Electrophysiologic Testing in the Diagnosis and Management of Cardiac Arrhythmias

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Electrophysiologic testing has become an established procedure in cardiology which has markedly expanded our understanding of the mechanisms of cardiac arrhythmias. In the past decade, extra-stimulation pacing techniques, in conjunction with the intracardiac recordings, have been used as a more objective means to assess therapeutic efficacy, to determine whether medical treatment will be useful, whether pacemaker implantation is necessary, and whether surgical treatment would be curative. These studies are presently under investigation for the identification of patients at high risk of sudden death and to diagnose the etiology of syncope.

There has been a large number of review articles and symposia in the recent literature discussing the clinical and research applications of electrophysiologic testing. This article will attempt to review the most frequently encountered clinical applications of these techniques, with discussions of clinical situations in which they may be useful. On the other hand, there are several circumstances in which these techniques have limited value, and the standard electrocardiographic tools are sufficient. The purpose of this review will therefore be to develop some guidelines for the clinician to permit a logical approach to the analysis and management of arrhythmias.

Bradyarrhythmias

Disorders of Sinus Impulse Formation and Conduction

Abnormal sinus node function must be differentiated from physiologic sinus bradycardia and from disorders of the autonomic nervous system, such as carotid sinus hypersensitivity. The term "sick sinus syndrome" is a commonly used term, but is inappropriate, since it probably incorporates a variety of etiologies and electrophysiologic mechanisms. These arrhythmias are most frequently encountered in elderly patients as an idiopathic degenerative process, or in association with chronic hypertension, cardiomyopathy, or ischemic heart disease. The surface ECG may reveal an inappropriate sinus bradycardia, sinus exit block, or paroxysms of supraventricular tachycardia (classically, atrial flutter or fibrillation) followed by prolonged suppression of subsidiary escape pacemaker rhythm.

The electrophysiologic evaluations of sinus node function that are in clinical use include the measurement of sinoatrial conduction time (SACT), sinus node recovery time (SNRT), and the effect of parasympathetic stimulation (such as carotid sinus pressure) on sinus and AV nodal function. Direct catheter electrode recordings of the sinus node have been recently developed and applied to human studies but are presently in investigative stages. The usefulness of invasive testing for suspected sinus node dysfunction or sick sinus syndrome is subject to some controversy, since these arrhythmias are probably best detected by ambulatory recordings. The decision regarding pacemaker therapy should be largely based upon the correlation of the patient's symptoms at the time of the bradyarrhythmia. Complete electrophysiologic studies, incorporating an evaluation of the cardiac conduction as well as assessing the propensity toward tachyarrhythmias, should be performed in patients with suspected but undocumented cardiac syncope.

Atrioventricular Conduction Disturbances

One of the earliest clinical applications of intracardiac electrograms was the study of AV nodal and His-Purkinje conduction. The surface P-R interval can be subdivided into AV nodal (AH) and His-ventricular (HV) intervals. Thus, a prolonged P-R interval on the surface ECG may represent conduction delay at any point along the conduction system from intra-atrial delay proximally, to delay in the bundle branch system distally. The normal AH interval (50 to 130 msec) is an approximation of AV nodal conduction since it is measuring the conduction time from the low right atrial activation to proximal His bundle. It is strongly influenced by changes in the autonomic nervous system. First or second degree AV block (Wenckebach) on
the resting ECG is occasionally seen in the normal population, due to increased parasympathetic tone. The HV interval remains constant and is independent of autonomic influences.

Incremental atrial pacing, usually pacing the high right atrium or coronary sinus, is a routine part of electrophysiologic evaluation of the conduction system. The atrial paced rate at which AV nodal (AH) Wenckebach is observed in the adult population, is usually 130 to 180 per minute. If HV prolongation or infra-His block develops in response to rapid atrial pacing, permanent pacemaker implantation may be considered. Such a response connotes His-Purkinje disease and is associated with the development of complete AV block.

