



Diagnosis of Cardiac Arrhythmias

John M. Miller and Douglas P. Zipes

34

HISTORY, 662

PHYSICAL EXAMINATION, 662

ELECTROCARDIOGRAM, 663

ADDITIONAL TESTS, 664

Exercise Testing, 666

In-Hospital Electrocardiographic Recording, 666

Long-Term Electrocardiographic Recording, 666

Heart Rate Variability, 668

Heart Rate Turbulence, 669

QT Dispersion, 669

Signal-Averaged Electrocardiography and Late Potentials, 669

T Wave Alternans, 669

Baroreceptor Reflex Sensitivity Testing, 669

Body Surface Mapping, 669

Electrocardiographic Imaging, 670

Upright Tilt-Table Testing, 670

Esophageal Electrocardiography, 670

INVASIVE ELECTROPHYSIOLOGIC STUDIES, 670

Complications of Electrophysiologic Studies, 674

DIRECT CARDIAC MAPPING: RECORDING POTENTIALS DIRECTLY FROM THE HEART, 674

REFERENCES, 676

GUIDELINES, 676

In managing clinical arrhythmias, physicians must evaluate and treat the whole patient, not just the rhythm disturbance.¹ Some arrhythmias are hazardous to the patient, regardless of the clinical setting (e.g., ventricular fibrillation [VF]), whereas others are hazardous because of the clinical setting (e.g., rapidly conducted atrial fibrillation [AF] in a patient with severe coronary artery stenoses). Some rhythm abnormalities, such as premature ventricular complexes (PVCs), may be highly symptomatic but not associated with any adverse outcomes, whereas some patients with AF have no symptoms at all but may still be at significant risk for stroke. Evaluation of the patient begins with a careful history and physical examination and should usually progress from the simplest to the most complex test, from the least invasive and safest to the most invasive and risky, and from the least expensive out-of-hospital evaluations to those that require hospitalization and sophisticated, costly, and potentially risky procedures. On occasion, depending on the clinical circumstances, the physician may wish to proceed directly to an expensive procedure associated with some risk, such as an electrophysiologic study (EPS), before obtaining a 24-hour electrocardiographic recording. In most cases, management of arrhythmia has a dual purpose: evaluation and treatment must address not only the patient's symptoms but also whatever risks that the arrhythmia poses to the individual.

HISTORY

Patients with disturbances in cardiac rhythm can have various complaints, but symptoms such as palpitations, syncope, presyncope, or dyspnea commonly cause them to seek a physician's help. Their awareness of palpitations and a regular or irregular cardiac rhythm varies greatly. Some patients perceive slight variations in their heart rhythm with uncommon accuracy, whereas others are oblivious to even sustained episodes of ventricular tachycardia (VT); still others complain of palpitations when they actually have regular sinus rhythm.

In assessing a patient with a known or suspected arrhythmia, several key pieces of information should be obtained that can help determine a diagnosis or guide further diagnostic testing. The mode of onset of an episode may provide clues about the type of arrhythmia or preferred treatment option. For example, palpitations that occur in the setting of exercise, fright, or anger are often caused by catecholamine-sensitive automatic or triggered tachycardias that may respond to adrenergic blocking agents (see Chapter 35); palpitations that occur at rest or that awaken the patient may be caused by vagal initiation, such as AF. Lightheadedness or syncope occurring in the setting of a tightly fitting collar, shaving the neck, or turning the head suggests carotid sinus hypersensitivity. The triggering event

may help establish the presence of an inherited ion channel abnormality (see Chapter 32). The mode of termination of episodes can also be helpful: palpitations that are reliably terminated by breath-holding or by Valsalva or other vagal maneuvers probably involve the atrioventricular (AV) node as an integral part of a tachycardia circuit; on occasion, focal atrial tachycardias or VTs can be terminated with vagal maneuvers. Patients should be asked about the frequency and duration of episodes and the severity of symptoms. In some women the features of their episodes vary according to the menstrual cycle. These features can help guide how aggressively and quickly the physician needs to pursue a diagnostic or therapeutic plan (a patient with daily episodes associated with near-syncope or severe dyspnea warrants a more expeditious evaluation than does one with infrequent episodes of mild palpitations and no other symptoms). Patients can sometimes report their heart rate during an episode (either rapid or slow, regular or irregular) by counting the pulse directly or by using an automatic blood pressure or heart rate monitor or smart phone application. Characteristics of the mode of onset and frequency of episodes can guide the choice of diagnostic tests (see later).

A careful drug and dietary history should also be sought; some nasal decongestants can provoke tachycardia episodes, whereas beta-adrenergic blocking eye drops for the treatment of glaucoma can drain into tear ducts, be absorbed systemically, and precipitate syncope secondary to bradycardia. Dietary supplements, particularly those containing stimulants such as ephedrine, can cause arrhythmias. A growing list of drugs can directly or indirectly affect ventricular repolarization and produce or exacerbate long-QT interval-related tachyarrhythmias (see Chapter 9; see also www.crediblemeds.org). The patient should be questioned about the presence of systemic illnesses that may be associated with arrhythmias, such as chronic obstructive pulmonary disease, thyrotoxicosis (see Chapter 81), pericarditis (see Chapter 71), and chronic heart failure (see Chapters 24 and 25), as well as previous chest injury, surgery, or radiation therapy or chemotherapy. A family history of rhythm disturbances is often present in those with long-QT syndrome, AF or other inherited arrhythmia syndromes, hypertrophic cardiomyopathy (see Chapter 66), and muscular or myotonic dystrophy (see Chapter 87).

PHYSICAL EXAMINATION

Examination of a patient during an arrhythmia episode can be revealing. Heart rate and blood pressure should be evaluated, as well as how ill the person appears. Assessment of jugular venous pressure and waveform can disclose the rapid oscillations of atrial flutter or "cannon" A waves indicative of contraction of the right atrium against



a closed tricuspid valve in patients with AV dissociation in disorders such as complete heart block or VT. Variations in the intensity of the first heart sound and systolic blood pressure have the same implications.

Physical maneuvers during tachycardia can have diagnostic and therapeutic value. As noted, the Valsalva maneuver² (as well as carotid sinus massage) causes a transient increase in vagal tone; tachyarrhythmias that depend on the AV node for continuation can terminate or slow with these maneuvers but may also show no change. Even though focal atrial and VTs occasionally terminate in response to vagal stimulation, sinus tachycardia slows slightly but returns to its original rate soon thereafter; the ventricular response during atrial flutter and fibrillation and other atrial tachycardias can decrease briefly. During wide-QRS tachycardias with a 1:1 relationship between P waves and QRS complexes, vagal influence can terminate or slow a supraventricular tachycardia (SVT) that depends on the AV node for perpetuation; on the other hand, vagal effects on the AV node can transiently block retrograde conduction and thus establish the diagnosis of VT by demonstrating AV dissociation. Because the effect of either of these physical maneuvers typically lasts only seconds, clinicians must be ready to observe or record any changes in rhythm on an electrocardiogram (ECG) when the maneuver is performed or the response may not be appreciated.

Carotid massage is performed with the patient supine and comfortable and the head tipped away from the side being stimulated. Careful auscultation for carotid bruits must always precede any attempt at carotid massage (embolic events have been associated with massage³). The area of the carotid sinus, at the artery's bifurcation, is palpated with two fingers at the angle of the jaw until a good pulse is felt. Even this minimal amount of pressure can induce a hypersensitive response in susceptible individuals. If no initial effect is noted, a side-to-side or rotating motion of the fingers over the site is performed for up to 5 seconds. A negative response is lack of effect on the ECG after 5 seconds of pressure adequate to cause mild discomfort. Because responses to carotid massage may differ on the two sides, the maneuver can be repeated on the opposite side; however, both sides should never be stimulated simultaneously. Findings may not be readily reproducible, even within minutes of a prior attempt.

Physical findings can suggest the presence of structural heart disease (and thus generally a clinically more serious situation with a worse overall prognosis), even in the absence of an arrhythmia episode. For example, a laterally displaced or dyskinetic apical impulse, a regurgitant or stenotic murmur, or a third heart sound in an older adult can denote significant myocardial or valvular dysfunction or damage.

ELECTROCARDIOGRAM

The ECG is the primary tool for analysis of arrhythmias (see Chapter 12); an EPS, in which intracardiac catheters are used to record activity from several regions of the heart at one time, is more definitive but not always immediately available. Initially, a 12-lead ECG is recorded. In addition, a long continuous recording with use of the lead that shows distinct P waves is often helpful for closer analysis; typically, this is one of the inferior leads (2, 3, aVF), V₁, or aVR. The ECG obtained during an arrhythmia episode may be diagnostic by itself and obviate the need for further diagnostic testing. Figure 34-1 depicts an algorithm for the diagnosis of specific tachyarrhythmias from the 12-lead ECG (see Chapter 37). A major branch point in the differential diagnosis concerns the QRS duration: wide-QRS (>0.12 second) tachycardias are often VTs, and narrow-QRS (≤ 0.12 second) tachycardias are almost always SVTs, but there is some overlap (Table 34-1). Next, the most important questions to answer, regardless of QRS width, concern the characteristics of P waves. If P waves are not clearly visible on the regular ECG, atrial activity can occasionally be discerned by placing the right and left arm leads in various anterior chest positions (so-called Lewis leads), by recording atrial

TABLE 34-1 Electrocardiographic Distinctions for Diagnosis of Wide-QRS Complex Tachycardia

FAVOR SUPRAVENTRICULAR TACHYCARDIA	FAVOR VENTRICULAR TACHYCARDIA
Initiation with a premature P wave	Initiation with a premature QRS complex
Tachycardia complexes identical to those in resting rhythm	Tachycardia beats identical to PVCs during sinus rhythm
"Long-short" sequence preceding initiation	"Short-long" sequence preceding initiation
Changes in the P-P interval preceding changes in the R-R interval	Changes in the R-R interval preceding changes in the P-P interval
QRS contours consistent with aberrant conduction (V ₁ , V ₆)	QRS contours inconsistent with aberrant conduction (V ₁ , V ₆)
Slowing or termination with vagal maneuvers	AV dissociation or other non-1:1 AV relationship
Onset of the QRS to its peak (positive or negative) <50 msec	Onset of the QRS to its peak (positive or negative) ≥ 50 msec
QRS duration ≤ 0.14 sec	Fusion beats, capture beats
	QRS duration >0.14 sec
	Left axis deviation (especially -90° to 180°)
	Concordant R wave progression pattern
	Contralateral bundle branch block pattern from the resting rhythm
	Initial R, q, or r >40 msec or notched Q in aVR
	Absence of an "rS" complex in any precordial lead

electrograms using intracardiac right atrial recordings (via permanent or temporary transvenous pacing leads), or by using esophageal electrodes or an echocardiogram; the last methods are not readily available in most clinical situations and consume valuable time when dealing with a sick patient. A long rhythm strip can usually be obtained and may yield important clues by revealing P waves if perturbations occur during the arrhythmia (e.g., changes in rate, premature complexes, sudden termination, and effect of physical maneuvers, as noted earlier).

Each arrhythmia should be approached in a systematic manner to answer several key questions; as suggested earlier, many of these questions relate to P wave characteristics and underscore the importance of assessing the ECG carefully for them. If P waves are visible, are the atrial and ventricular rates identical? Are the P-P and R-R intervals regular or irregular? If irregular, is it a consistent, repeating irregularity? Is there a P wave related to each QRS complex? Does the P wave seem to precede (long RP interval) or follow (short RP interval) the QRS complex (Fig. 34-2)? Are the resultant RP and PR intervals constant? Are all P waves and QRS complexes identical? Is the P wave vector normal or abnormal? Are P, PR, QRS, and QT durations normal? Once these questions have been addressed, one needs to assess the significance of the arrhythmia in view of the clinical setting. Should it be treated, and if so, how? For SVTs with a normal QRS complex, a branching decision tree such as that shown in Figure 34-1 may be useful.⁴

The Ladder Diagram

A ladder diagram, derived from the ECG, is used to depict depolarization and conduction schematically to aid in understanding the rhythm. Straight or slightly slanting lines drawn on a tiered framework beneath an ECG represent electrical events occurring in the various

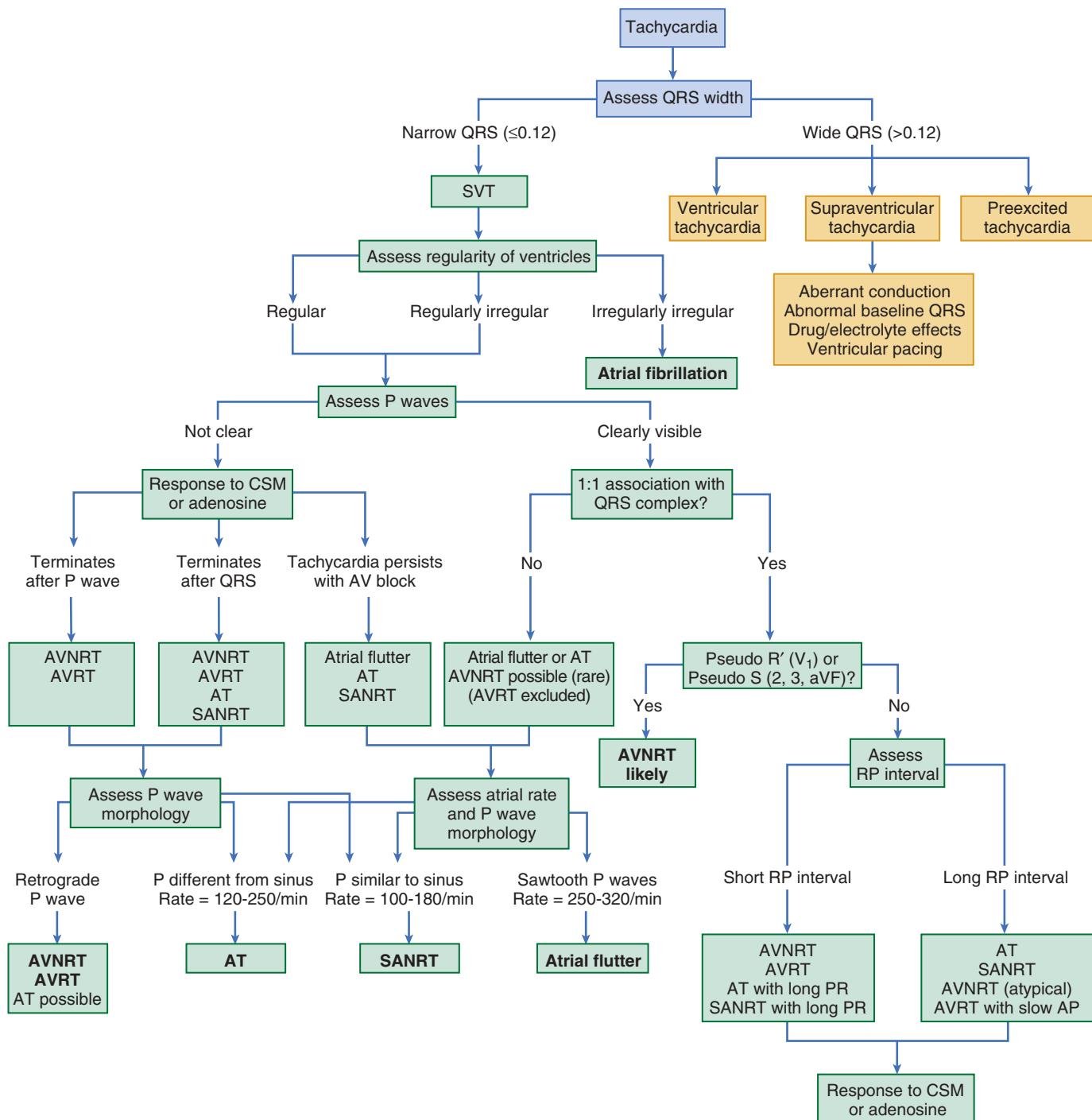


FIGURE 34-1 Stepwise approach to diagnosis of the type of tachycardia based on a 12-lead ECG during the episode. The initial step is to determine whether the tachycardia has a wide or narrow QRS complex. For wide-complex tachycardia, see Table 34-1; the remainder of the algorithm is helpful in diagnosis of the type of narrow-complex tachycardia. AP = accessory pathway; AT = atrial tachycardia; AVNRT = atrioventricular nodal reentrant tachycardia; AVRT = atrioventricular reciprocating tachycardia; CSM = carotid sinus massage; SANRT = sinoatrial nodal reentry tachycardia.

cardiac structures (Fig. 34-3). Because the ECG and therefore the ladder diagram represent electrical activity against a time base, conduction is indicated by the lines of the ladder diagram sloping in a left-to-right direction. A steep line represents rapid conduction; more slanting lines depict slower conduction. A short bar drawn perpendicular to a sloping line represents blocked conduction. Activity originating in an ectopic site such as the ventricle is indicated by lines emanating from that tier. Sinus nodal discharge and conduction and, under certain circumstances, AV junctional discharge and conduction can only be inferred; their activity is not directly recorded on the ECG.

