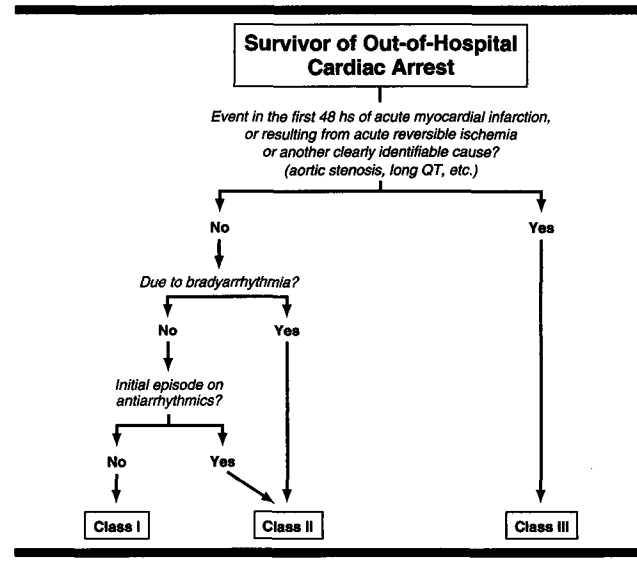


FIGURE 8. Algorithm describing indications for electrophysiologic study in survivors of cardiac arrest.

## GUIDING DRUG THERAPY

The management of patients with malignant ventricular arrhythmias is a delicate task requiring rapid and reliable identification of effective therapy: the trial-and-error approach used in more benign conditions has no place here. Although some preliminary data from Europe[50] suggest that a good outcome can be achieved with empiric therapy (eg, antiarrhythmic drugs prescribed solely on clinical grounds), we believe that some objective measurement of efficacy is crucial to optimize therapy in this population. Most of the study of EPS-guided therapy has been done in patients with chronic ischemic heart disease; the role of EPS in ventricular tachyarrhythmias of nonischemic etiology (eg, idiopathic cardiomyopathy) is not known, and should be the focus of future research.

Noninvasive techniques (combining Holter monitoring and stress tests)[51] and EPS[13] are the two approaches which are extensively used to guide therapy in patients with malignant ventricular arrhythmias. When noninvasive techniques are used, it is assumed that suppression of ambient ventricular ectopy with drugs reflects modifications in the underlying electrical instability which reduce the likelihood of recurrent arrhythmias and sudden death. Similarly, when using EPS it is assumed that antiarrhythmic agents which prevent induction of a previously inducible VT will prevent clinical recurrences of VT, and that antiarrhythmic agents which fail to prevent induction will also fail to prevent clinical recurrences.

Serial EPS technique differs from institution to institution. Although testing after several doses of oral drug will better mimic chronic treatment, frequently one or two drugs are tested acutely after inducibility of VT is demonstrated during baseline. An indwelling electrode-catheter may be left in place for several days during the testing protocol, or a new venous puncture may be performed each time a new drug is tested. Criteria for a positive response vary: less than 15 beats or 5 beats of induced VT are accepted by different groups.[13,14]

The assumptions which are the basis for noninvasive and electrophysiologically guided therapies are not strictly true, so these approaches have limitations.[52] Their relative merits are the subject of current research.[53]

More patients (90%) are candidates for electrophysiologically guided therapy than for Holter monitoring. About 40% of patients do not present with enough ambient ventricular ectopy to allow assessing changes after therapy; in these cases, the consensus is that EPS is the method of choice to guide therapy.[4]

When applicable, noninvasive techniques identify a potentially effective drug regimen in 70% to 80% of