Programmed atrial extrastimulation techniques are also routinely performed to evaluate AV nodal and His-Purkinje conduction and refractoriness. In the normal situation, the refractory periods of the AV nodal or atrial tissue exceed that of the His bundle. However, there are certain situations in which infra-His block may also be a normal finding, the most common examples being in the Wolff-Parkinson-White syndrome and accelerated AV conduction (referred to as the Lown-Ganong-Levine syndrome). This phenomenon will be discussed in detail under supraventricular tachycardias.

In general, first, second (Wenckebach), and third degree AV block on the surface ECG with a narrow QRS can be ascribed to AV nodal delay or block (AH) and do not warrant invasive electrophysiologic studies. Similarly, congenital complete AV block occurs within the AV node and further studies are generally not required. The decision regarding pacemaker insertion must be made in the context of the clinical circumstances and the symptomatology.

The presence of atrioventricular block in association with bundle branch block lessens the predictability of the site of block from surface ECG analysis. Data from a large series of patients with chronic bifascicular block

![Figure 1. Demonstration of intra- and infra-His disease. Surface ECG leads, AVF, V1, HRA, HBE, RVA, LV, and T, are recorded simultaneously with intracardiac recordings from high right atrium (HRA), His bundle (HBE), right ventricular apex (RVA), and left ventricle (LV). In the top panel, a single premature atrial extrastimulus is introduced 300 msec following a drive cycle length of 500 msec. During the drive cycle length, two His spikes are seen prior to the first QRS. The second atrial stimulus is followed by only one H, diagnostic of infra-His block. The same phenomenon is seen following the premature atrial depolarization. The H is seen to move slightly away from the HBE-A, demonstrating that the H spike is not part of a fractionated atrial electrogram. Conduction of the premature atrial depolarization to the ventricle in the bottom panel is seen, despite an earlier premature beat (coupling interval 290 msec). The premature beat is conducted to the ventricle in this case because the previous complex was blocked infra-His.](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21379/)
indicate that 50 percent to 60 percent may have prolonged HV intervals (>55 msec) regardless of the length of the PR interval. Abnormal HV intervals may occasionally be seen in the presence of a narrow QRS; about 25 percent of patients with RBBB will have a long HV and as many as 60 percent of patients with LBBB. When bifascicular block is present (RBBB + LAFB, RBBB + LPFB), the likelihood of finding intra-His or infra-His disease (diagnostic of trifascicular disease) is greater than 50 percent (Fig 1). The prognostic value of the HV interval in patients with bifascicular block is an unsettled and controversial issue. Recent data from Dhingra et al suggests that the patients with prolonged HV intervals have an increased incidence of complete AV block and spontaneous trifascicular block. These patients have a significantly higher incidence of sudden death and overall cardiovascular mortality when compared to a group with bifascicular block with normal HV intervals. There is a strong association of prolonged HV interval with the severity of organic heart disease. Therefore, the significance of the HV interval cannot be analyzed as an independent predictor of the development of AV block or sudden death. Moreover, it has been suggested that the mechanism of sudden death in this group of patients may be due to ventricular tachycardia or fibrillation, which would not be prevented by pacemaker insertion.

At present, electrophysiologic studies in patients with bifascicular block are of greatest utility as part of the evaluation of syncope. Asymptomatic patients do not warrant invasive studies or prophylactic pacemaker insertion. If a patient has electrophysiologic findings, pacemaker therapy is recommended if second or third degree intra-His (split H potentials) block is present, or if the H-V interval is greater than 90 to 100 msec. The practice in our laboratory has been to routinely perform a ventricular stimulation protocol in patients with bifascicular block and a history of dizziness or syncope, since the initiation of ventricular tachycardia or fibrillation would also warrant aggressive treatment.

Atrioventricular and/or intraventricular conduction disturbances that appear during acute myocardial infarction are generally treated with temporary pacemaker insertion. While new bundle branch block or bifascicular block with acute infarction is associated with a higher mortality, permanent pacemaker implantation marginally affects prognosis, since most patients die of severe myocardial failure or ventricular arrhythmias. Most of the data regarding conduction disturbances following acute infarction are based upon surface ECG findings. The role of electrophysiologic studies in this setting is not clearly established except for diagnostic purposes to determine the site of block.