ADDITIONAL TESTS

Most patients have only occasional episodes of arrhythmia and spend most of the time in their baseline rhythm (e.g., sinus, AF). The ECG during the patient's resting rhythm can provide clues about the presence of a substrate for arrhythmia (i.e., structural or physiologic abnormalities from which arrhythmias can arise). Several of these abnormalities are shown in Figure 34-4. Recently, the common finding on the ECG of early repolarization (in the lateral precordial and inferior leads) has been observed in some patients with primary VF (i.e., without identifiable structural heart disease). In most patients

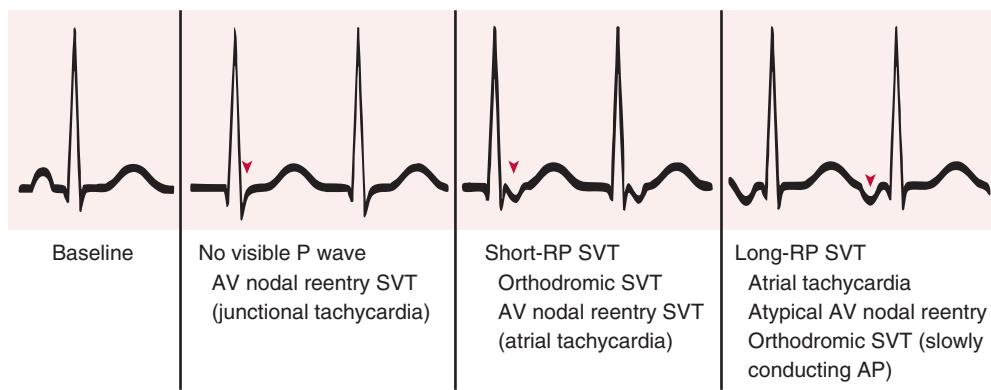


FIGURE 34-2 Differential diagnosis of different types of SVT based on timing of atrial activity (RP and PR intervals). **Left**, Normal beat. Different types of tachycardia are listed below the representative electrocardiographic patterns that they can produce, as categorized by P wave position relative to the QRS complex. An arrowhead shows the location of the P wave in each example. Diagnoses in parentheses are rare causes of the noted findings. AP = accessory pathway.

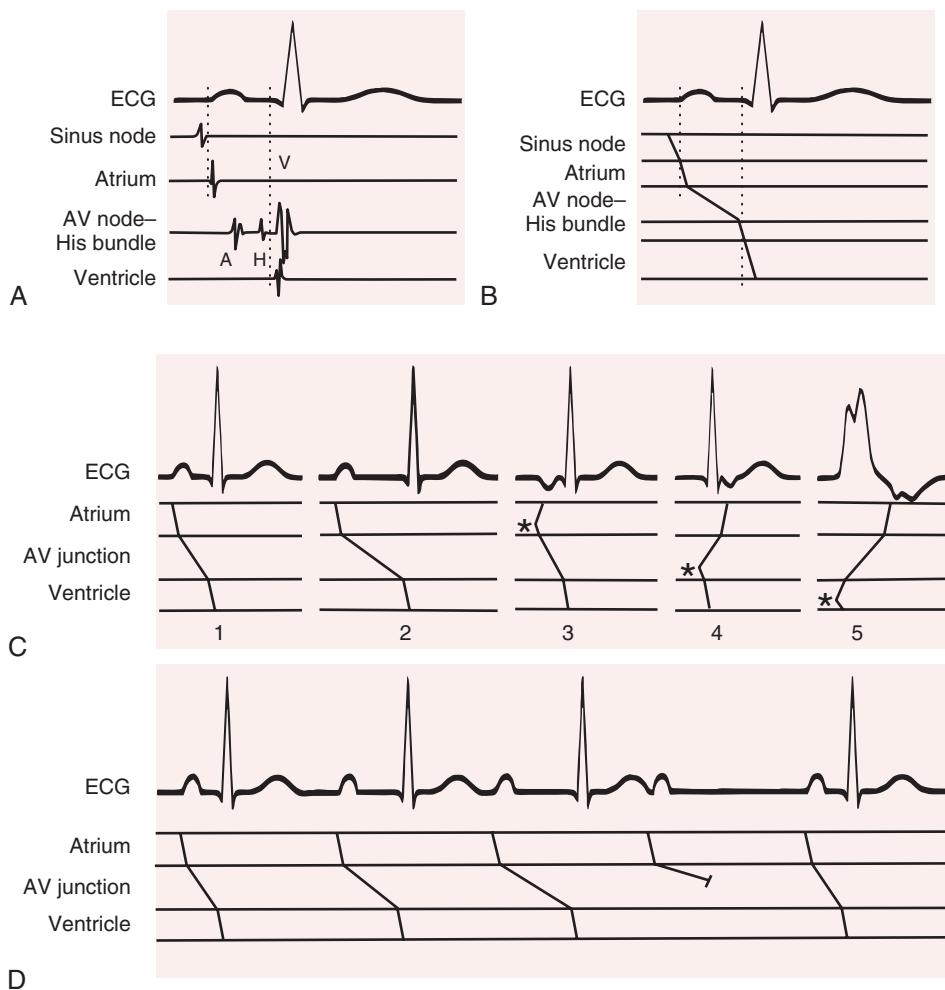


FIGURE 34-3 Intracardiac signals and ladder diagrams. **A**, A single beat is shown with accompanying intracardiac signals from the sinus node, right atrium, AV nodal and His bundle regions, and right ventricle. **B**, The same beat is shown with the accompanying ladder diagram below. Cardiac regions have been divided into tiers separated by horizontal lines. Vertical dotted lines denote onset of the P wave and QRS complexes. Note the relatively steep lines (rapid conduction through the atrium, His bundle, and ventricular muscle) and more gently sloping lines as the impulse traverses the sinus and AV nodes (signifying slow conduction). **C**, Several different situations are depicted with accompanying explanatory ladder diagrams. Beat 1 is normal, as in **B**; beat 2 shows first-degree AV delay, with the more gradual slope than normal in the AV nodal tier signifying very slow conduction in this region. In beat 3, an atrial premature complex is shown (starting in the atrial tier at the asterisk) and is producing an inverted P wave on the ECG. In beat 4, an ectopic impulse arises in the His bundle (asterisk) and propagates to the ventricle, as well as retrogradely through the AV node to the atrium. In beat 5, a ventricular ectopic complex (asterisk) conducts retrogradely through the His bundle and AV node and eventually to the atrium. **D**, A Wenckebach AV cycle (type I second-degree block) is shown. As the PR interval progressively increases from left to right in the figure, the slope of the line in the AV nodal region is progressively less steep until it fails to propagate at all after the fourth P wave (small line perpendicular to the sloping AV nodal conduction line), after which the cycle repeats. A = atrial recording; H = His recording; V = ventricular recording.

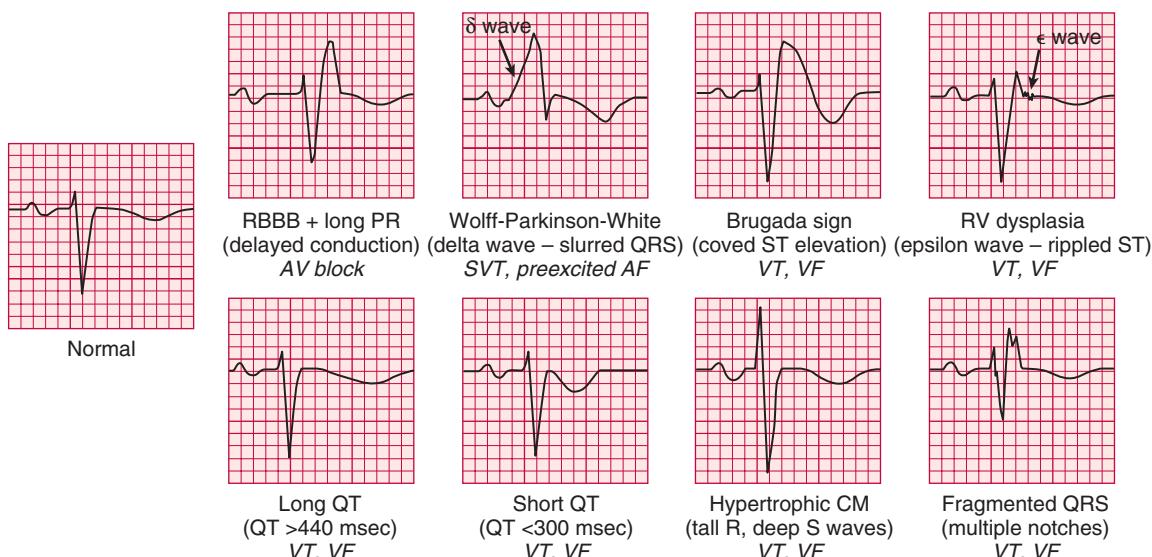


FIGURE 34-4 Electrocardiographic abnormalities in resting rhythm that suggest potential for arrhythmia. Lead V₁ is shown in each example; a normal complex is presented at the left for reference. CM = cardiomyopathy; RBBB = right bundle branch block; RV = right ventricular.

with SVT (aside from those with Wolff-Parkinson-White syndrome), findings on the resting ECG are normal. This is also true for many patients with ventricular tachyarrhythmias. Thus although it is capable of showing an abnormality with possible arrhythmic implications, the resting ECG is not a very sensitive tool. In light of this, the following additional tests can be used to evaluate patients who have cardiac arrhythmias. The physician's choice of which test to use depends on the clinical circumstances. For example, a patient with multiple daily episodes of presyncope is likely to have an event recorded on a 24-hour ambulatory electrocardiographic (Holter) monitor, whereas in a patient who complains of infrequent exercise-induced palpitations, exercise stress testing may be more likely to provide a diagnosis.

Exercise Testing

Exercise can induce various types of supraventricular and ventricular tachyarrhythmias and, uncommonly, bradyarrhythmias (see Chapter 13). Ventricular ectopy develops in approximately a third of normal subjects in response to exercise testing. Ectopy is more likely to occur at faster heart rates, usually in the form of occasional PVCs of constant morphology or even pairs of PVCs, and is often not reproducible from one stress test to the next. Three to six beats of nonsustained VT can occur in normal patients, especially the elderly, and its occurrence does not establish the existence of ischemic or other forms of heart disease or predict increased cardiovascular morbidity or mortality. PVCs are often more common during exercise than at rest and increase in frequency with age; their occurrence does not imply the presence of structural heart disease. A persistent elevation in heart rate after the end of exercise (delay in return to baseline) is associated with a worse cardiovascular prognosis.

PVCs develop in approximately 50% of patients with coronary artery disease in response to exercise testing. Ventricular ectopy appears in these patients at lower heart rates (<130 beats/min) than in the normal population and often occurs in the early recovery period as well. Frequent (>7 PVCs/min) or complex ectopy is associated with a worse prognosis. Exercise reproduces sustained VT or VF in less than 10% of patients with spontaneous VT or VF late after myocardial infarction, and these patients have a worse prognosis. The relationship of exercise to ventricular arrhythmia in patients with structurally normal hearts has no prognostic implications.

Patients who have symptoms consistent with an arrhythmia induced by exercise (e.g., syncope, sustained palpitations) should be considered for stress testing. Stress testing may be indicated to provoke supraventricular and ventricular arrhythmias, to determine

the relationship of the arrhythmia to activity, to aid in choosing antiarrhythmic therapy and uncovering proarrhythmic responses, and possibly to provide some insight into the mechanism of the tachycardia. The test can be performed safely; however, prolonged ambulatory recording is more sensitive than exercise testing in detecting most arrhythmias. Because either technique can uncover serious arrhythmias that the other technique misses, both examinations may be indicated for selected patients. Stress testing is frequently useful in patients with long-QT syndrome and catecholaminergic VT (see Chapters 13 and 32).⁵

In-Hospital Electrocardiographic Recording

Electrocardiographic monitoring systems are used in increasing proportions of inpatients regardless of history or suspicion of arrhythmias. These systems can provide valuable information about rhythm abnormalities, including mode of onset and termination, and allow prompt acquisition of a full 12-lead ECG for more detail. Telemetry may disclose intermittent heart block in a patient with presyncope that may warrant consideration of pacemaker implantation or reveal nonsustained VT in a patient with previous myocardial infarction and left ventricular dysfunction and prompt an electrophysiology study for further assessment of risk. As often as telemetry is helpful in such cases, however, it can be misleading: artifact can simulate VT or VF, heart block, or asystole. Careful scrutiny is necessary to avoid unnecessary tests and procedures in patients with these artifactual arrhythmias (Fig. 34-5; real and artifact on monitor).

Long-Term Electrocardiographic Recording

Prolonged electrocardiographic recording in patients engaged in normal daily activities is the most useful noninvasive method to document and quantitate the frequency and complexity of an arrhythmia, to correlate the arrhythmia with the patient's symptoms, and to evaluate the effect of antiarrhythmic therapy on spontaneous arrhythmia. For example, recording normal sinus rhythm during the patient's typical symptomatic episode effectively excludes cardiac arrhythmia as a cause. In addition, some recorders can document alterations in QRS, ST, and T contours.