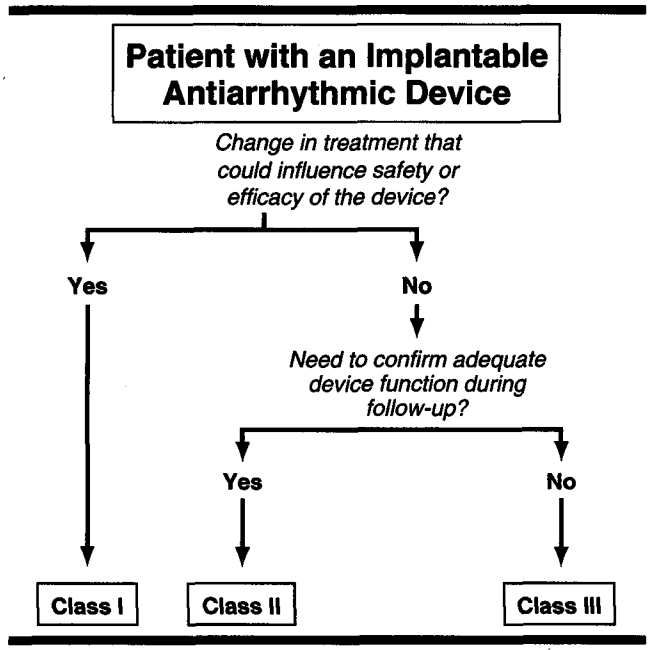


**FIGURE 9. Algorithm describing indications for electrophysiologic study in patients with antiarrhythmic devices**

patients. The approach is specific, but rather insensitive: many patients will have a clinical recurrence on therapy which was predicted to be effective. Conversely, EPS is sensitive for detecting drug failures, but less specific: a considerable number of patients will do well if discharged on drugs which were predicted to be ineffective. As alternative nonpharmacological therapies with relatively low morbidity become increasingly available for these patients, it may be preferable, cost considerations aside, to err in overpredicting drug failures.

The proportion of patients in whom an effective drug is identified through EPS depends on characteristics of the population being tested, such as the number of previous failed empiric drug trials and the severity of heart disease, and also depends on the criteria for a positive response and the number of drugs tested.[13] The goal of serial EPS should be to render VT noninducible, since this correlates with a good prognosis (10% recurrence of VT or sudden death at 1 to 2 years). Nevertheless, other responses also may be beneficial, especially with drugs like amiodarone. Patients in whom VT remains inducible, but with a slower rate and without hemodynamic compromise, are also protected from sudden death. They may still be at risk for nonfatal recurrences of VT, but this may be an acceptable end point of therapy.[54]

As more drugs are tested, the probability increases of finding an effective one. Currently in the United States 25% to 40% of patients become "noninducible," but when as many as 11 drug trials per patient are used, a 70% response rate can be achieved.[55] However, of these, 70% become noninducible after the first two trials, and after failure of five drugs the yield of subsequent tests is negligible. The "appropriate" number of EPSs in each patient should be defined individually, depending on clinical characteristics, the risk and expected benefit of alternative approaches, and local institutional idiosyncrasies. We believe that two to three drug trials represent a reasonable compromise in most circumstances.

Our present approach to therapy in patients with malignant ventricular arrhythmias is as follows: When sustained VT is inducible on baseline, noninducibility under drugs is the end point of therapy. In patients with well-tolerated arrhythmias, we accept slowing of the VT by amiodarone as an end point. We are reluctant to guide therapy solely by Holter monitoring in patients without inducible VT, in whom we would use an implantable cardioverter-defibrillator, unless they are felt clinically to be at low risk. However, we believe that noninvasive techniques have an adjuvant role in guiding therapy. It is uncommon for a drug to be effective at EPS without suppressing ambient ectopy, so Holter monitoring can be used to screen for drugs which merit EPS, thus minimizing the number of studies performed.[52] Furthermore, exercise stress tests are useful to detect proarrhythmic responses that are not disclosed with other methods.[56]

## IMPLANTABLE ELECTRICAL DEVICES

There is an increased use of implantable electrical devices capable of automatically detecting and terminating ventricular tachyarrhythmias. High-energy (about 30 J) internal defibrillation is the only available modality capable of terminating both VT and VF, and so it has been most widely used.[5] Pacing and low-energy cardioversion can terminate VT almost imperceptibly, but they can also accelerate it or cause it to degenerate into VF. Consequently, these modalities require back-up defibrillation. This has been achieved with the combination of separate antitachycardia pacemakers and defibrillators.[58] More recently, single units capable of delivering tiered therapy have become available.[59]

To determine the efficacy, safety, and reproducibility of the intended therapy, all patients being considered

for treatment of ventricular tachyarrhythmias with an electrical device must undergo thorough EPS.

Intraoperative EPS to determine the defibrillation threshold (the minimum amount of electrical energy likely to provide successful ventricular defibrillation) is crucial during defibrillator implantation.[60] Every effort should be made to achieve a margin of safety of at least 10 J between the defibrillation threshold and the maximum output of the device.[61] Defibrillation testing must be performed in all patients with ventricular tachyarrhythmias, even in those presenting clinically with VT alone, because electrical therapy of VT may provoke VF that the device must then be able to terminate. Since energy levels that terminate VF are invariably effective in terminating VT, specific testing of the ability of defibrillators to convert VT is generally not required. Finally, correct sensing of VF should also be assured.