**SUPRAVENTRICULAR TACHYCARDIA**

Clinical electrophysiologic techniques have greatly expanded our understanding of the mechanisms and site of origin of supraventricular tachycardias. These tachycardias may now be managed more specifically, according to their type. Supraventricular tachycardias may be classified as atrial (e.g., atrial flutter, fibrillation, paroxysmal or nonparoxysmal atrial tachycardia), or a

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**Figure 2. Mechanism of AV nodal reentrant SVT.** The atrium, two pathways (α and β) within the AV node, and His-Purkinje system are shown schematically. In the left panel, during sinus rhythm, the sinus impulse is conducted via the faster (β) pathway, appearing on the surface ECG as a normal PR interval. In the middle panel, an atrial premature depolarization blocks antegrade in the fast (β) pathway due to its longer refractory period, but is conducted instead via the slower (α) pathway. If the previously refractory beta pathway has recovered (middle panel), the impulse will travel retrogradely, producing an atrial echo. Sustained SVT will result if the alpha pathway has sufficiently recovered once the impulse has traveled the full circuit (right panel). Thus, the ability to initiate and sustain the SVT is dependent upon a critical balance of conduction and refractoriness of both pathways. (Adapted from Josephson ME. Supraventricular tachycardia: mechanisms and management. Ann Intern Med 1977; 87:350).
reentrant tachycardia incorporating the AV node, (such as AV nodal reentrant tachycardia or accessory pathways).

The most common type of supraventricular tachycardia is due to reentry within the AV node. Using atrial extrastimulation techniques with His bundle recordings, Denes et al demonstrated that supraventricular tachycardia could be initiated by a critical coupling interval (Fig 2). The tachycardia was typically heralded by a sudden prolongation of the AH interval, and if all A₁ A₄ versus A₂ H₄ or H₁ vs H₄ are plotted on a graph, a discontinuous curve results. This discontinuity represents functional longitudinal dissociation within the AV node, giving rise to an electrophysiologic "fast" and "slow" pathway. The major determinants of these "dual AV nodal pathways" to generate and sustain a tachycardia are as follows: (1) the effective refractory period of the fast pathway must be longer than that of the slow pathway and of the atrium; (2) conduction time via the alternate, or slow pathway, must exceed the retrograde pathway refractoriness; (3) the cycle length of the ensuing tachycardia is longer than the effective refractory period of all components of the circuit.

Ability to induce the tachycardia is also cycle length dependent. If the antegrade fast pathway has a relatively short refractory period, a faster pacing cycle length and/or two atrial extrastimuli may be necessary to shift conduction from the fast to the slower pathway (Fig 3). Occasionally, ventricular pacing technique will initiate the tachycardia; the ventricular premature complex must block in the retrograde slow pathway, conduct through the fast pathway and reenter via the antegrade slow pathway. It is possible, during AV nodal reentrant SVT, to dissociate the atrium and the ventricle without interrupting the tachycardia which adds support to the concept that the reentrant mechanism is indeed confined to the AV node. The features of typical AV nodal reentrant supraventricular tachycardia are illustrated in Figure 2.

This type of SVT is usually recognized from the surface ECG by the axis of the P wave and its relationship to the QRS during the tachycardia. Characteristically, the P wave appears at the terminal part of

Figure 3. AV nodal reentrant SVT. The organization of electrograms is similar to Figure 1. Top panel, using a drive cycle length of 500 msec, a single atrial premature complex is introduced at 260 msec. No atrial depolarization follows the stimulus, defining the atrial effective refractory period. Dual AV nodal pathways were not demonstrated in this patient by single extrastimuli. Bottom panel, using the same drive cycle length (500 msec) with two atrial extrastimuli, the fast pathway is blocked, the impulse travels antegradely over the slow pathway (AH 565 msec) and sustained AV nodal reentrant tachycardia ensues. Note that there are no discernible P waves visible in the surface ECG during the SVT. This is because retrograde fast pathway atrial activation occurs simultaneously with ventricular depolarization, and the retrograde P wave is "buried" in the QRS.
the QRS, the beginning of the ST segment, or is buried within the QRS, and is inverted in the inferior limb leads (due to retrograde atrial depolarization via the fast pathway) (Fig 2). The SVT can usually be terminated by vagal maneuvers such as carotid sinus pressure, or by acute administration of drugs known to prolong AV nodal conduction and refractoriness, such as intravenous administration of verapamil, ouabain, or a beta-blocker. Chronic prophylaxis for this arrhythmia may be given if the arrhythmia is frequent and causes disabling symptoms. This can be accomplished with oral verapamil, digitalis, and beta-blockers, or in some cases, quinidine or procainamide. The mechanisms of the latter, type I drugs, are probably twofold: reduction of spontaneous atrial or ventricular premature complexes that may initiate the tachycardia, and prolongation of retrograde fast pathway refractoriness.