Ambulatory Electrocardiographic (Holter) Recording

Continuous electrocardiographic tape recorders represent the traditional Holter monitor and digitally record three or more electrocardiographic channels for 24 to 48 hours. Computers scan the recording media, with human oversight, to provide a report with snapshot

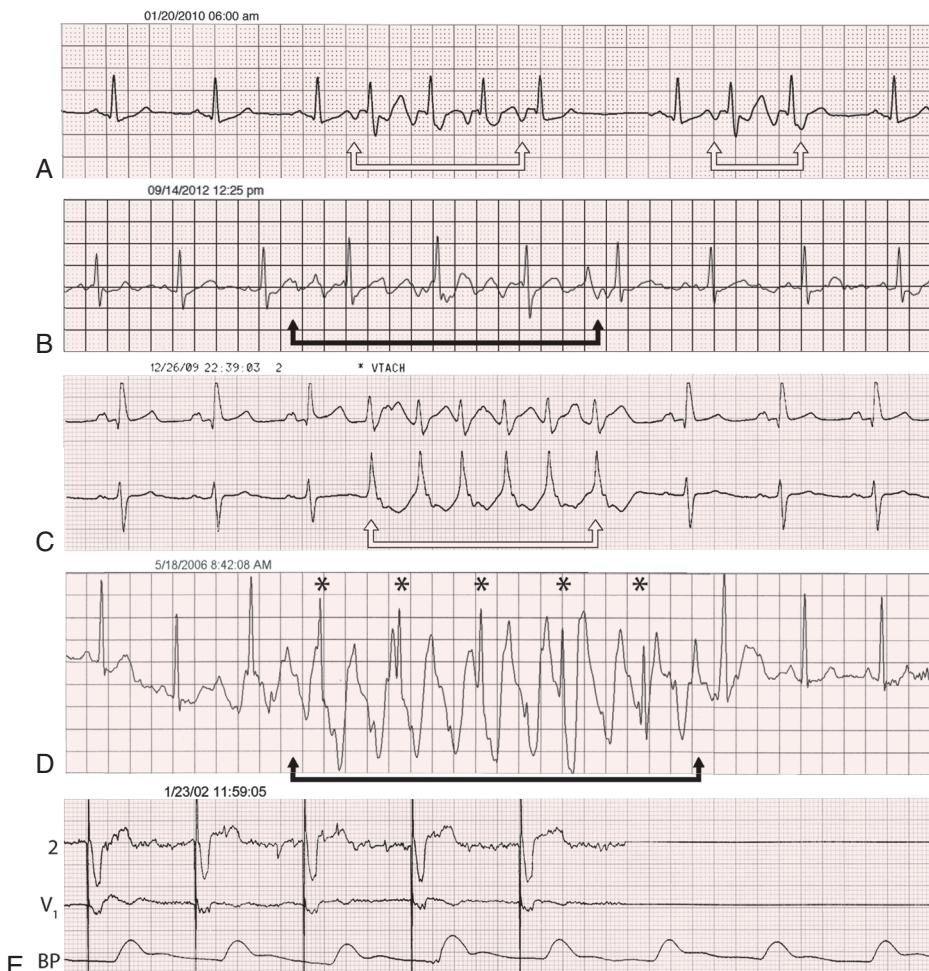


FIGURE 34-5 Electrocardiographic events and artifacts. **A**, Sinus rhythm punctuated by short episodes of atrial tachycardia with a more rapid ventricular rate (between the white arrows). **B**, Pseudo-atrial arrhythmia. Sinus rhythm is present throughout (no variation in the R-R interval) despite the appearance of a short episode of atrial flutter or fibrillation (between the black arrows). **C**, Nonsustained VT (between the white arrows) with wide rapid QRS complexes not preceded by a P wave and seen in two monitor leads. **D**, Pseudo-VT. Despite the appearance of VT (between the black arrows), sinus rhythm is present throughout (including complexes indicated by asterisks). **E**, Pseudo-pacemaker failure. After the first five paced complexes, the ECG is flat in both monitor leads, thus suggesting failure of pacemaker output; however, the pulse contour on the blood pressure (BP) tracing indicates that the heart is still contracting and the pacemaker is still working whereas the ECG monitor is not.

recordings of symptomatic events and other important findings (asymptomatic arrhythmias, ST-segment changes). All systems can potentially record more information than the physician needs or can assimilate. As long as the system detects important episodes of ectopic activity, VT, or asystolic intervals and semiquantifies these abnormalities, the physician probably receives all the clinical information that is needed. Twenty-five percent to 50% of patients experience a complaint during a 24-hour recording; in 2% to 15% the complaint is caused by an arrhythmia (**Fig. 34-6**). The ability to correlate symptoms temporally with abnormalities on the ECG is one of the strengths of this technique.

Significant rhythm disturbances are uncommon in healthy young persons. Sinus bradycardia with heart rates of 35 to 40 beats/min, sinus arrhythmia with pauses exceeding 3 seconds, sinoatrial exit block, type I (Wenckebach) second-degree AV block (often during sleep), wandering atrial pacemaker, junctional escape complexes, and premature atrial complexes and PVCs can be observed and are not necessarily abnormal. Frequent and complex atrial and ventricular rhythm disturbances are less commonly observed, however, and type II second-degree AV conduction disturbances (**see Chapter 37**) are not recorded in normal patients. Elderly patients (**see Chapter 76**) have a higher prevalence of arrhythmias, some of which may be responsible for neurologic symptoms (**Fig. 34-7**; also **see Chapter**

37). The long-term prognosis in asymptomatic healthy subjects with frequent and complex PVCs usually resembles that of the healthy U.S. population, without an increased risk for death. However, frequent PVCs (>15% of the total) have recently been shown to produce cardiomyopathy and heart failure in some people, which can be reversed following elimination of the PVCs.

Most patients with ischemic heart disease, particularly after myocardial infarction (**see Chapters 51 and 52**), exhibit PVCs when they are monitored for 24 hours. The frequency of PVCs progressively increases during the first several weeks and then decreases at about 6 months after infarction. Frequent and complex PVCs are associated with a twofold to fivefold increased risk for cardiac or sudden death in patients after myocardial infarction, but treating these PVCs may not improve the prognosis. CAST (Cardiac Arrhythmia Suppression Trial) showed that PVCs identified patients at increased risk for sudden death but that successful suppression of PVCs with flecainide, encainide, or moricizine was associated with increased mortality in comparison to placebo. Recent data indicate that ablation of PVCs after myocardial infarction may improve previously depressed ventricular function.

Long-term recording of the ECG has also exposed potentially serious arrhythmias and complex ventricular ectopy in patients with left ventricular hypertrophy, as well as in those with hypertrophic, dilated, and ischemic cardiomyopathy; in those with mitral valve prolapse (**see Chapter 63**); in those with otherwise unexplained syncope (**see Chapter 40**) or transient vague cerebrovascular symptoms; and in those with conduction disturbances, sinus node dysfunction, bradycardia-tachycardia syndrome, Wolff-Parkinson-White syndrome (**see Chapter**

37), and pacemaker malfunction (**see Chapter 36**). It has been shown that asymptomatic AF occurs far more often than symptomatic episodes in patients with AF.

Variations of Holter recording have been used for particular applications. Some monitoring systems are able to reconstruct a full 12-lead ECG from a 7-electrode recording system. This is especially useful in trying to document the electrocardiographic morphology of VT before an ablation procedure or a consistent morphology of PVCs that may arise from an ablatable focus of VT or VF. Most Holter recording and analysis systems can place a clearly recognizable deflection on the recording when a pacemaker stimulus is detected. This greatly facilitates diagnosis of potential pacemaker malfunction. On occasion, artifacts on the ECG caused by alterations in tape recording or playback speed can mimic bradycardias or tachycardias and lead to erroneous therapy. Newer digital Holter systems are less subject to this phenomenon. Finally, most systems can also provide heart rate variability and QT data (see later). Use of these systems for detection of myocardial ischemia (ST-segment analysis) has yielded mixed results (in both specificity and sensitivity).

Event Recording

In many patients, the 24- or 48-hour snapshot provided by the Holter recording is incapable of documenting the cause of the patient's

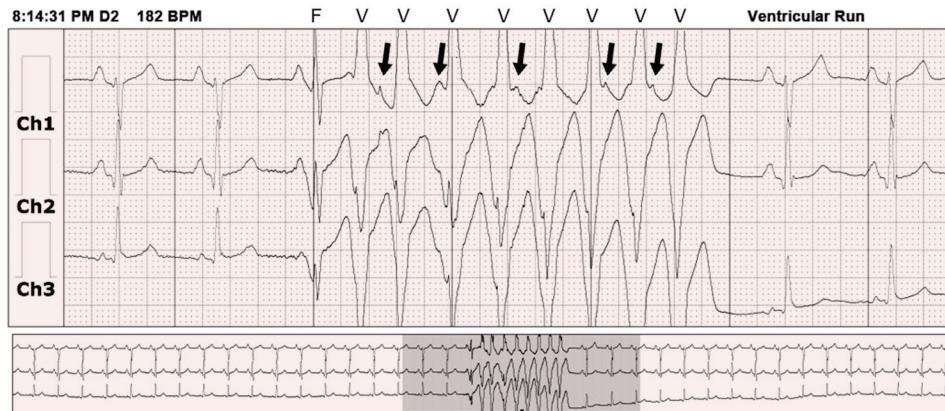


FIGURE 34-6 Long-term electrocardiographic recording in a patient with palpitations. A three-channel monitor shows sinus rhythm followed by nine wide QRS complexes of VT (labeled “V”); the complex that precedes these is a fusion between the normal complex and wide (“F”). Arrows indicate retrograde P waves during tachycardia. The presence of fewer P waves than QRS complexes and a fusion complex at the outset confirm the diagnosis of VT (which correlated with the patient’s palpitations).



FIGURE 34-7 Continuous electrocardiographic recording from a patient-activated event monitor during an episode of lightheadedness. Sinus rhythm at 75 beats/min with sudden AV block is present with pauses of longer than 4 seconds, and in the bottom strip there is an effective heart rate of approximately 8 beats/min.

symptoms. Longer-term monitoring, such as with an event recorder, is necessary in these cases, which occur frequently. These devices are about the size of a pager and are kept by the patient for 30 days. During that time, digital recordings can be made during symptomatic episodes and be transmitted to a receiving station over standard telephone lines at the patient’s convenience (see Fig. 34-7). Some of these recorders store more than 30 seconds of the ECG before the patient activates the recording. These loop recorders record continuously, but only a small window of time is present in memory at any moment; when the event button is pressed by the patient, the current window is frozen while the device continues recording for another 30 to 60 seconds, depending on how it is configured. Event recorders are highly effective in documenting infrequent events, but the quality of the recordings is more subject to motion artifact than with Holter recorders, and usually only one channel can be recorded. With some systems the patient must be able to press the event button to begin recording; if syncope occurs without warning and the patient is not able to activate the device, it cannot provide diagnostic information. With other systems, the device automatically begins recording the rhythm when the heart rate increases or decreases outside preset parameters. Some systems incorporate cell phone technology that automatically notifies a central monitoring facility when certain conditions are met (e.g., extreme bradycardia or tachycardia). In this way the time between occurrence and effective treatment of serious arrhythmias can be significantly shortened.⁶

Most currently available pacemakers and implantable defibrillators are capable of providing Holter-like data when premature beats or tachycardia episodes occur and can store electrograms of these events from the implanted leads.⁷ The device can then be interrogated and the electrograms printed for analysis. Many implanted device systems incorporate remote monitoring so that if symptoms develop, patients can perform device interrogation at home; the information is then transmitted via the Internet to the physician’s office and thus enables more prompt diagnosis and treatment than if the patient had to schedule an outpatient visit.

Implantable Loop Recorder

For patients with very infrequent symptoms, neither Holter recorders nor 30-day event recorders may yield diagnostic information. In such patients, an implantable loop recorder may be used. This device (about the size of a pack of chewing gum) is inserted under the skin at about the second rib on the left front aspect of the chest and is activated by passing a special magnet over the device. It is capable of recording up to 42 minutes of a single channel of the ECG that can be partitioned for one to seven episodes, with up to 20 minutes of the preactivation ECG being saved for subsequent downloading to a programming unit for analysis. Both P waves and QRS complexes can usually be identified. The device can be configured to store patient-activated episodes, automatically activated recordings (heart rate outside preset parameters), or a combination of these. In one report of patients with unexplained syncope, a

diagnosis was ultimately made in 80% by long-term monitoring, in 26% of them after 18 months of monitoring.⁸

A variety of additional noninvasive tests have been developed primarily to assess the risk for arrhythmic death in different groups of patients; although each has some applicability, none has enjoyed widespread use because of suboptimal sensitivity and specificity. Several of these tests are discussed in the following sections.

Heart Rate Variability

Heart rate variability is used to evaluate vagal and sympathetic influences on the sinus node (inferring that the same activity is also occurring in the ventricles) and to identify patients at risk for a cardiovascular event or death.⁹ Frequency domain analysis resolves parasympathetic and sympathetic influences better than time domain analysis does, but both types of analysis are useful. R-R variability predicts all-cause mortality, as well as left ventricular ejection fraction or nonsustained VT in patients after myocardial infarction, and can be added to other measures of risk to enhance predictive accuracy. Similar results have been obtained in patients with dilated cardiomyopathy (see Chapters 25 and 65). High-frequency components of R-R interval variability reflect tonic vagal activity. Reduced R-R interval variability, a marker of increased risk, indicates loss or reduction of the physiologic periodic sinus node fluctuations, which has many potential causes and may not necessarily represent a significant shift in autonomic modulation. New indices of heart rate variability are continually being evaluated. Even the simple measure of resting heart rate has been shown to be an independent cardiovascular risk factor, although

a target “safe” heart rate has not been established, as has the heart rate obtained during and after exercise.

Heart Rate Turbulence

Heart rate turbulence is an index of changes in the sinus discharge rate after a PVC that is followed by a compensatory pause.¹⁰ In normal individuals, the sinus rate initially accelerates and then slows; this phenomenon is blunted or absent in patients with various heart diseases. Heart rate turbulence is a measure of reflex vagal control of the heart, whereas heart rate variability is more indicative of overall vagal tone. Abnormal heart rate turbulence is a strong independent predictor of mortality in patients with coronary artery disease and dilated cardiomyopathy; abnormal indices in some patients can be improved or normalized after treatment with beta blockers and statin drugs.

QT Dispersion

Heterogeneity in refractoriness and conduction velocity is a hallmark of reentrant arrhythmias. One index of the heterogeneity of ventricular refractoriness can be found in differences in the length of the QT interval on surface leads of the ECG. The index most commonly used to calculate this QT dispersion has been the difference between the longest and shortest QT intervals on the 12-lead ECG, which is often adjusted for heart rate and the number of leads sampled (when the T wave is flat in some). Other indices have also been developed. Abnormally high QT dispersion has been correlated with risk for arrhythmic death in patients with various disorders, although the results are not consistent. Its mechanism has been characterized in several disease states. QT dispersion has been correlated with both efficacy and the proarrhythmic potential of drug therapy. Different techniques exist for determining dispersion (including automated algorithms), and the results of one study are often difficult to compare with those of another; in addition, the test is sensitive to age, time of day, season of year, and even body position.¹¹ Overall, assessment of QT dispersion has not gained popularity as a useful clinical tool.

Signal-Averaged Electrocardiography and Late Potentials

Signal averaging is a method that improves the signal-to-noise ratio when the signals are recurrent and the noise is random. In conjunction with appropriate filtering and other methods of noise reduction, signal averaging can detect cardiac signals of a few microvolts in amplitude and reduce noise amplitude, such as muscle potentials, which are typically 5 to 25 mV, to less than 1 mV. With this method, very low-amplitude electrical potentials generated by the sinus and AV nodes, His bundle, and bundle branches are detectable at the body surface.

One constituent of reentrant ventricular arrhythmias in patients with previous myocardial damage is slow conduction. Direct cardiac mapping techniques can record the myocardial activation from damaged areas that occurs after the end of the surface electrocardiographic QRS complex during sinus rhythm. These delayed signals have a very low amplitude that cannot be discerned by routine electrocardiography and correspond to the delayed and fragmented conduction in the ventricles recorded with direct mapping techniques (Fig. e34-1). Signal averaging has been applied clinically most often to detect such late ventricular potentials of 1 to 25 mV. Criteria for late potentials are the following: (1) filtered QRS complex duration longer than 114 to 120 milliseconds, (2) less than 20 mV of the root mean square signal amplitude in the last 40 milliseconds of the filtered QRS complex, and (3) terminal filtered QRS complex remaining below 40 mV for longer than 39 milliseconds. Such late potentials have been recorded in more than 70% of patients with spontaneous sustained and inducible VT after myocardial infarction but in only 0% to 6% of normal volunteers. Late potentials can be detected as early as 3 hours after the onset of coronary artery occlusion, increase in prevalence in the first week after myocardial infarction, and disappear in some patients after 1 year. If they are not present initially, late potentials do not usually appear later. Patients with bundle branch block or paced ventricular rhythms already have wide QRS complexes, thus rendering the technique less useful in these cases.

Late potentials have also been recorded in patients with VT not related to ischemia, such as in those with dilated cardiomyopathy. The presence of a late potential is a sensitive but not specific marker of arrhythmic risk, and therefore its prognostic use is limited. In specific situations it can be helpful, for example, in a patient suspected of having arrhythmogenic right ventricular cardiomyopathy¹² or a patient with a previous inferior wall myocardial infarction (normally the last portion of the heart to be activated), in whom the absence of a late potential suggests very low risk of having VT episodes.

The high-pass filtering used to record late potentials meeting the criteria just noted is called time domain analysis because the filter output corresponds in time to the input signal. Because late potentials are high-frequency signals, Fourier transform can be applied to extract high-frequency content from the signal-averaged ECG, called frequency domain analysis. Some data suggest that frequency domain analysis provides useful information not available with time domain analysis.

Signal averaging has been applied to the P wave to determine risk for the development of AF (especially after cardiac surgery), as well as maintenance of sinus rhythm after cardioversion.¹³ Overall use of the technique remains limited at present.