The use of pacing, low-energy cardioversion, or both in patients with ventricular tachyarrhythmias requires meticulous testing. This can be done before or at the time of implantation, or during the early post-implantation phase. The safest, most effective mode of stimulation for each type of tachycardia that occurs must be determined, and the most effective detection algorithm should be sought. With devices that have extensive programmable capabilities, these steps are time-consuming, requiring multiple inductions and terminations of the tachyarrhythmia. If defibrillation backup is provided by a separate unit, specific testing must be done to rule out deleterious device-to-device interactions.[58,62]

All the EPS should be done while the patient receives the intended chronic antiarrhythmic drug regimen, because drugs can affect the efficacy and safety of the devices by increasing the defibrillation threshold or slowing a tachycardia to a rate below the programmed detection rate.[63] Consequently, patients with an electrical device already implanted and in whom changes in therapy that may influence the safety or efficacy of the device are contemplated also should undergo repeated EPS (*Figure 9*).

There is some debate over whether all patients who receive an implantable cardioverter-defibrillator that worked correctly at the time of surgery need to undergo repeated EPS before being discharged. We and others[64] have found that about 5% of these patients will not be successfully defibrillated at the time of predischarge testing and will require revision of the defibrillation lead system. We recommend routine predischarge testing of defibrillator function in all patients.

## EPS IN ABLATIVE THERAPY

Most clinically occurring recurrent sustained VTs (eg, in coronary heart disease) arise from discrete foci where reentrant circuits may develop. These regions are generally located in the border zone between healthy and scarred myocardium. Ablative therapies directed to those substrates are rational, and, when appropriately indicated, may be the best alternative available.[65]

Surgical ablative procedures for VT include direct resection or ablation of specific sites responsible for the genesis of tachyarrhythmias (endocardial resection, cryoablation) and indirect procedures that isolate or exclude all or part of the chamber of arrhythmia origin from the rest of the heart (encircling endocardial ventriculotomy, disconnection of the right ventricular free wall). Percutaneous VT ablation (eg, the delivery of high-energy electrical shocks, radiofrequency, or laser energy through intravascular catheters to specific areas of the heart) is also considered a direct procedure.[66]

Successful mapping of the tachycardia(s) to be ablated is crucial to these approaches. Although some visually or anatomically guided resection techniques[67] do not rely on mapping information, map-guided approaches should be used whenever possible. In this way unnecessary resection or destruction of tissue may be avoided; furthermore, anatomic landmarks to guide surgery may not be present in some patients–for instance, those with recent myocardial infarction.

Candidates for surgical treatment of VT should first have their arrhythmias mapped during preoperative EPS. Epicardial and endocardial (after opening of the heart) intraoperative localization procedures are then used to define the arrhythmogenic substrate more precisely.[2]

It is necessary to localize the arrhythmias by catheter techniques in the electrophysiologic laboratory, because the effects of anesthetics, hypothermia, and extracorporeal circulation may render some of the clinically relevant arrhythmias noninducible in the operating room. When this happens, the ablation procedures must be targeted to areas defined by the preoperative EPS. There is no intraoperative mapping during catheter ablation procedures, so the information acquired through preoperative catheter mapping is even more crucial.[68]

Catheter mapping of VT is demanding and time-consuming. The arrhythmias must be induced repeatedly, and, as long as they are safely tolerated by

the patient, the mapping catheter is moved along the ventricles to find the site of earliest depolarization. It may be impossible to map very fast VTs.[68] All different VT morphologies must be mapped, but patients with more than two different VTs are not good candidates for ablative procedures.[69] Pace-mapping (trying to emulate the morphology of VT through pacing different ventricular endocardial sites) is a useful adjuvant.[70]

All patients who undergo ablative therapy for VT should have postoperative EPS to evaluate the results of the procedure and to assess the need for further therapy.[4]

## SUMMARY

When appropriately performed, EPS is very useful in the management of patients with known or suspected ventricular tachyarrhythmias. EPS should be used when there is uncertainty about the origin of a wide QRS complex tachycardia. In patients with heart disease, VT can often be unmasked as the cause of syncope. EPS is useful in stratifying prognosis and guiding therapy in patients with malignant ventricular arrhythmias. Finally, EPS is indispensable whenever ablative therapy or use of an electrical device is contemplated in patients with malignant drug-refractory ventricular arrhythmias.

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