Electrophysiologic testing is recommended if the mechanism of the tachycardia is in doubt, if the patient experiences syncope or presyncope with the episodes, or if the patient continues to have episodes of SVT in spite of reasonable trials of the usual medications. Electrophysiologic studies are helpful in delineating the optimal medical regimen in a controlled in-hospital fashion, identifying patients for whom a special antitachycardia pacemaker would be most efficacious, and elucidating other electrophysiologic etiologies of the patients’ symptomatology.

Pre-excitation Syndrome

The most commonly encountered preexcitation syndrome is the Wolff-Parkinson-White syndrome. The characteristic surface ECG findings of the delta wave and abbreviated PR interval during normal sinus rhythm represent a fusion of simultaneous ventricular activation from the normal AV conduction system and the accessory pathway. The contribution to ventricular activation via the accessory pathway may be variable. The greater the aberrant activation, the more pronounced the delta wave becomes (Fig 4). Unlike the AV node, an accessory pathway does not respond to atrial pacing with decremental conduction; thus, the typical response to rapid atrial pacing is increased AV nodal delay and greater antegrade preexcitation over the bypass tract.

The circus movement tachycardia of the Wolff-Parkinson-White syndrome is typically initiated when a critically timed atrial or ventricular premature complex blocks in one limb of the circuit but is conducted successfully via the alternative route. Antegrade bypass refractoriness is longer than the retrograde counterpart in the most common situation. Thus, an atrial premature complex blocks in the accessory pathway, traverses the AV node and His-Purkinje system, and

![Figure 4. The fusion complex of WPW. Surface ECG leads 1, 2 and V1 are simultaneously displayed with intracardiac recordings of the HRA, HBE, proximal, mid and distal coronary sinus (CS), and RVA. The surface ECG during sinus rhythm (top panel), demonstrates a short P-R and delta wave (beginning of dotted line). The intracardiac recordings demonstrate normal AV nodal conduction (AH), and an HV of 90 msec (dotted line to H). Thus, the ventricular activation coincides with conduction antegrade through the normal A-V conduction system. More pronounced preexcitation via the accessory pathway is seen in the bottom panel. Following propranolol administration which further slows AV nodal conduction, the His activation follows the onset of ventricular activation by 20 msec, and the resultant QRS is widened.](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21379/ on 06/26/2017)
depolarizes the ventricles. This is manifested on the surface ECG as an abrupt loss of the delta wave and normalization of the QRS complex. The wave of depolarization then proceeds retrograde via the accessory pathway to reexcite the atria. The ability of the SVT to sustain itself will depend upon the continued recovery of excitability of each part of the reentrant loop. Rarely the antegrade limb is the accessory pathway, and the ventriculoatrial activation is through the AV node; this, of course, will produce a wide QRS tachycardia which may be difficult to differentiate from ventricular tachycardia.

The surface ECG during sinus rhythm has previously been used to classify WPW into type A and type B, depending upon the polarity of the delta wave in V1. This type of classification may be less useful now, since extensive intracardiac and epicardial mapping data have shown a poor correlation between these two patterns and the anatomic location of the bypass tract.\(^{a}\) These accessory pathways may occur anywhere around either AV valve ring; thus, they are more appropriately classified as right or left lateral, posterior, anterior, or paraseptal. The location of the tract is approximated during intracardiac electrophysiologic studies and can be identified precisely by intraoperative mapping if surgical interruption of the pathway is contemplated.