T Wave Alternans

Beat-to-beat alternation in the amplitude or morphology of the electrocardiographic recording of ventricular repolarization, the ST segment and T wave, has been found in conditions favoring the development of ventricular tachyarrhythmias, such as ischemia and long-QT syndrome, and in patients with ventricular arrhythmias. The electrophysiologic basis appears to be the alternation of repolarization of ventricular myocytes. In the presence of a long QT interval, the cellular basis of alternation may be beat-to-beat repolarization changes in midmyocardial cells (so-called M cells). Whether this mechanism applies to different disease states is not known. T wave alternans testing requires exercise or atrial pacing to achieve a heart rate of 100 to 120 beats/min with relatively little atrial or ventricular ectopic activity. The test is less useful in patients with a wide QRS complex (>120 milliseconds). A positive T wave alternans test result (Fig. e34-2) has been associated with a worse arrhythmic prognosis in various disorders, including ischemic heart disease and nonischemic cardiomyopathy. Although the predictive value of a positive test result varies greatly, depending on the population studied, a negative test result strongly predicts freedom from VT and VF in all group studied thus far, at least during a short follow-up period. Thus the test's best application appears to be in patients whose arrhythmic risk is equivocal, in whom a negative T wave alternans test result suggests low risk for the development of life-threatening ventricular arrhythmias. T wave alternans testing alone has not helped segregate patients more likely to benefit from implantable cardioverter-defibrillator (ICD) use (positive test result) from those who are not (negative test result) and should therefore not undergo ICD implantation. Both frequency domain (spectral method) and time domain (modified moving average) analyses have usefulness in risk stratification. T wave alternans may represent a fundamental marker of an electrically unstable myocardium prone to the development of VT or VF, but because of its relatively minor incremental value in defining arrhythmic risk, it is not frequently used at present.¹⁴

Baroreceptor Reflex Sensitivity Testing

Acute blood pressure elevation triggers a baroreceptor reflex that augments vagal tone to the heart and slows the sinus rate. The increase in sinus cycle length per millimeter mercury increase in systolic blood pressure is a measure of the sensitivity of the baroreceptor reflex and, when reduced, identifies patients susceptible to the development of VT and VF. The mechanism of the reduction in baroreceptor reflex sensitivity is not certain. However, this test may be useful to identify patients at risk for the development of a serious ventricular arrhythmia after myocardial infarction.

Body Surface Mapping

Isopotential body surface maps are used to provide a complete picture of the effects of currents from the heart on the body surface. The potential distributions are represented by contour lines of equal potential, and each distribution is displayed instant by instant throughout activation, recovery, or both.

Body surface maps have been used clinically to localize and size areas of myocardial ischemia, to localize ectopic foci or accessory pathways, to differentiate aberrant supraventricular conduction from ventricular origin, to recognize patients at risk for the development of arrhythmias, and possibly to understand the mechanisms involved. Although these procedures are of interest, their clinical usefulness has not yet been established. In addition, the technique is cumbersome and the analysis is complex.

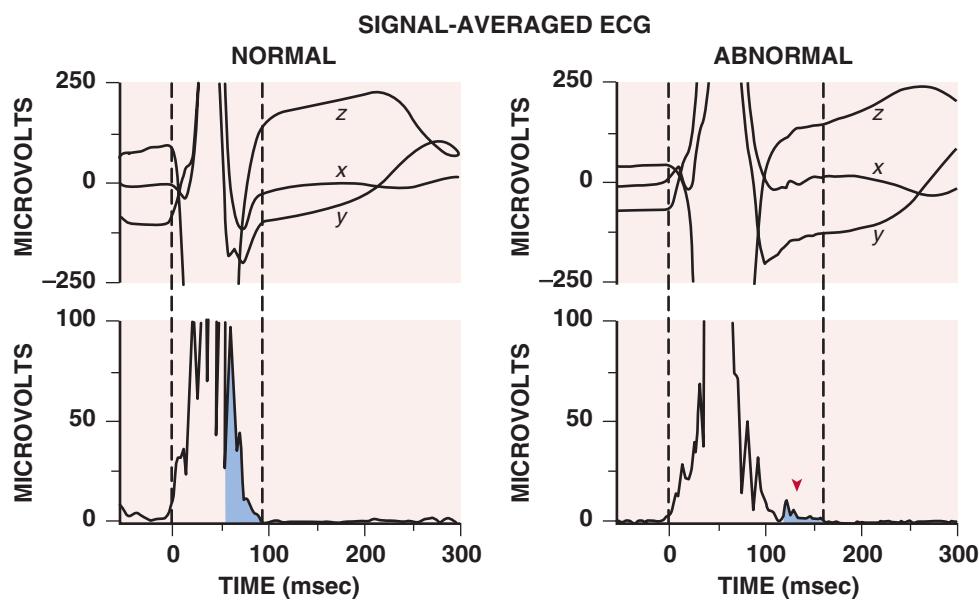


FIGURE e34-1 Signal-averaged ECG. Normal (left) and abnormal (right) results are shown from a patient with previous myocardial infarction and VT. Bottom panels, Shaded blue areas at the end of each tracing represent the voltage content of the last 40 milliseconds of the filtered QRS integral. The small shaded area (red arrowhead) in the abnormal study denotes prolonged, slow conduction and suggests the potential for reentrant ventricular arrhythmias.

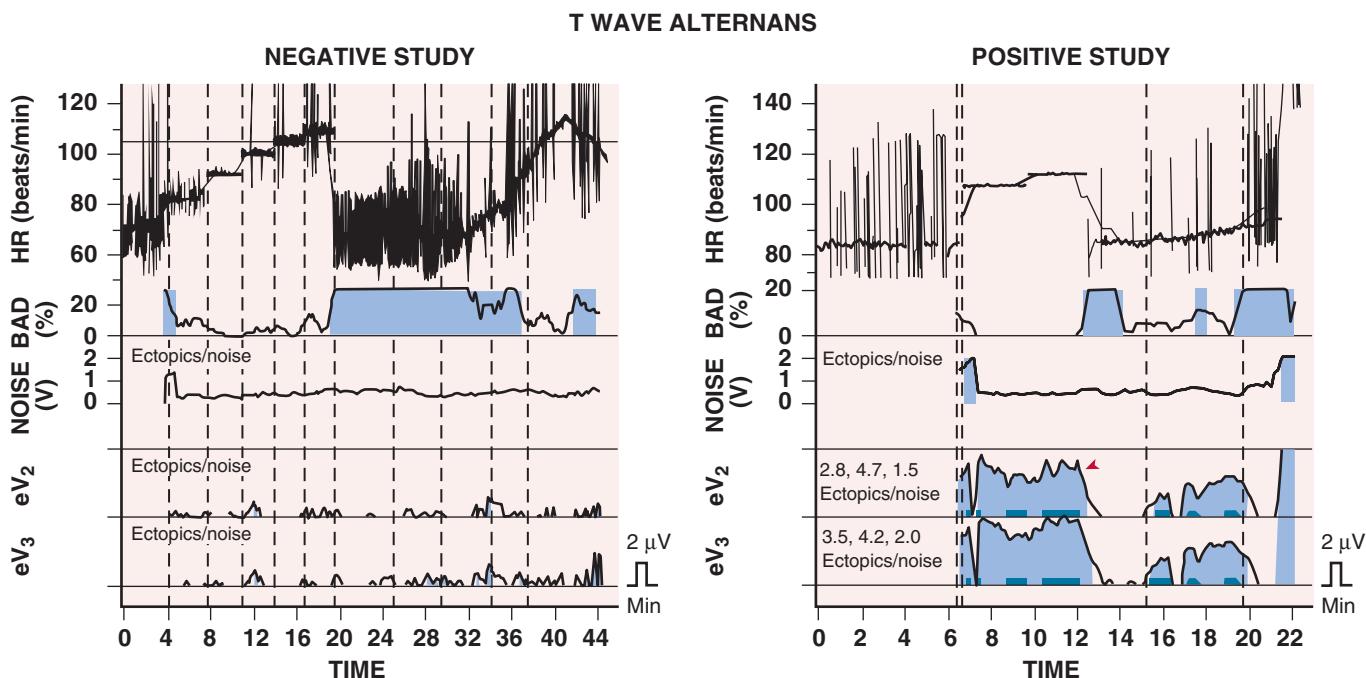


FIGURE e34-2 T wave alternans. Reports of T wave alternans analysis from two patients are shown and display the heart rate in beats/min (HR BPM), proportion of beats rejected from analysis (% BAD), ECG noise level (in microvolts), and selected precordial leads (eV₂ and eV₃) as a function of time. **Left panel**, Records from a patient with no structural heart disease; the amplitude of T wave alternans was minimal. **Right panel**, Records from a patient hospitalized for sustained VT after myocardial infarction show T wave alternans (blue shaded area, arrowhead).



Electrocardiographic Imaging

Another promising technology is electrocardiographic imaging, in which cardiac electrical activity recorded at the skin surface is spatially integrated with imaging data (currently, cardiac computed tomography scanning). Using complex mathematical processing of electrical data collected from 224 electrodes on the skin surface, this technique can plot or project atrial and ventricular electrical activity on an epicardial “shell” of the patient’s own heart and thereby follow the course of activation during sinus rhythm or an arrhythmia. Clinical experience is limited thus far, but both SVTs and VTs have been able to be localized in a variety of settings.¹⁵

Upright Tilt-Table Testing

The tilt-table test is used to identify patients who have a vasodepressor or cardioinhibitory response as a cause of syncope (see Chapter 40). Patients are placed on a tilt table in the supine position and tilted upright to a maximum of 60 to 80 degrees for 20 to 45 minutes or longer if necessary (Fig. e34-3). Isoproterenol, administered as a bolus or infusion, may provoke syncope in patients whose initial upright tilt-table test result shows no abnormalities or, after a few minutes of tilt, may shorten the time needed to produce a positive response on the test. An initial intravenous isoproterenol dose of 1 µg/min can be increased in 0.5-µg/min steps until symptoms occur or a maximum of 4 µg/min is given. Isoproterenol induces a vasodepressor response in upright susceptible patients (decrease in heart rate and blood pressure along with near-syncope or syncope). Tilt-table test results are positive in two thirds to three fourths of patients susceptible to neurally mediated syncope and are reproducible in approximately 80% but have a 10% to 15% false-positive response rate. A positive test result is more meaningful when it reproduces symptoms that have occurred spontaneously. Positive responses can be divided into cardioinhibitory, vasodepressor, and mixed categories. Therapy with beta blockers, disopyramide, theophylline, selective serotonin reuptake inhibitors, midodrine, fludrocortisone, salt loading, and thigh-high support stockings, alone or in combination, has been reported to be successful but not with reliable reproducibility. Tilt training, in which the patient leans against a wall for prolonged periods to increase tolerance to this body position, as well as isometric muscle flexing to abort or lessen an episode, may help. Permanent pacing has been useful in a subset of patients with significant bradycardia.

A variant of the neurocardiogenic response, postural orthostatic tachycardia syndrome (POTS), is characterized by dramatic increases in heart rate during the first 10 minutes of tilt-table testing. POTS appears to be distinct from simple orthostatic hypotension, as well as from standard neurocardiogenic responses, and is thought to be caused by various forms of autonomic imbalance. Relief of symptoms has been effected with fludrocortisone, beta blockers, or combinations.

Esophageal Electrocardiography

Esophageal electrocardiography is a useful noninvasive technique for diagnosing arrhythmias. The esophagus is located immediately behind the left atrium, between the left and right pulmonary veins. An electrode in the lumen of the esophagus can record atrial potentials. Bipolar recording is superior to unipolar recording because far-field ventricular events can lead to possible diagnostic confusion with unipolar recording. In addition, atrial and occasionally ventricular pacing can be performed by means of a catheter electrode inserted into the esophagus, and tachycardias can be initiated and terminated. Optimal electrode position for atrial pacing correlates with the patient’s height and is within about 1 cm of the site at which the maximum amplitude of the atrial electrogram is recorded. When it is recorded simultaneously with the surface ECG, the esophageal atrial electrogram can be used to differentiate SVT with aberrancy from VT and to define the mechanism of SVTs. Complications of transesophageal recording and pacing are uncommon, but the technique is cumbersome and uncomfortable for most patients, and it is therefore not commonly used.

INVASIVE ELECTROPHYSIOLOGIC STUDIES

An invasive EPS involves introducing multipolar catheter electrodes into the venous or arterial system and positioning them at various

intracardiac sites to record or stimulate cardiac electrical activity. Assessment of AV conduction at rest is done by positioning the catheter along the septal leaflet of the tricuspid valve and measuring the atrial-His interval (an estimate of AV nodal conduction time; normally, 60 to 125 milliseconds) and the His-ventricular (H-V) interval (a measure of infranodal conduction; normally, 35 to 55 milliseconds). The heart is stimulated from portions of the atria or ventricles and from the region of the His bundle, bundle branches, accessory pathways, and other structures. Such studies are performed diagnostically to provide information about the type of clinical rhythm disturbance and insight into its electrophysiologic mechanism. An EPS is used therapeutically to terminate a tachycardia by electrical stimulation or electroshock, to evaluate the effects of therapy by determining whether a particular intervention modifies or prevents electrical induction of a tachycardia or whether an electrical device properly senses and terminates an induced tachyarrhythmia, and to ablate myocardium involved in the tachycardia and prevent further episodes. Finally, these tests have been used prognostically to identify patients at risk for sudden cardiac death. The study may be helpful in patients with AV block, intraventricular conduction disturbance, sinus node dysfunction, tachycardia, and unexplained syncope or palpitations (see Chapter 40).

An EPS is effective at initiating VT and SVT when these tachyarrhythmias have occurred spontaneously. This enables the use of similar stimulation techniques after an intervention (e.g., drug therapy or catheter or surgical ablation) to assess the efficacy of treatment. However, false-negative responses (not finding a particular electrical abnormality known to be present) and false-positive responses (induction of a nonclinical arrhythmia) may complicate interpretation of the results because many lack reproducibility. Altered autonomic tone in a supine patient undergoing study, hemodynamic or ischemic influences, changing anatomy (e.g., new infarction) after the study, day-to-day variability, and the fact that the test uses an artificial trigger (electrical stimulation) to induce the arrhythmia are several of many factors that can explain the occasional disparity between test results and spontaneous occurrence of arrhythmia. Overall, the diagnostic validity and reproducibility of these studies are good, and they are safe when performed by skilled clinical electrophysiologists.

Atrioventricular Block

In patients with AV block, the site of block usually dictates the clinical course of the patient and whether a pacemaker is needed (see Chapter 37). In general, the site of AV block can be determined from analysis of the regular ECG. When the site of block cannot be determined from such an analysis and when knowing the site of block is imperative for management of the patient, an invasive EPS is indicated. Candidates include symptomatic patients in whom a His-Purkinje block is suspected but not established and patients with AV block treated with a pacemaker who continue to be symptomatic and in whom a causal ventricular tachyarrhythmia is suspected. Possible candidates are those with second- or third-degree AV block, for whom information about the site of block or its mechanism may help direct therapy or assess prognosis, and patients suspected of having concealed His bundle extrasystoles. Patients with block in the His-Purkinje system become symptomatic because of periods of bradycardia or asystole and require pacemaker implantation more often than do patients who have AV nodal block. Type I (Wenckebach) AV block in older patients may have clinical implications similar to those for type II AV block. The results of EPS for evaluating the conduction system must be interpreted with caution, however. In rare cases, the process of recording conduction intervals alters their values. For example, catheter pressure on the AV node or His bundle can cause prolongation of the atrial-His or H-V interval and could lead to erroneous diagnosis and therapy.