Electrophysiologic evaluation of patients with a preexcitation syndrome and history of tachycardia is of clinical value in several respects. Because the purpose of this paper is to give a general overview of the usefulness of electrophysiologic techniques, only a brief summary of the preexcitation electrophysiology can be presented here. The interested reader should consult the references for specific in-depth discussions of this complex topic.\(^{a1,a2}\) The following measurements are routinely studied in cases of WPW: (1) determination of antegrade and retrograde conduction and refractoriness of the bypass tract and the AV node; (2) the accessory tract location can be approximated by noting the pattern of retrograde atrial activation during the SVT or during ventricular pacing; (3) the rate of the tachycardia is observed as well as the "tachycardia zone," or range of coupling intervals during programmed atrial or ventricular stimulation which initiate the tachycardia; (4) methods of terminating the tachycardia can be evaluated, ie, single or double programmed extrastimulation, rapid atrial pacing, or a combination of these; (5) the response to antiarrhythmic medications can be immediately assessed by stimulation protocols before and following the drug challenge; if a drug is efficacious, the precise electropharmacologic site of block can be identified; (6) many patients with preexcitation syndromes have more than one type of supraventricular tachycardia; proper laboratory assessment of these patients should include search for AV nodal reentry as well as the presence of multiple bypass tracts; (7) atrial fibrillation or flutter can be initiated in the laboratory to assess the ability of the bypass tract for rapid antegrade (AV) conduction.\(^{a2}\)

Patients may have circus-movement tachycardia utilizing an accessory pathway in the retrograde direction, but examination of the surface ECG during sinus rhythm discloses no evidence of preexcitation. This is known as a concealed accessory pathway, a situation in which the antegrade refractoriness of the tract is prolonged while the retrograde conduction and refractoriness are normal. Such a case may be clinically confused with AV nodal reentrant SVT unless the characteristic eccentric retrograde atrial activation during the tachycardia is appreciated (characteristically, a negative P wave in lead 1 immediately following the QRS).

Very uncommonly, a bypass tract with an origin in or below the AV node, with insertion into ventricular tissue, is encountered. The electrophysiologic features of these nodoventricular and fasciculoventricular fibers depend largely on their anatomic origin in relation to the AV node. Thus, the more proximal the insertion, the greater preexcitation on the surface ECG. The typical nodoventricular bypass tract is diagnosed during electrophysiologic studies utilizing atrial pacing or programmed atrial extrastimulation techniques which demonstrate A-H prolongation, increased preexcitation of the QRS, and the ability to depolarize the ventricle prior to, or simultaneous with, the proximal His deflection.

The approach to the medical therapy of the circus movement tachycardia of the preexcitation syndrome tachycardias is as follows: (1) use medications that prevent or at least decrease spontaneous atrial or ventricular premature complexes which may initiate the tachycardia; (2) increase the retrograde refractoriness of the accessory pathway; and (3) depress AV nodal conduction and prolong its antegrade refractory period. The most frequently employed drugs which depress AV nodal function are verapamil, the beta blockers, and digitalis. For patients with rapid antegrade bypass conduction and who have a history of atrial fibrillation, these drugs should not be used empirically; the initial drugs of choice may be medications which more preferentially prolong refractoriness in the accessory pathway such as procainamide, disopyramide, or quinidine. The electrophysiology laboratory is best suited to determine the relative safety and efficacy of drugs for individual patients, minimizing potential worsening of the arrhythmia, as frequently happens when patients are subjected to empiric treatment regimens.

Specially designed programmable pacemakers may be useful for certain patients with reentrant forms of SVT. These units, which may be patient-triggered radiofrequency pacemakers or automatic triggering
(rate sensitive) pacemakers, have been used successfully in many patients who are unable or unwilling to take chronic prophylactic medications. Surgical interruption of accessory pathways should be performed in the following situations: (1) the patient is young, and long-term medical therapy is unacceptable; (2) the patient has atrial fibrillation with a rapid response, and the ventricular rate responds unsatisfactorily to medical therapy; (3) cardiac surgery is contemplated for other reasons. This type of surgery requires special equipment and personnel of considerable experience and expertise, and thus is restricted to certain medical centers with these facilities.

There is considerable debate regarding the mechanism of the Lown-Ganong-Levine syndrome. This eponym is probably best reserved for the clinical constellation of short P-R interval, no delta wave, in patients with narrow QRS and palpitations. A number of electrophysiologic mechanisms have been proposed to explain these findings. Therefore, the syndrome may actually represent a spectrum or variety of anatomic-electrophysiologic explanations, such as an AV nodal bypass tract (James fiber), accelerated AV nodal conduction, and short PR interval with dual AV nodal pathways. True AV nodal bypass fibers are probably quite rare; most episodes of SVT in the LGL syndrome are due to AV nodal reentry, with a smaller fraction attributable to a concealed bypass tract circus-movement tachycardia.

Accelerated AV nodal conduction, frequently seen in LGL, is an electrophysiologic diagnosis, characterized by a short AH interval during normal sinus rhythm (60 msec or less), 1:1 AV conduction maintained during atrial pacing at cycle lengths of 300 msec or less, and a maximum change in the A-H interval of 100 msec or less in response to programmed atrial extrastimulation. Not uncommonly, the effective refractory period of the His-Purkinje system exceeds that of the AV node, producing a physiologic form of infra-His block during atrial extrastimuli.

**Wide QRS Tachycardia—SVT vs VT**

Clinicians are frequently confronted with patients exhibiting a wide complex tachycardia and must differentiate between ventricular tachycardia and supraventricular tachycardia with aberrant conduction. In most cases, surface electrocardiographic features will permit accurate diagnosis. However, if there is a preexisting bundle branch block or accessory bypass tract, the correct diagnosis may be difficult without electrophysiologic intracardiac recordings (Fig 5). Wellens et al.19 recently studied the electrocardiographic features of 100 patients with supraventricular tachycardia and 100 with ventricular tachycardia. The diagnosis was established by electrophysiologic studies in all cases. Their findings are as follows: (1) approximately one half of the VT patients had AV dissociation during the tachycardia: the rest had ventriculoatrial (VA) conduction, either 1:1, 2:1, or VA Wenckebach; (2) extreme leftward axis in the frontal plane was a common finding in the VT group; 48 of 65 patients (74 percent) with RBBB morphology VT and 20 of 35 (57 percent) with LBBB VT had an axis of greater than −30°, uncommon in the SVT group (7 percent of the 100 patients); (3) the QRS width in patients with VT tended to be greater than 0.14 sec (on no antiarrhythmic drugs that would prolong the QRS duration); (4) certain BBB configurations were of value.

![Figure 5. Wide QRS tachycardia. ECGs from a 15-year-old boy who had complete correction of Fallot's tetralogy. Panels A and B show a wide QRS tachycardia. Panel C shows the QRS complex during normal sinus rhythm. Electrophysiologic studies demonstrated the tachycardia shown in panel A to be ventricular tachycardia. Panel B was shown to be AV nodal reentry. Without the sinus rhythm trace (panel C), using the usual surface ECG criteria, both tachycardias would have been diagnosed as ventricular in origin. This illustration shows the necessity of comparison of the 12 lead of the tachycardia with the sinus rhythm trace. Occasionally, only invasive electrophysiologic studies will yield the correct diagnosis. (From: Wellens HJJ, et al. Ventricular tachycardia-the clinical problem. In: Ventricular tachycardia. 9th ed. Mt Kisco, NY: Futura Publishing, 1982.)](image-url)
—if a RBBB tachycardia is present, a monophasic R wave in V6, or a rS or qS pattern in V1 or V6 is consistent with ventricular origin; if LBBB configuration is present, the presence of a QR or QS in V6 is suggestive of VT; if an initial small r is seen during the tachycardia in V1, and if a tracing during sinus rhythm discloses no r or a smaller r than that observed during the tachycardia, this is suggestive of ventricular tachycardia; (5) capture beats, fusion complexes, and concordant precordial QRS patterns were uncommon in this study. When present, they are helpful in diagnosing ventricular tachycardia.

As seen in Figure 5, the reliability of these general guidelines is diminished if preexistent bundle branch block is present. This shows the morphologies of SVT and VT in a 15-year-old patient 11 years after corrective surgery for tetralogy of Fallot. Panel B is the supraventricular tachycardia due to AV nodal reentry with QRS morphology and axis similar to that seen in normal sinus rhythm. Had the sinus rhythm trace not been available, panel B would have been mistaken as ventricular in origin.