Intraventricular Conduction Disturbance

For patients with an intraventricular conduction disturbance, an EPS provides information about the duration of the H-V interval, which can be prolonged with a normal PR interval or normal with a

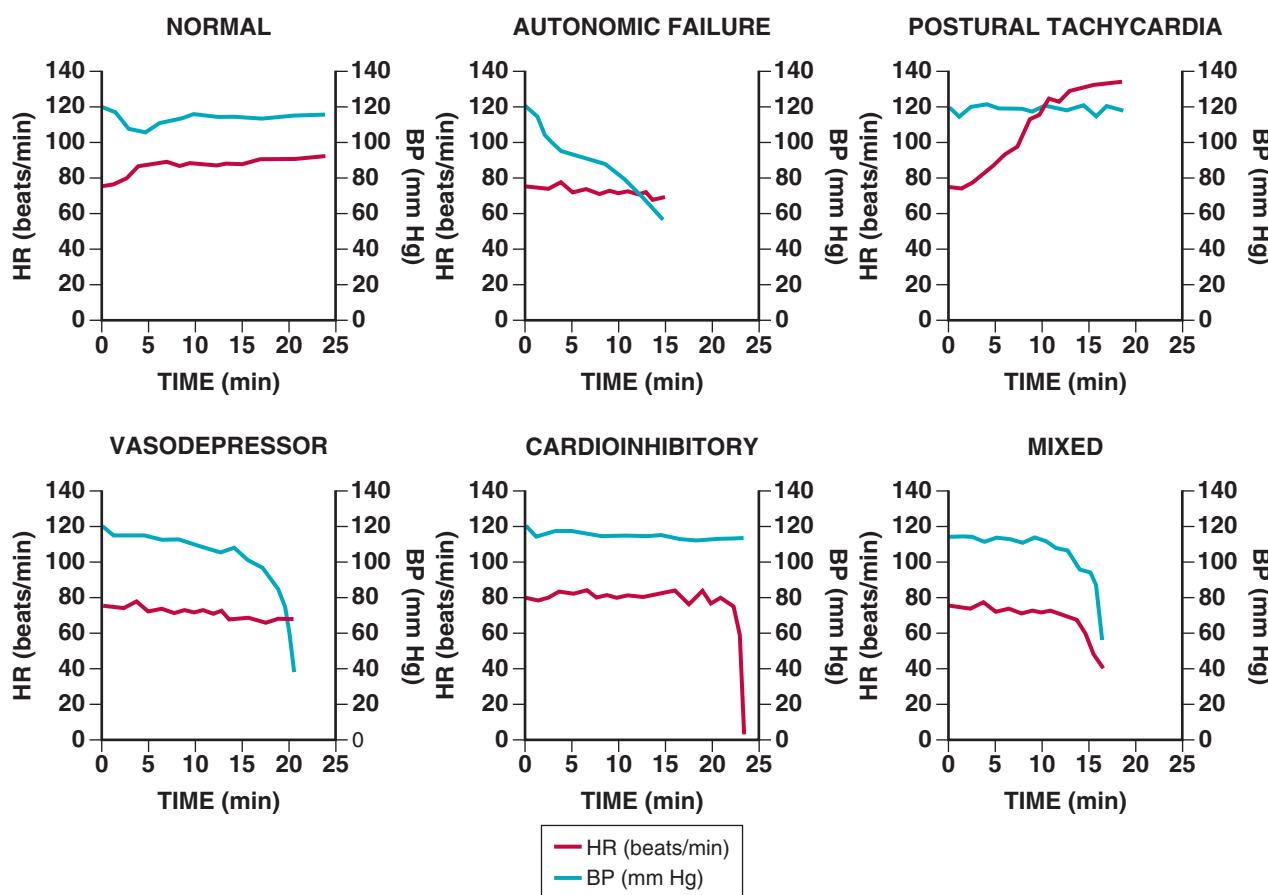


FIGURE e34-3 Head-up tilt-table testing. Responses to tilt-table testing are shown; heart rate (HR) and systolic blood pressure (BP) are plotted as time functions. In the **upper panel**, a normal response is an early, slight drop in BP with a compensatory increase in HR mediated by the autonomic nervous system. With autonomic dysfunction, a progressive fall in BP is not counteracted by an increase in HR. In postural tachycardia syndrome, an exaggerated increase in HR is seen. The **bottom panel** depicts findings with neurocardiogenic syncope: a pure vasodepressor response is a relatively sudden drop in BP without a marked change in HR, whereas a pure cardioinhibitory response shows a sudden decrease in HR without a change in BP. A mixed response shows decreases in both HR and BP.

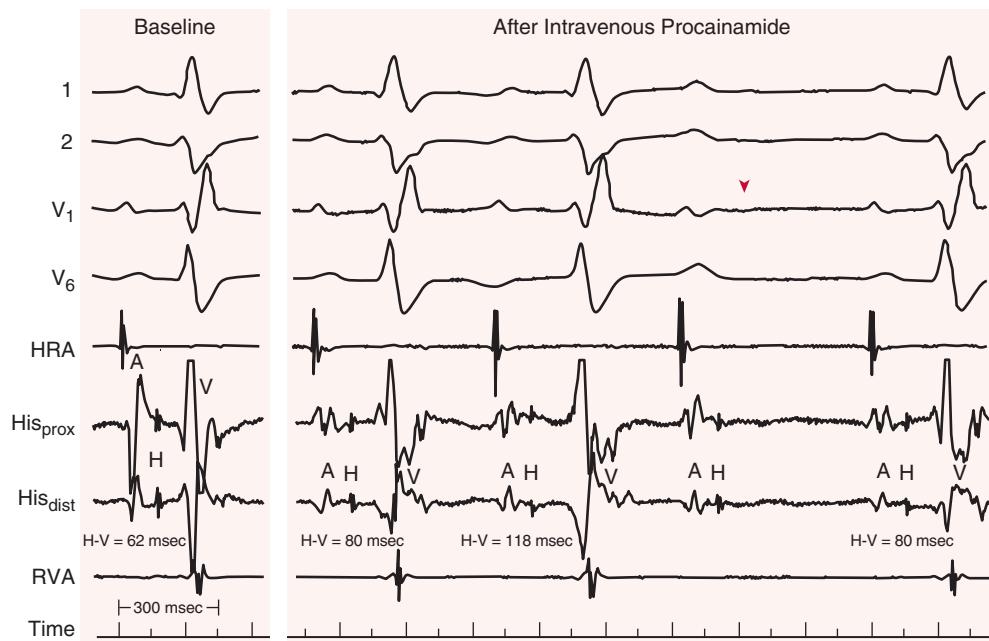


FIGURE 34-8 Testing the His-Purkinje system. A 43-year-old woman with sarcoid underwent EPS after a syncopal episode. Surface leads 1, 2, V_1 , and V_6 are shown, with intracardiac recordings from catheters in the high right atrium (HRA), the proximal (His_{prox}) and distal (His_{dist}) electrode pairs of a catheter at the AV junction to record the His potential, and right ventricular apex (RVA). During baseline recording, the H-V interval is only slightly prolonged (62 milliseconds). After infusion of intravenous procainamide, the H-V interval is longer and an infra-His Wenckebach block is present. The arrowhead denotes the missing QRS complex caused by infra-His block. A = atrial electrogram; H = His potential; V = ventricular electrogram.

prolonged PR interval. A prolonged H-V interval (>55 milliseconds) is associated with a greater likelihood for the development of a trifascicular block (but the rate of progression is slow, 2% to 3% annually) and for having structural disease and with higher mortality.¹⁶ The finding of very long H-V intervals (>80 to 90 milliseconds) identifies patients at increased risk for the development of AV block. The H-V interval has high specificity (~80%) but low sensitivity (~66%) for predicting the development of complete AV block. During the study, atrial pacing is used to uncover abnormal His-Purkinje conduction. A positive response is provocation of distal His block during 1:1 AV nodal conduction at rates of 135 beats/min or less. Again, sensitivity is low but specificity is high. Functional His-Purkinje block caused by normal His-Purkinje refractoriness is not a positive response. Drug infusion, such as with procainamide or ajmaline, sometimes exposes abnormal His-Purkinje conduction (Fig. 34-8). Ajmaline (not available in the United States) can cause arrhythmias and should be used cautiously.

An EPS is indicated in patients with symptoms (syncope or presyncope) that appear to be related to a bradyarrhythmia or tachyarrhythmia when no other cause of symptoms is found. For many of these patients, ventricular tachyarrhythmias rather than AV block can be the cause of their symptoms, with obvious therapeutic implications.

Sinus Node Dysfunction

Demonstration of slow sinus rates, sinus exit block, or sinus pauses temporally related to symptoms suggests a causal relationship and usually obviates further diagnostic studies (see Chapter 37). Carotid sinus pressure that results in several seconds of complete asystole or AV block and reproduces the patient's usual symptoms exposes the presence of a hypersensitive carotid sinus reflex. Carotid sinus massage must be done cautiously; rarely, it can precipitate a stroke. Neurohumoral agents, adenosine, or stress testing can be used to evaluate the effects of autonomic tone on sinus node automaticity and sinoatrial conduction time. An EPS should be considered in patients who have symptoms attributable to bradycardia or asystole, such as presyncope or syncope, and for whom noninvasive approaches have provided no explanation for the symptoms.

Sinus Node Recovery Time. Sinus node recovery time (SNRT) is a technique that can be useful for evaluating sinus node function. The interval between the last paced high right atrial response and the first spontaneous (sinus) high right atrial response after termination of pacing is measured to determine SNRT. Because the spontaneous sinus rate influences SNRT, the value is corrected by subtracting the spontaneous sinus node cycle length (before pacing) from the SNRT (Fig. 34-9). This value, the corrected SNRT (CSNRT), is generally shorter than 525 milliseconds. A prolonged CSNRT has been found in patients suspected of having sinus node dysfunction. After cessation of pacing, the first return sinus cycle can be normal but can be followed by secondary pauses. Secondary pauses appear to be more common in patients whose sinus node dysfunction is caused by sinoatrial exit block (a potential cause of sinus pauses on the ECG). Direct recordings of the sinus node electrogram have been made, but the technique is cumbersome. Finally, it is important to evaluate AV node and His-Purkinje function in patients with sinus node dysfunction because many also exhibit impaired AV conduction.

Sinoatrial Conduction Time.

Sinoatrial conduction time (SACT) can be estimated by simple pacing techniques based on the assumptions that (1) conduction times into and out of the sinus node are equal, (2) no depression of sinus node automaticity occurs, and (3) the pacemaker site does not shift after premature stimulation (see Chapter 34). These assumptions can be erroneous, particularly in patients with sinus node dysfunction. SACT can also be measured directly with special recording techniques, as noted above, from the region of the sinus node. This direct measurement correlates well with the SACT measured indirectly in patients with normal sinus node function. The sensitivity of the SACT and SNRT tests is only approximately 50% for each test alone and around 65% when they are combined. The specificity, combined, is approximately 88%, with a low predictive value. Thus if these test results are abnormal, the likelihood of the patient having sinus node dysfunction is great. However, normal results do not exclude the possibility of sinus node disease. Candidates for invasive EPS to evaluate sinus node function are symptomatic patients in whom sinus node dysfunction has not been established as a cause of the symptoms. Potential candidates are patients with clinical sinus node dysfunction in whom other causes of symptoms (e.g., tachyarrhythmias) are to be excluded.

Tachycardia

In patients with tachycardias, an EPS can be used to diagnose the arrhythmia, to determine and deliver therapy, to establish the anatomic sites involved in the tachycardia, to identify patients at high risk for the development of serious arrhythmias, and to gain insight into the mechanisms responsible for the arrhythmia (see Chapter 37). The study can differentiate aberrant supraventricular conduction from ventricular tachyarrhythmias when standard electrocardiographic criteria are equivocal.

An SVT is recognized electrophysiologically by an H-V interval equaling or exceeding that recorded during normal sinus rhythm (Fig. 34-10). In contrast, during VT, the H-V interval is shorter than normal or the His deflection cannot be recorded clearly because of superimposition of the larger ventricular electrogram. Only two situations exist in which a consistently short H-V interval occurs: during retrograde activation of the His bundle from activation originating in the ventricle (i.e., PVC, ventricular pacing, or VT) and during AV conduction over an accessory pathway (preexcitation syndrome).

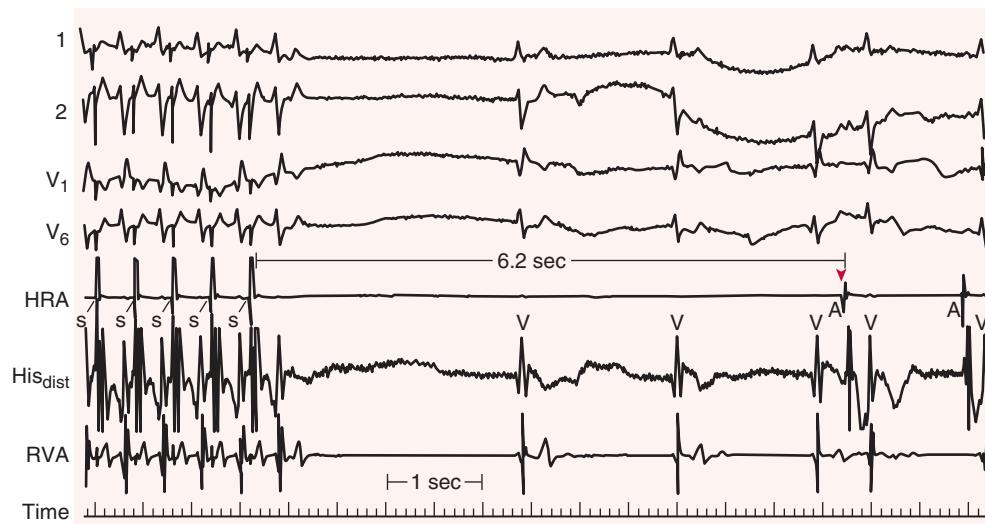


FIGURE 34-9 Abnormal sinus node function. Recordings are similar to those in Figure 34-8. The last five complexes of a 1-minute burst of atrial pacing (S) at a cycle length of 400 milliseconds are shown, after which pacing is stopped. The sinus node does not spontaneously discharge (SNRT) until 6.2 seconds later (arrowhead). Three junctional escape beats occurred before this time. His_{dist} = distal electrode pair; HRA = high right atrium; RVA = right ventricular apex.

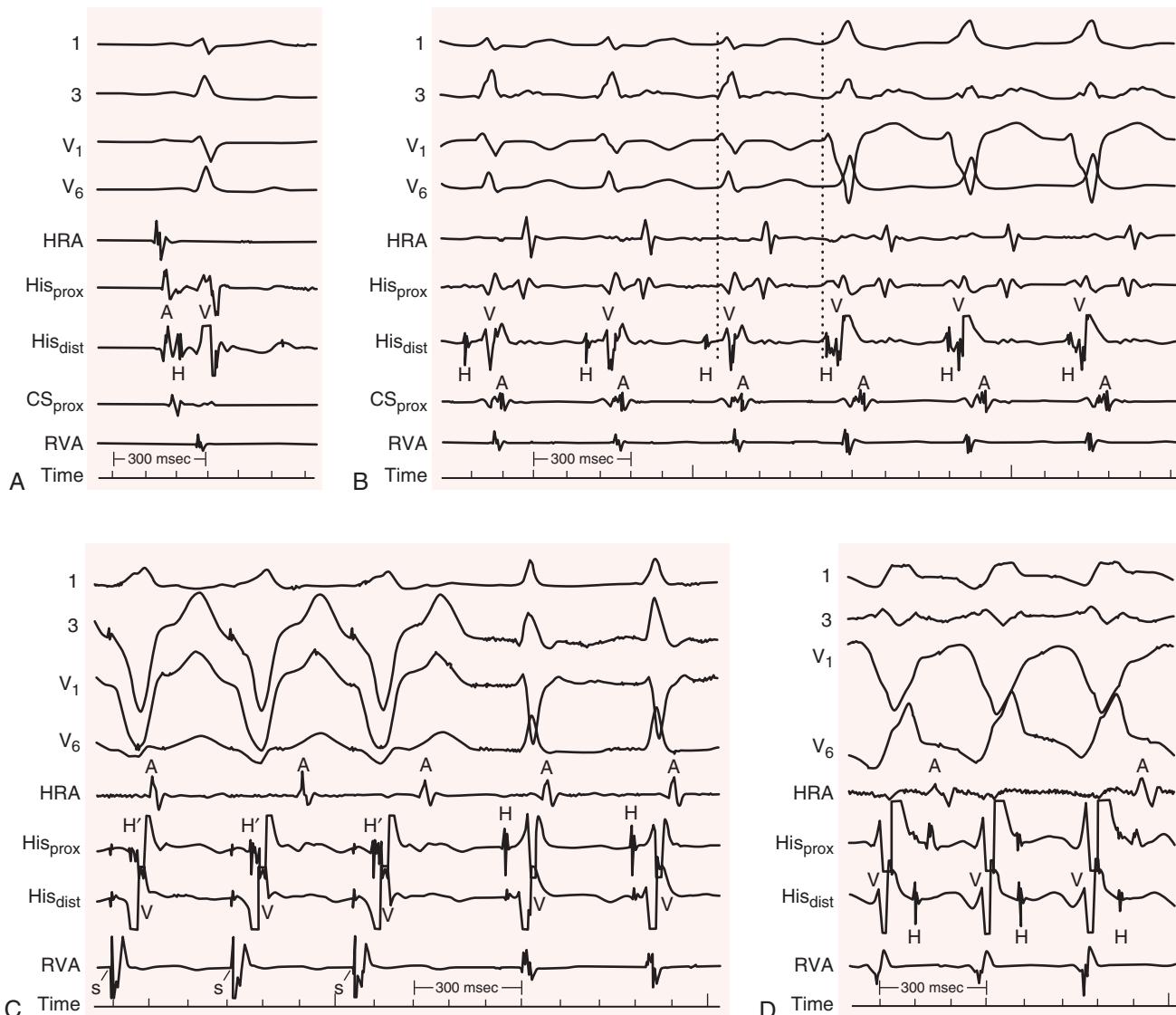


FIGURE 34-10 Bundle of His recordings in different situations similar to those in Figures 34-8 and 34-9. **A**, Baseline sinus rhythm with normal AV conduction. **B**, Orthodromic SVT with retrograde conduction over a left-sided accessory pathway throughout the tracing. The first three beats have a narrow QRS complex with a normal H-V interval; the last three QRS complexes represent a fusion of conduction over the AV node-His bundle and a slowly conducting right-sided accessory pathway. The His potential occurs after onset of the wide QRS complex (dashed lines). **C**, Three paced ventricular beats are shown with a retrograde His potential (H'), followed by initiation of AV node reentrant SVT (atrial depolarization near the end of the QRS complex, as seen in the HRA tracing). **D**, VT with delayed activation of the His potential and complete retrograde AV node block (dissociated atrial complexes). CS_{prox} = proximal coronary sinus; His_{dist} = distal electrode pair; His_{prox} = proximal electrode pair; HRA = high right atrium; RVA = right ventricular apex.