Intracardiac recordings and programmed electrical stimulation provide the best means for establishing the mechanism as well as the origin of the tachycardia. During supraventricular tachycardia, a His deflection precedes each QRS, while in most cases of VT, the His will be disassociated from the ventricular electrograms. In the absence of a preexcitation syndrome, the inability to record a His deflection during a wide-complex tachycardia is virtually diagnostic of ventricular tachycardia.

**Ventricular Tachycardia**

In the past decade, electrophysiologic testing has revolutionized the management of patients with chronic recurrent sustained ventricular tachycardia. In earlier years, empiric drug therapy or surgical management were the only means of treatment, yet the high morbidity and mortality statistics were altered little by these interventions. Invasive electrophysiologic testing has now permitted a safe, reasonably reliable method for the proper selection of antiarrhythmic drug therapy, determining the efficacy of possible pacemaker therapy, and as a guide for mapping-directed surgical ablative procedures. These facets of VT management will be briefly summarized here.

Because the majority of cases of chronic recurrent ventricular tachycardia are due to a reentrant mechanism, the tachycardia can be reproducibly initiated and terminated by programmed stimulation. The pacing protocol used at our institution involves the introduction of single and double ventricular extrastimuli during sinus rhythm, and at least at two paced cycle lengths, from the apex of the right ventricle. If these techniques fail to initiate the tachycardia, the catheter is moved to other right ventricular sites and the pacing protocol repeated. Triple extrastimuli during a paced cycle length is then performed if the tachycardia has not been initiated. Occasionally, only left ventricular stimulation will initiate the tachycardia (Fig 6). In our experience, the majority of cases of sustained ventricular tachycardia are inducible using these procedures. Nonsustained ventricular tachycardia (VT lasting less than 30 seconds, self-terminating) has been less amenable to this form of evaluation; about 60 percent of clinical cases can be reproduced in the laboratory with these methods. Laboratory induction of ventricular tachycardia or ventricular fibrillation in the setting of acute infarction (less than four weeks postinfarction) has been disappointing. One half of the patients thus studied at our institution are not inducible at all, or have a tachycardia initiated in the laboratory which was not observed clinically.

After it is established that the ventricular tachycardia can be initiated by programmed stimulation techniques, antiarrhythmic drugs are administered and the studies repeated to study drug efficacy. If the tachycardia is no longer inducible following an antiarrhythmic drug, the patient may be given the medication on a long-term oral basis. Noninducibility signifies clinical suppression of the arrhythmia, supported by long-term follow-up of these patients. A serum level of the drug is obtained at the time of acute drug testing in the laboratory, and the patient is placed on oral doses of the medication to match the blood level that was found to be efficacious. While persistent inducibility of the ventricular tachycardia correlates with clinical recurrence of the arrhythmia, the experience with the experimental drug amiodarone has deviated from this pattern. Persistent inducibility of patients with sustained VT treated with amiodarone appears to have no prognostic value. These patients experience reduction in spontaneous recurrences of the tachycardia as well as significant decrease in spontaneous ventricular ectopic activity. Thus, the rationale for repeat electrophysiologic studies in patients treated with amiodarone is presently controversial. At our institution, these studies are still routinely employed for the following reasons: (1) if the rate of the induced tachycardia is not sufficiently slowed by amiodarone, causing hemodynamic compromise should the tachycardia recur clinically, a type 1 agent may be used as an adjuvant to reduce the rate; (2) occasional patients have experienced a worsening of the arrhythmia in response to amiodarone, in which case, alternative therapy is undertaken; (3) there is little information available concerning the prognosis of these patients. Prospective studies are needed to determine whether patients whose tachycardias are still inducible on amiodarone eventually have clinical recurrences or sudden death
compared to patients where tachycardias are not inducible prior to discharge.