Atrial pacing at rates exceeding the tachycardia rate can demonstrate the ventricular origin of the wide-QRS tachycardia by producing fusion and capture beats and normalization of the H-V interval. The only VT that exhibits an H-V interval equal to or slightly exceeding the normal sinus H-V interval is bundle branch reentry, but His activation will be in the retrograde direction.

An EPS should be considered for the following circumstances: (1) in patients who have symptomatic, recurrent, or drug-resistant supraventricular or

ventricular tachyarrhythmias to help select optimal therapy; (2) in patients with tachyarrhythmias occurring too infrequently to permit adequate diagnostic or therapeutic assessment; (3) for differentiation of SVT and aberrant conduction from VT; (4) whenever nonpharmacologic therapy, such as the use of electrical devices, catheter ablation, or surgery, is contemplated; (5) in patients surviving an episode of cardiac arrest occurring more than 48 hours after acute myocardial infarction or without evidence of an acute Q wave myocardial infarction; and (6) for assessment of the risk for sustained VT in patients with a previous myocardial infarction, ejection fraction of 0.3 to 0.4, and nonsustained VT on an ECG. In general, EPS is not indicated in patients with long-QT syndrome and torsades de pointes.

The process of initiation and termination of SVT or VT with programmed electrical stimulation to establish precise diagnoses and help select sites for catheter ablation is the most common application of EPS in patients with tachycardia. The role of drug therapy in clinically significant arrhythmias continues to diminish; although EPS was once widely used to predict the efficacy of drug therapy in suppressing spontaneous tachycardia recurrences, the technique is now rarely used for this purpose. Noninvasive stimulation from an implanted pacemaker or defibrillator can be used to test the effects of drug therapy given in an attempt to decrease the frequency of arrhythmias, as well as to test the ICD's ability to detect and treat VT that has been slowed or otherwise altered by drug effect.

Unexplained Syncope

The three common arrhythmic causes of syncope are sinus node dysfunction, AV block, and tachyarrhythmias (see Chapter 40). Of the three, tachyarrhythmias are most reliably evaluated in the electrophysiology laboratory, followed by sinus node abnormalities and His-Purkinje block.

The cause of syncope remains uncertain in up to 50% of patients, depending in part on the extent of the evaluation. A careful, accurately performed history and physical examination begin the evaluation, followed by noninvasive tests, including a 12-lead ECG, and can lead to a diagnosis in 50% or more of patients. In a small percentage (<5%) of patients, an arrhythmia develops coincident with syncope or presyncope during 24- or 48-hour ECG monitoring, whereas a larger percentage (15%) have symptoms without an arrhythmia, thereby excluding an arrhythmic cause. Prolonged ECG monitoring with patient-activated transtelephonic event recorders that have memory loops may increase the yield. Tilt-table and stress testing can be useful for selected patients.

An EPS helps explain the cause of syncope or palpitations when it induces an arrhythmia that replicates the patient's symptoms or is associated with significant hypotension. Patients with a single episode of syncope and no evidence of structural heart disease, as well as those with a nondiagnostic EPS, have a low incidence of sudden death and an 80% remission rate over the ensuing 10 years. In those with recurrent syncope, the test is falsely negative in 20%, usually because of failure to find AV block or sinus node dysfunction.

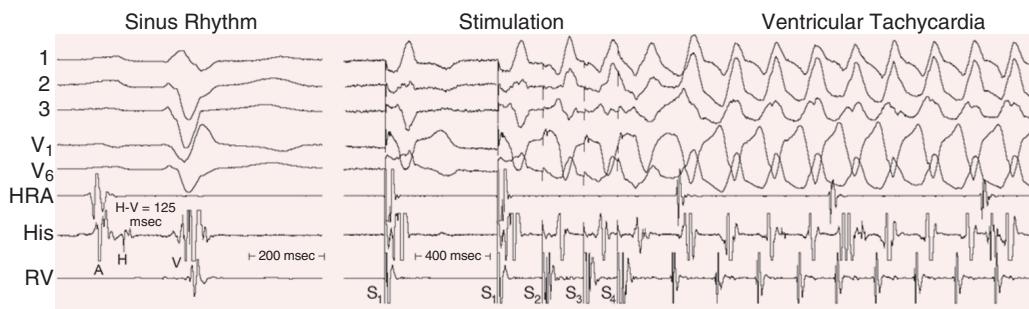


FIGURE 34-11 Multiple abnormalities in a patient with prior myocardial infarction and syncope. Recordings are similar to previous figures. In the **left panel**, a sinus rhythm complex shows a right bundle branch block and left axis deviation, with a very prolonged H-V interval of 125 milliseconds (normal, 35 to 55 milliseconds); thus heart block could have caused syncope. However, in the **right panel**, ventricular stimulation with three extrastimuli (S_2 , S_3 , S_4) induces sustained VT, another potential cause of syncope (note the different time scales in the two panels).

On the other hand, in many patients with structural heart disease, several abnormalities may be present that could account for syncope and can be diagnosed at EPS. Deciding which among these abnormalities is responsible for syncope and therefore requires therapy, and of what type, can be difficult (Fig. 34-11). Mortality and the incidence of sudden cardiac death are determined mainly by the presence of underlying heart disease.

Syncope patients considered for an EPS are those whose spells remain undiagnosed despite general, neurologic, and noninvasive cardiac evaluation, particularly if the patient has structural heart disease.¹⁷ The diagnostic yield is approximately 70% in that group but only around 12% in patients without structural heart disease. Therapy for a putative cause found during EPS prevents recurrence of syncope in approximately 80% of patients. Among arrhythmic causes of syncope, intermittent conduction disturbances are the most difficult to diagnose. EPS is poor in establishing this diagnosis despite an array of provocative tests that can be used. When tachyarrhythmias have been thoroughly sought and excluded and clinical suspicion for intermittent heart block is high (e.g., bundle branch block or long H-V interval), empiric permanent pacing may be justified.

In patients with a nondiagnostic EPS, injection of adenosine triphosphate (different from plain adenosine) distinguishes patients who may benefit from permanent pacing (those with longer than a 10-second sinus pause or AV block) from those who do not. Some have suggested that this test be performed before EPS in some cases or after a negative EPS but before an implantable loop recorder is placed.¹⁸

Palpitations

An EPS is indicated in patients with palpitations who have had a pulse documented by medical personnel to be inappropriately rapid or slow without an electrocardiographic recording and in those suspected of having clinically significant palpitations without electrocardiographic documentation.

In patients with syncope or palpitations, the sensitivity of EPS may be low but can be increased at the expense of specificity. For example, more aggressive pacing techniques (e.g., use of three or four premature stimuli), administration of drugs (e.g., isoproterenol), or left ventricular pacing can increase the likelihood of induction of ventricular arrhythmias by precipitating *nonclinical* ventricular tachyarrhythmias, such as nonsustained polymorphic or monomorphic VT or VF. Similarly, aggressive techniques during atrial pacing can induce non-specific episodes of AF or atrial flutter. A diagnostic dilemma arises when the patient's clinical, symptom-producing arrhythmia is one of these nonspecific arrhythmias that can be produced in a normal patient who has no arrhythmia. In most patients, these arrhythmias are regarded as nonclinical (i.e., nonspecific responses to intense stimulation). In other patients, such as those with hypertrophic or dilated nonischemic cardiomyopathy, they may be clinically relevant arrhythmias. However, induction of sustained SVT (e.g., AV nodal reentry, AV reciprocating tachycardia) or monomorphic VT is almost

never an artifact of stimulation, no matter how intense. Initiation of these arrhythmias in patients who have not had known spontaneous episodes of these tachycardias is uncommon and provides important information; for example, the induced tachyarrhythmia may be clinically significant and responsible for the patient's symptoms. In addition, inducible SVT episodes can have important implications for patients with ICDs that may deliver inappropriate therapy for such arrhythmias. In general, other abnormalities, such as prolonged sinus pauses after overdrive atrial pacing or His-Purkinje AV block, are not induced in patients who do not or may not experience these abnormalities spontaneously. Provocation of these abnormalities has a high degree of specificity for clinical relevance.

Complications of Electrophysiologic Studies

The risks associated with undergoing only an EPS are small. Myocardial perforation with cardiac tamponade, pseudoaneurysms at arterial access sites, and provocation of nonclinical arrhythmias can occur, each with less than a 1 per 500 incidence. The addition of therapeutic maneuvers (e.g., ablation) to the procedure increases the incidence of complications. In a European survey of 4398 patients reported from 68 institutions, the rate of ablation procedure-related complications ranged from 3.2% to 8%. Five deaths occurred within the perioperative period of the ablation. In a Heart Rhythm Society (formerly North American Society of Pacing and Electrophysiology) survey of 164 hospitals reporting in 1998 on more than 3300 patients who had undergone radiofrequency ablation, complications developed in 1% to 3%, with procedure-related deaths occurring in approximately 0.2%. In a study of 1050 patients undergoing temperature-controlled ablation for supraventricular arrhythmias, 32 (3%) had a major complication. Predictors of major complications were ejection fractions lower than 0.35 and multiple ablation targets. The improvement in the complication rate probably reflects the learning curve for radiofrequency ablation. In many centers, diagnostic EPS and even ablation procedures are performed on an outpatient basis (i.e., same-day discharge). With the increasing use of extensive ablation in the left atrium to treat AF, an increase in systemic thromboembolic complications has been observed, as have pericardial effusion and tamponade, valve damage, and phrenic nerve injury (see Chapter 37).¹⁹

DIRECT CARDIAC MAPPING: RECORDING POTENTIALS DIRECTLY FROM THE HEART

Cardiac mapping is a method whereby potentials recorded directly from the heart are spatially depicted as a function of time in an integrated manner (Fig. 34-12). The location of recording electrodes (e.g., epicardial, intramural, or endocardial) and the recording mode used (unipolar versus bipolar), as well as the method of display (isopotential, isochronal, unipolar, or bipolar voltage maps), depend on the problem under consideration.

Direct cardiac mapping by catheter electrodes or, less commonly, at the time of cardiac surgery can be used to identify and localize the areas responsible for rhythm disturbances in patients with supraventricular and ventricular tachyarrhythmias for catheter or surgical ablation, isolation, or resection. Conditions amenable to this approach include accessory pathways associated with Wolff-Parkinson-White syndrome, the pathways in AV node reentry, AV node-His bundle ablation, sites of origin of focal atrial tachycardia and VTs, isolated pathways essential for the maintenance of reentrant atrial tachycardia or VTs, and various substrates responsible for episodes of AF (Videos 34-1 and 34-2) (see Chapter 38). Mapping can also be used to delineate the anatomic course of the His bundle to avoid injury during catheter ablation or open heart surgery for repair of congenital heart disease.

Early efforts at mapping involved moving an electrode from location to location, acquiring data from a single point at a time, and comparing the timing of local activation with some reference recording, as well as other mapped sites. Knowing when enough data points had been obtained to determine where ablation should be performed relied heavily on the memory of the operator. Specialized mapping systems have now been developed that use computers to log not only the activation times and electrogram amplitude (voltage) at various points in the heart but also the physical locations from which they were obtained.²⁰ The mapping information acquired in this way can be displayed on a screen to show relative activation times in a color-coded sequence. By use of such systems, dozens or even hundreds of sites can be sampled relatively quickly, thereby leading to a clear picture of cardiac activation and potential target sites for ablation (Figs. 34-13 and 34-14). These systems can also record the signal amplitude at each site sampled to allow differentiation of normal from scarred myocardium, which can help in planning ablation strategies (Fig. 34-15). Other mapping systems can acquire data from several thousand points simultaneously by using a multipolar electrode array. This is particularly useful for hemodynamically unstable tachycardias or those that terminate spontaneously within seconds, which precludes detailed point-to-point mapping.

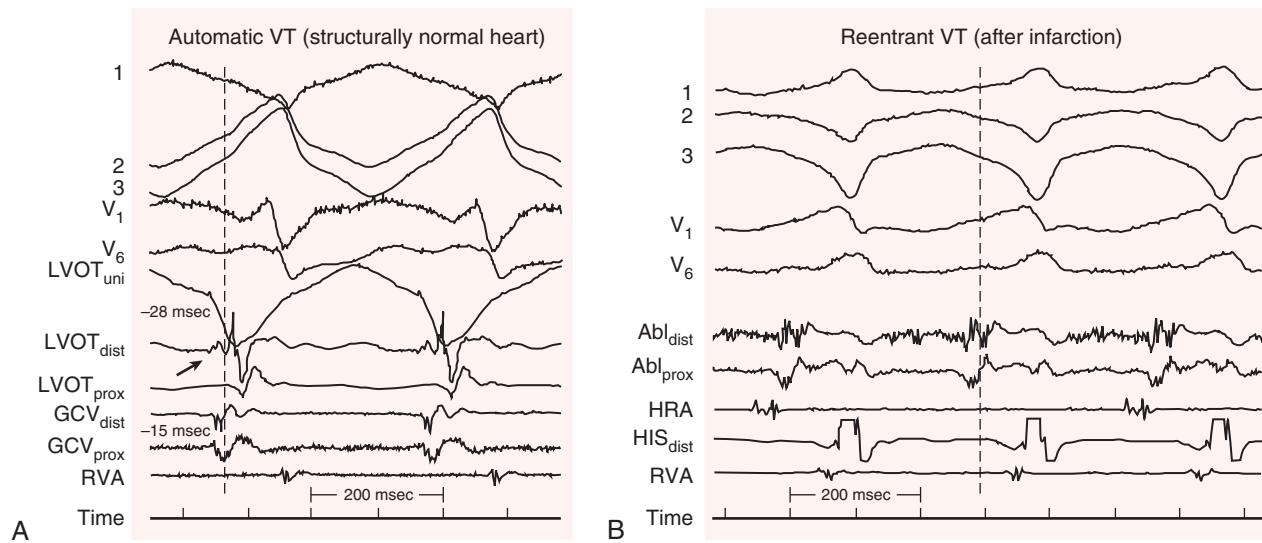


FIGURE 34-12 Endocardial catheter recordings during VT in two patients. Dashed lines denote onset of the QRS complexes. **A**, A woman without structural heart disease had a sustained VT arising from the left ventricular outflow tract (LVOT). Note the unipolar (uni) electrogram with a sharp "QS" complex and onset (arrow) of the distal bipolar recording (LVOT_{dist}) preceding the right ventricular recording, as well as recordings from a multielectrode catheter in the great cardiac vein (GCV_{dist} and GCV_{prox}) on the epicardial surface opposite the endocardial recording. Ablation at this site (LVOT) terminated the VT. **B**, A patient with reentrant VT caused by a previous inferior wall infarction. The ablation catheter (Abl_{dist}) on the inferomedial wall shows a prolonged, fragmented electrogram indicative of slow conduction that spans the entire diastolic interval between QRS complexes. Abl_{prox} = proximal ablation catheter electrodes.

**VIDEO 34-1**

Focal atrial tachycardia. The right atrium is shown in an antero-posterior view. The purple tube at top represents the superior vena cava, the circle in the middle right, the tricuspid annulus. Red indicates the head of the activation wavefront. Note the centrifugal propagation from a focus on the anterolateral right atrial wall. Ablation at the focus cured the patient from further tachycardia episodes.

VIDEO 34-2

Macroreentrant atrial tachycardia following atrial fibrillation ablation. The left atrium is shown from a right posterior oblique view; tubes emanating from the left atrium represent pulmonary veins. The red patch in the center is a diastolic corridor of reentry; the moving red border indicates the head of the advancing activation wavefront. Note the progression of the wavefront from the bottom of the diastolic corridor spreading to left and right, coming back around the top and coalescing at the top of the diastolic corridor to complete a reentrant cycle. Ablation across the diastolic corridor cured the patient from further tachycardia episodes.

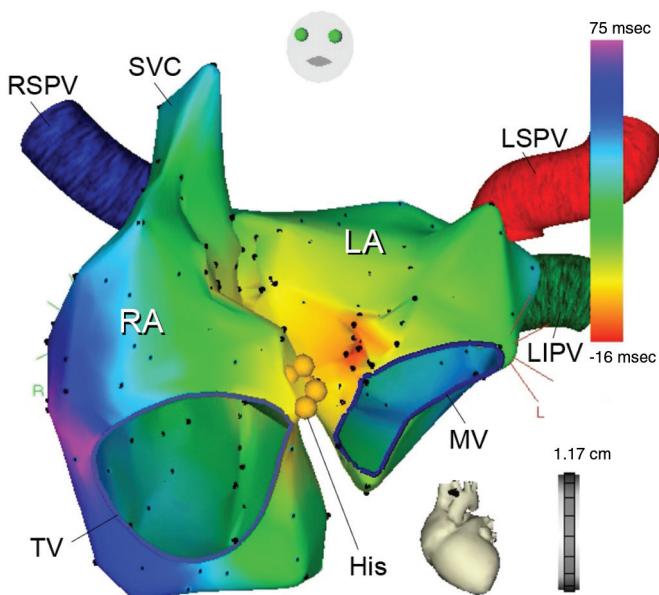


FIGURE 34-13 Electroanatomic map of focal atrial tachycardia. Both the right (RA) and left atrium (LA) are shown in an almost head-on view. A color-coded time scale of activation is shown at the right; red indicates earliest activation, purple latest. A distance scale is shown below. This atrial tachycardia arose in the anteromedial portion of the left atrium (red spot), with all other areas activated centrifugally. Ablation at this site eliminated the tachycardia. LIPV = left inferior pulmonary vein (PV); LSPV = left superior PV; MV = mitral valve ring; RSPV = right superior PV; SVC = superior vena cava; TV = tricuspid valve ring; His bundle area indicated by orange circles.

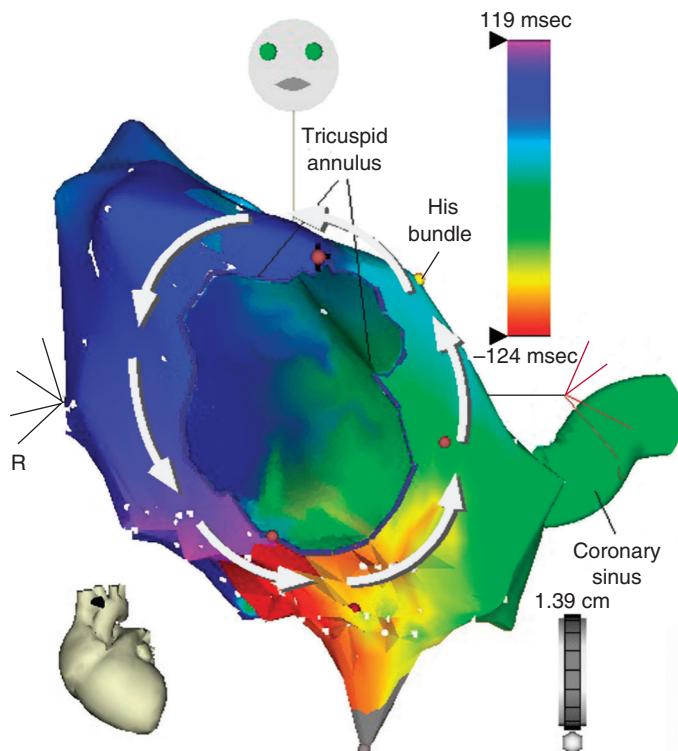


FIGURE 34-14 Electroanatomic map of reentrant atrial flutter. A left anterior oblique view of the right atrium is shown, along with depiction of the coronary sinus. See Figure 34-14 for other details. The electrical wave front propagates around the tricuspid annulus in a counterclockwise direction; in this complete circuit, early activation (in red) abuts late activation (purple) near the bottom of the tricuspid annulus. The cycle length of the tachycardia was 250 milliseconds, almost completely described by the points shown in the figure (from -124 to +119 milliseconds, a total of 243 milliseconds). A distance scale is shown below.

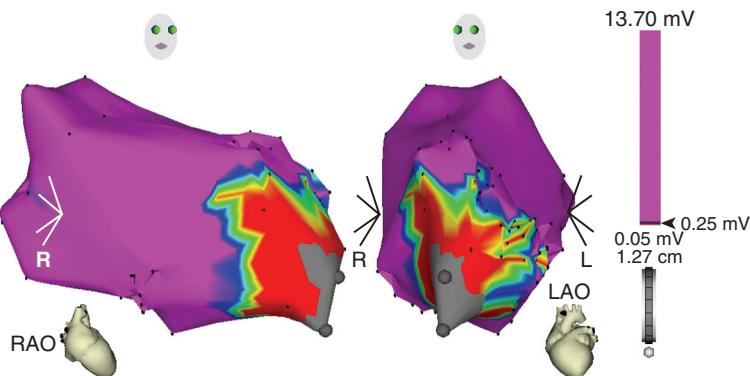


FIGURE 34-15 Electroanatomic left ventricular voltage maps during sinus rhythm. Right and left anterior oblique views are shown; the voltage scale at the right indicates normal areas (purple) versus scarred (gray) or very low-voltage areas (red, with gradation to higher voltages through green and blue). The patient had an old anteroapical myocardial infarction leading to reentrant VT that originated in the border between scar and more normal myocardium, LAO = left anterior oblique; RAO = right anterior oblique.

Pace mapping is a technique in which pacing is performed at putative sites from which arrhythmias arise (a focus) or exit (reentrant circuit). The greater the degree of "match" in QRS complexes (for VT) or intracardiac activation sequences (for atrial tachycardias), the more likely that the paced site may be an appropriate site for ablation. Software has been developed to calculate the fidelity of match of the paced complexes to the target arrhythmia; ideally, this should approach 100%. Other algorithms have been developed to analyze propagation patterns during complex arrhythmias such as AF by recording signals from multielectrode "basket" catheters in the atrium (Fig. 34-16); this has resolved many cases of an apparently chaotic rhythm to one in which erratic patterns of propagation emanate from a stable rapid source (either rotor or focus). Ablation at

these source sites can eliminate AF.²¹ Work is ongoing in this area. Finally, although computerized mapping systems acquire activation time and voltage at given sites in the heart, these features have been displayed separately. "Ripple mapping" is a new technique that integrates time and voltage information on the same display. Experience with this technique is limited, but the early results are promising.²²

Current mapping systems have the ability to both integrate previous imaging studies (computed tomography, magnetic resonance imaging) into the procedure for additional anatomic reference and derive anatomic information by moving a catheter throughout a cardiac chamber to develop a contour of its inner surface on which activation or voltage data can be plotted.

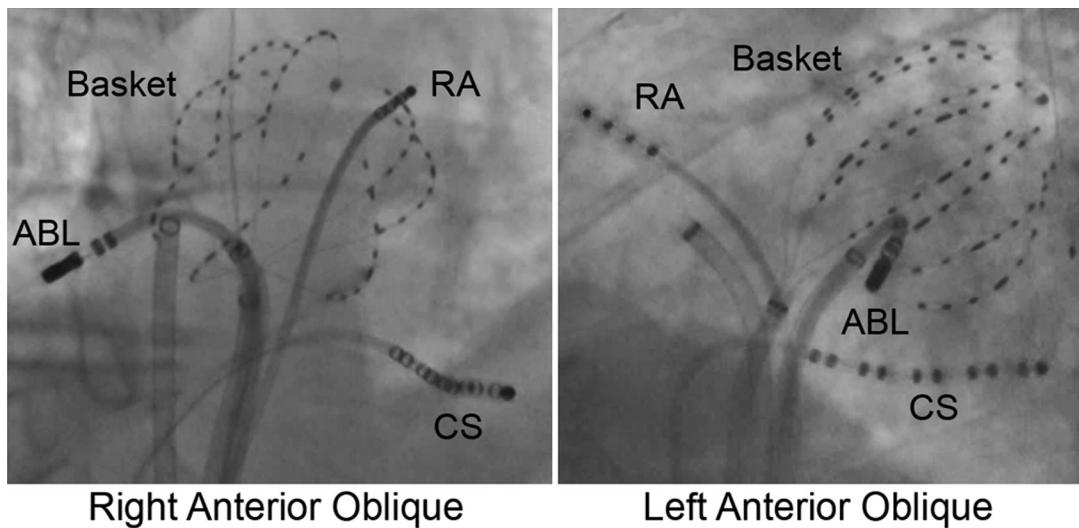


FIGURE 34-16 Basket catheter for mapping AF. Right and left anterior oblique fluoroscopic views are shown of an 8-spline, 8 electrode per spline (64 total electrodes) “basket” catheter in the left atrium; other catheters are right atrial (RA), coronary sinus (CS), and an ablation catheter (ABL) in the right inferior pulmonary vein.

References

- Das M, Zipes DP: Assessment of the patient with a cardiac arrhythmia. In Zipes DP, Jalife J (eds): *Cardiac Electrophysiology: From Cell to Bedside*. 5th ed. Philadelphia, WB Saunders, 2009, pp 831-836.
- Junqueira LF, Jr: Teaching cardiac autonomic function dynamics employing the Valsalva (Valsalva-Weber) maneuver. *Adv Physiol Educ* 32:100, 2008.
- Lacerda Gde C, Pedrosa RC, Lacerda RC, et al: Complications related to carotid sinus massage in 502 ambulatory patients. *Arq Bras Cardiol* 92:78, 2009.
- Miller JM, Das MK: Differential diagnosis of narrow and wide complex tachycardias. In Zipes DP, Jalife J (eds): *Cardiac Electrophysiology: From Cell to Bedside*. New York, Elsevier, 2013.
- Hayashi M, Denjoy I, Hayashi M, et al: The role of stress test for predicting genetic mutations and future cardiac events in asymptomatic relatives of catecholaminergic polymorphic ventricular tachycardia probands. *Europace* 14:1344, 2012.
- Miller DJ, Khan MA, Schultz LR, et al: Outpatient cardiac telemetry detects a high rate of atrial fibrillation in cryptogenic stroke. *J Neurol Sci* 324:57, 2013.
- Daoud EG, Glotzer TV, Wyse DG, et al: Temporal relationship of atrial tachyarrhythmias, cerebrovascular events, and systemic emboli based on stored device data: A subgroup analysis of TRENDS. *Heart Rhythm* 8:1416, 2011.
- Furukawa T, Maggi R, Bertolone C, et al: Additional diagnostic value of very prolonged observation by implantable loop recorder in patients with unexplained syncope. *J Cardiovasc Electrophysiol* 23:67, 2012.
- Xhyheri B, Manfrini O, Mazzolini M, et al: Heart rate variability today. *Prog Cardiovasc Dis* 55:321, 2012.
- Bauer A, Zurn CS, Schmidt G: Heart rate turbulence to guide treatment for prevention of sudden death. *J Cardiovasc Pharmacol* 55:531, 2010.
- Molnar J, Somberg JC: The dynamics of QT dispersion. *Cardiology* 113:169, 2009.
- Santangeli P, Pieroni M, Dello Russo A, et al: Correlation between signal-averaged ECG and the histologic evaluation of the myocardial substrate in right ventricular outflow tract arrhythmias. *Circ Arrhythm Electrophysiol* 5:475, 2012.
- Militaru C, Donou I, Ionescu DD: P wave signal-averaged ECG in normal population and in patients with converted atrial fibrillation. *Ann Noninvasive Electrocardiol* 16:351, 2011.
- Verrier RL, Klingenheben T, Malik M, et al: Microvolt T-wave alternans: physiological basis, methods of measurement, and clinical utility—Consensus guideline by International Society for Holter and Noninvasive Electrocadiology. *J Am Coll Cardiol* 58:1309, 2011.
- Rudy Y: Noninvasive electrocardiographic imaging of arrhythmogenic substrates in humans. *Circ Res* 112:863, 2013.
- Scheinman MM: Role of the His-Purkinje system in the genesis of cardiac arrhythmia. *Heart Rhythm* 6:1050, 2009.
- Mitro P, Kirsch P, Valocik G, Murin P: A prospective study of the standardized diagnostic evaluation of syncope. *Europace* 13:566, 2011.
- Flammang D, Church TR, De Roy L, et al: Treatment of unexplained syncope: A multicenter, randomized trial of cardiac pacing guided by adenosine 5'-triphosphate testing. *Circulation* 125:31, 2012.
- Aldhoom B, Wichterle D, Peichl P, et al: Complications of catheter ablation for atrial fibrillation in a high-volume centre with the use of intracardiac echocardiography. *Europace* 15:24, 2013.
- Bhakta D, Miller JM: Principles of electroanatomic mapping. *Indian Pacing Electrophysiol J* 8:32, 2008.
- Narayan SM, Krummen DE, Shivkumar K, et al: Treatment of atrial fibrillation by the ablation of localized sources: CONFIRM (Conventional Ablation for Atrial Fibrillation With or Without Focal Impulse and Rotor Modulation) trial. *J Am Coll Cardiol* 60:628, 2012.
- Linton NW, Koa-Wing M, Francis DP, et al: Cardiac ripple mapping: A novel three-dimensional visualization method for use with electroanatomic mapping of cardiac arrhythmias. *Heart Rhythm* 6:1754, 2009.

GUIDELINES

Ambulatory Electrocardiographic and Electrophysiologic Testing

John M. Miller and Douglas P. Zipes

Guidelines for the appropriate use of ambulatory electrocardiography (ECG) were first published by the American College of Cardiology/American Heart Association (ACC/AHA) in 1989¹ and updated in 1999.² In conjunction with other professional societies, the ACC/AHA issued a statement of requirements for clinical competence in ambulatory ECG in 2001.³ Guidelines for performance of electrophysiologic testing were first published in 1985⁴ and updated in 1989 and 1995.⁵ A clinical competence statement was issued by the ACC/AHA for electrophysiologic studies and catheter ablation in 2000⁶; this was updated by a statement on training in electrophysiology, cardiac pacing, and arrhythmia management in 2006⁷ and again in 2008.⁸ The AHA and the North American Society of Pacing and Electrophysiology (NASPE, now the Heart Rhythm Society) made recommendations on safety-related topics, such as restrictions on driving,

for patients with arrhythmia in 1996⁹ and updated them in 2007¹⁰ (covered in the Guidelines in **Chapter 38**). Since then, efforts to update the guidelines have focused on appropriate indications for the use of pacemakers and implantable cardioverter-defibrillators (ICDs) because of rapid advances in knowledge about the ability of ICDs to improve the survival of patients with arrhythmia with or without electrophysiologic testing. These guidelines were issued in 2002¹¹ and updated in 2008 and 2013.¹² Guidelines on ICD use are further addressed in **Chapter 38**.