Until recently, surgical therapy for ventricular tachycardia consisted of aneurysmectomy, infarctectomy, and coronary artery bypass grafting. The morbidity and mortality of such surgical intervention was quite high, and frequently, the arrhythmias recurred postoperatively. The reason for the disappointing results with these procedures is that there was no method by which the surgeon could verify that the site of origin of the tachycardia had been altered or excised. More recently, endocardial mapping studies have been employed in the electrophysiology laboratory, which are used as a guide for aneurysmectomy and endocardial resection.28-30 This involves the placement of a specially designed multielectrode catheter in 10 to 12 sites in the left ventricle and one to three sites in the right ventricle during the tachycardia. The earliest onset of electrical activity is determined, which corresponds to the origin of the reentrant circuit. At the time of surgery, extensive epicardial and endocardial mapping is repeated to verify the laboratory data and to further localize the circuit.

Aneurysmectomy with encircling endocardial ventriculotomy has also been employed as a method of surgical ablation.30 While the map-guided endocardial resection surgery has been an effective means of treating patients with drug-refractory ventricular tachycardia, if the site is in or close to a papillary muscle, mitral valve replacement must be performed. The encircling ventriculotomy procedure has been most efficacious in managing this problem. The rationale for this type of surgery is that the reentrant circuit arises in the border zone, between ischemic or necrotic tissue and adjacent normal tissue. Thus, the ventriculotomy incision isolates the pathologic reentrant circuit to a small area of myocardium, inhibiting the electrical wave of activation.

Analysis of the QRS morphology of ventricular tachycardia has been examined as a means of identifying the site of origin of the tachycardia.26,32 However, the surface QRS configuration correlates better with epicardial activation, based upon intraoperative mapping studies.28 Epicardial mapping of ventricular tachycardia is frequently misleading, since the earliest epicardial activation site may be several centimeters...

**Figure 6.** Laboratory induction of sustained ventricular tachycardia by left ventricular stimulation. Surface ECG leads, 1, 2, and V1 are recorded simultaneously with intracardiac recordings from the right ventricular apex (RVA), and left ventricular lateral wall (LV site 10). Top panel, following drive cycle length of 400 msec, two ventricular extrastimuli are delivered to the RVA. A single repetitive ventricular response is induced. Using similar pacing techniques, applied to the lateral wall of the LV (the margin of the patient's anteroseptal aneurysm), sustained ventricular tachycardia is initiated. The ability to induce the tachycardia is dependent upon the distance between the site of stimulation and the reentrant circuit.
distant from the endocardial site. This is particularly true for tachycardias arising from the interventricular septum. Therefore, if surgical therapy is contemplated, thorough laboratory mapping as well as studies at the time of surgery are essential.

Pacemaker therapy has been used occasionally in a few selected patients who have been refractory to medical therapy. Pacemaker therapy is somewhat hazardous because of the problems of potential pacemaker-triggering of ventricular tachycardia or acceleration of the tachycardia rate. Specially designed units have been implanted which sense the tachycardia and deliver a series of rapid depolarizations to terminate the arrhythmia. Such therapy must be undertaken only if the patient is not a surgical candidate, and if electrophysiologic studies have repeatedly demonstrated the efficacy and safety of ventricular pacing techniques in terminating tachycardia. More recently, the automatic implanted internal defibrillator has been used as a backup device for certain patients treated with antiarrhythmic medications. This investigational device has been used successfully in patients with recurrent ventricular fibrillation. Research is presently underway to alter this device for sensing ventricular tachycardia.

Conclusions

Although the tools of electrophysiologic testing have greatly advanced our understanding of cardiac arrhythmias and have enabled a more rational approach to treatment, much work is still needed. Programmed stimulation techniques, especially with regard to ventricular arrhythmias, have answered some questions but raised new ones. The role of such studies, if any, in the peri-infarction ventricular arrhythmias and in patients with chronic nonsustained ventricular tachycardia, are problems that are currently being investigated. Better mapping and surgical techniques are needed which will effectively ablate the tachycardia with minimal morbidity and mortality. Electrophysiologic testing has become an accepted diagnostic and therapeutic tool for managing arrhythmias. However, these techniques require specially trained personnel and specialized equipment. The physician performing these procedures must be experienced in clinical electrophysiology, having had special training in qualified teaching programs.

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