The standard ACC/AHA classification system is used for the following indications:

- Class I: conditions for which there is evidence and/or general agreement that the test is useful and effective
- Class II: conditions for which there is conflicting evidence and/or a divergence of opinion about the usefulness or efficacy of performing the test
- Class IIa: weight of evidence or opinion in favor of usefulness or efficacy
- Class IIb: usefulness or efficacy less well established by evidence or opinion
- Class III: conditions for which there is evidence and/or general agreement that the test is not useful or effective and in some cases may be harmful

Three levels are used to rate the evidence on which recommendations have been based. Level A recommendations are derived from data from multiple randomized clinical trials, level B recommendations are derived from a single randomized trial or nonrandomized studies, and level C recommendations are based on the consensus opinion of experts.

AMBULATORY ELECTROCARDIOGRAPHY

The evolution of guidelines for the use of ambulatory ECG from 1989 to 1999 reflected important progress in several areas, including the following:

- Understanding of the limited usefulness of suppression of ventricular ectopy with drug therapy
- Solid-state digital technology, which facilitates transtelephonic transmission of electrocardiographic data
- Technical advances in long-term event recorders
- Improved signal quality and interpretation
- Improved computer arrhythmia interpretation

- Increasingly sophisticated monitoring capacity of pacemakers and ICDs

As a result of progress in these areas and increased knowledge about arrhythmias, ambulatory ECG is now considered to be of uncertain appropriateness for many indications for which it was once an accepted strategy.

Diagnosis

In assessing symptoms that may be caused by arrhythmias, ambulatory ECG (Holter) monitoring is clearly established for the evaluation of syncope (**Table 34G-1; see Chapter 40**). A 2006 AHA/ACC Foundation scientific statement on the evaluation of syncope stipulates that the type and duration of ambulatory ECG monitoring are dictated by the frequency of symptoms.¹³ Holter monitors (24 to 48 hours) are appropriate for episodes that occur at least daily and event recorders (30 to 60 days) for episodes that occur at least monthly. Implantable loop recorders inserted subcutaneously can record bipolar ECG signals for up to 14 months. In patients with unexplained syncope, use of an implantable loop recorder for 1 year is more likely to

TABLE 34G-1 ACC/AHA Guidelines on Ambulatory Electrocardiography for Assessment of Symptoms and Arrhythmias

INDICATION	CLASS I (INDICATED)	CLASS IIA (GOOD SUPPORTIVE EVIDENCE)	CLASS IIB (WEAK SUPPORTIVE EVIDENCE)	CLASS III (NOT INDICATED)
Assessment of symptoms possibly related to rhythm disturbances	Patients with unexplained syncope, near-syncope, or episodic dizziness in whom the cause is not obvious Patients with unexplained recurrent palpitation		Patients with episodic shortness of breath, chest pain, or fatigue that is not otherwise explained Patients with neurologic events when transient atrial fibrillation or flutter is suspected Patients with symptoms such as syncope, near-syncope, episodic dizziness, or palpitation in whom a probable cause other than an arrhythmia has been identified but in whom symptoms persist despite treatment of this other cause	Patients with symptoms such as syncope, near-syncope, episodic dizziness, or palpitation in whom other causes have been identified by history, physical examination, or laboratory tests Patients with cerebrovascular accidents but without other evidence of arrhythmia
Arrhythmia detection to assess risk for future cardiac events in patients without symptoms from arrhythmia			Post-MI patients with LV dysfunction (ejection fraction <40%) Patients with CHF Patients with idiopathic hypertrophic cardiomyopathy	Patients who have sustained myocardial contusion Systemic hypertensive patients with LV hypertrophy Post-MI patients with normal LV function Preoperative arrhythmia evaluation of patients for noncardiac surgery Patients with sleep apnea Patients with valvular heart disease
Measurement of heart rate variability to assess risk for future cardiac events in patients without symptoms from arrhythmia			Post-MI patients with LV dysfunction Patients with CHF Patients with idiopathic hypertrophic cardiomyopathy	Post-MI patients with normal LV function Diabetic subjects to evaluate for diabetic neuropathy Patients with rhythm disturbances that preclude HRV analysis (e.g., atrial fibrillation)
Assessment of antiarrhythmic therapy	To assess antiarrhythmic drug response in individuals in whom the baseline frequency of arrhythmia has been characterized as reproducible and of sufficient frequency to permit analysis	To detect proarrhythmic responses to antiarrhythmic therapy in patients at high risk	To assess rate control during atrial fibrillation To document recurrent or asymptomatic nonsustained arrhythmias during therapy in the outpatient setting	

CHF = congestive heart failure; HRV = heart rhythm variability; LV = left ventricular; MI = myocardial infarction.

TABLE 34G-2 ACC/AHA Guidelines on Ambulatory Electrocardiography for Assessment of Pacemaker and Implantable Cardioverter-Defibrillator Function

CLASS	INDICATION
Class I (indicated)	Evaluation of frequent symptoms of palpitations, syncope, or near-syncope to assess device function, to exclude myopotential inhibition and pacemaker-mediated tachycardia, and to assist in the programming of enhanced features, such as rate responsiveness and automatic mode switching Evaluation of suspected component failure or malfunction when device interrogation is not definitive in establishing a diagnosis To assess response to adjunctive pharmacologic therapy in patients receiving frequent ICD therapy
Class IIa (good supportive evidence)	
Class IIb (weak supportive evidence)	Evaluation of immediate postoperative pacemaker function after pacemaker or ICD implantation as an alternative or adjunct to continuous telemetric monitoring Evaluation of the rate of supraventricular arrhythmias in patients with implanted defibrillators
Class III (not indicated)	Assessment of ICD or pacemaker malfunction when device interrogation, electrocardiogram, or other available data (e.g., chest radiograph) are sufficient to establish an underlying cause or diagnosis Routine follow-up in asymptomatic patients

identify the mechanism of syncope than is a conventional approach that uses Holter or event monitors and electrophysiologic testing and is cost-effective.

Ambulatory ECG is also supported for the evaluation of recurrent palpitations, particularly if the frequency of these symptoms makes it reasonably likely that they can be correlated with the tracings obtained during a 24-hour monitoring period. The guidelines note that data on the use of ambulatory ECG for near-syncope or dizziness are insufficient to describe the diagnostic performance of this technology for patients with such symptoms.

The ACC/AHA guidelines explicitly discourage ambulatory ECG for patients with syncope or palpitations if other causes have been identified during the clinical evaluation and for patients with cerebrovascular accidents and no other evidence of arrhythmia. The guidelines seek to reduce performance of ambulatory ECG “for completeness” in such cases. Little support is provided for use of ambulatory ECG in cases in which the cause of the patient’s symptoms is unclear but in which the likelihood of detecting an unsuspected arrhythmia is low (class IIb indications).

Assessment of Risk

The ACC/AHA guidelines discouraged the use of ambulatory ECG for either arrhythmia detection or analysis of heart rhythm variability for the purpose of risk assessment in patients without symptoms of arrhythmia, even if they had cardiovascular conditions such as myocardial contusions, left ventricular hypertrophy, or valvular heart disease (see Table 34G-1). Routine use for patients in whom arrhythmia is a common cause of death (left ventricular dysfunction, hypertrophic cardiomyopathy) was considered a class IIb indication. These recommendations preceded data demonstrating the beneficial impact of ICDs for patients with left ventricular dysfunction after acute myocardial infarction even without symptoms of arrhythmia. These more recent findings are leading to an expanded role for ambulatory ECG in determining which asymptomatic patients most need these expensive devices.

Efficacy of Antiarrhythmic Therapy

In the absence of data demonstrating that oral antiarrhythmic therapy can improve survival through control of ventricular arrhythmias, ambulatory ECG has a diminished role as a test for evaluation of the efficacy of treatment (see Table 34G-1). Oral antiarrhythmic agents are important for control of supraventricular arrhythmias, but most patients with such arrhythmias do not have episodes every day. Event recorders can be useful for documenting the relationship between symptoms and recurrent arrhythmia and the interval between episodes, which can help guide therapy.

The guidelines provide some support of the use of ambulatory ECG for detection of proarrhythmia during initiation of drug therapy, but

TABLE 34G-3 ACC/AHA Guidelines on Monitoring for Ischemia

CLASS	INDICATION
Class I (indicated)	
Class IIa (good supportive evidence)	Patients with suspected variant angina
Class IIb (weak supportive evidence)	Evaluation of patients with chest pain who cannot exercise Preoperative evaluation for vascular surgery in patients who cannot exercise Patients with known coronary artery disease and atypical chest pain syndrome
Class III (not indicated)	Initial evaluation of patients with chest pain who are able to exercise Routine screening of asymptomatic subjects

patients at high risk for such complications tend to have these medications initiated as inpatients.

Assessment of Pacemaker and Implantable Cardioverter-Defibrillator Function

Ambulatory ECG was considered to be appropriate for evaluation of the function of pacemakers and ICDs (see Chapter 36), but the role of ambulatory ECG is being reduced by increasing the diagnostic and monitoring functions being built into these devices, especially with the use of remote monitoring. Ambulatory ECG can provide useful information by correlating symptoms with device activity and by detecting abnormalities in sensing and capture during chronic follow-up (Table 34G-2). However, the ACC/AHA guidelines emphasize that ambulatory ECG should not be used when data available from device interrogation are sufficient to guide clinical management.

Monitoring for Myocardial Ischemia

The 1999 ACC/AHA guidelines do not provide strong support of any indications for routine clinical use of ambulatory ECG monitoring for myocardial ischemia (Table 34G-3). The only indication for which the task force thought that there was good supportive evidence was suspected variant angina. This technology was not considered a first-choice alternative to exercise testing for patients who are unable to exercise.

Clinical Competence

The ACC/AHA statement on clinical competence recommended that trainees interpret at least 150 ambulatory electrocardiograms under

supervision to acquire minimal competence in this technology.³ A minimum of 25 test interpretations per year was recommended to maintain competence.

ELECTROPHYSIOLOGIC PROCEDURES FOR DIAGNOSIS

The ACC/AHA guidelines for the use of intracardiac electrophysiologic procedures from 1985⁴ and 1995⁵ reflect the emerging role of catheter ablation as a therapeutic strategy but do not fully reflect the reduced importance of antiarrhythmic medications and the growing role of ICDs that have occurred. Nevertheless, most of the basic themes of these guidelines remain valid. An updated clinical competence statement for performing these procedures was issued in 2006.¹⁴

Evaluation of Sinus Node Function

Clinical evaluation of sinus node dysfunction is often difficult because of the episodic nature of symptomatic abnormalities and the wide

variability in sinus node function in asymptomatic individuals. Invasive tests of sinus function can test the ability of the sinus node to recover from overdrive suppression and assess sinoatrial conduction by introducing atrial extrastimuli or by atrial pacing.

The ACC/AHA guidelines consider electrophysiologic studies of sinus node function most appropriate for patients in whom dysfunction is suspected but not proved after a noninvasive evaluation (**Table 34G-4**). In contrast, the guidelines consider such studies inappropriate when a documented bradycardia has been found to be correlated with the patient's symptoms and management is unlikely to be influenced by an electrophysiologic study. Studies are also considered inappropriate in asymptomatic patients and those who have sinus pauses only during sleep. When bradycardias were recognized as the cause of the patient's symptoms, electrophysiologic studies were considered to have possible but uncertain appropriateness (class II) if such data might refine treatment choices.

Acquired Atrioventricular Block

The ACC/AHA guidelines emphasize that electrophysiologic studies are inappropriate (class III) when ECG findings correlate with

TABLE 34G-4 ACC/AHA Guidelines on Clinical Intracardiac Electrophysiologic Studies for Evaluation of Specific Electrocardiographic Abnormalities

INDICATION	CLASS I (APPROPRIATE)	CLASS II (EQUIVOCAL)	CLASS III (INAPPROPRIATE)
Evaluation of sinus node function	Symptomatic patients in whom sinus node dysfunction is suspected as the cause of symptoms but a causal relationship between an arrhythmia and the symptoms has not been established after appropriate evaluation	Patients with documented sinus node dysfunction in whom evaluation of AV or ventriculoatrial conduction or susceptibility to arrhythmias may aid in selection of the most appropriate pacing modality Patients with electrocardiographically documented sinus bradycardias to determine whether abnormalities are caused by intrinsic disease, autonomic nervous system dysfunction, or effects of drugs to help select therapeutic options Symptomatic patients with known sinus bradycardias to evaluate potential for other arrhythmias as the cause of symptoms	Symptomatic patients in whom an association between symptoms and a documented bradycardia has been established and the choice of therapy would not be affected by the results of an electrophysiologic study Asymptomatic patients with sinus bradycardias or sinus pauses observed only during sleep, including sleep apnea
Acquired AV block	Symptomatic patients in whom His-Purkinje block, suspected as a cause of symptoms, has not been established Patients with second- or third-degree AV block treated with a pacemaker who remain symptomatic and in whom another arrhythmia is suspected as a cause of the symptoms	Patients with second- or third-degree AV block in whom knowledge of the site of block or its mechanism or response to pharmacologic or other temporary intervention may help in directing therapy or assessing prognosis Patients with premature, concealed junctional depolarizations suspected as the cause of a second- or third-degree AV block pattern (e.g., pseudo-AV block)	Symptomatic patients in whom the symptoms and presence of AV block are correlated by ECG findings Asymptomatic patients with transient AV block associated with sinus slowing (e.g., nocturnal type I second-degree AV block)
Chronic intraventricular conduction delay	Symptomatic patients in whom the cause of symptoms is not known	Asymptomatic patients with bundle branch block in whom pharmacologic therapy that could increase conduction delay or produce heart block is contemplated	Asymptomatic patients with intraventricular conduction delay Symptomatic patients whose symptoms can be correlated with or excluded by ECG events
Narrow-QRS tachycardia (QRS complex <0.12 sec)	Patients with frequent or poorly tolerated episodes of tachycardia who do not adequately respond to drug therapy and for whom information about the site of origin, mechanism, and electrophysiologic properties of pathways of the tachycardia is essential for choosing appropriate therapy (e.g., drugs, catheter ablation, pacing, or surgery) Patients who prefer ablative therapy to pharmacologic treatment	Patients with frequent episodes of tachycardia requiring drug treatment for whom there is concern about proarrhythmia or effects of the antiarrhythmic drug on the sinus node or AV conduction	Patients with tachycardias easily controlled by vagal maneuvers and/or well-tolerated drug therapy who are not candidates for nonpharmacologic therapy

Continued

TABLE 34G-4 ACC/AHA Guidelines on Clinical Intracardiac Electrophysiologic Studies for Evaluation of Specific Electrocardiographic Abnormalities—cont'd

INDICATION	CLASS I (APPROPRIATE)	CLASS II (EQUIVOCAL)	CLASS III (INAPPROPRIATE)
Wide-complex tachycardias	Patients with wide-QRS complex tachycardia in whom the correct diagnosis is unclear after analysis of available ECG tracings and for whom knowledge of the correct diagnosis is necessary for care	None	Patients with VT or supraventricular tachycardia with aberrant conduction or preexcitation syndromes diagnosed with certainty by ECG criteria and for whom invasive electrophysiologic data would not influence therapy; however, data obtained at baseline electrophysiologic study in these patients might be appropriate as a guide for subsequent therapy
Prolonged–QT interval syndrome	None	Identification of proarrhythmic effect of a drug in patients experiencing sustained VT or cardiac arrest while receiving the drug Patients who have equivocal abnormalities in QT interval duration or TU wave configuration, along with syncope or symptomatic arrhythmias, in whom the effects of catecholamine may unmask a distinct QT abnormality	Patients with clinically manifest congenital QT prolongation, with or without symptomatic arrhythmias Patients with acquired prolonged–QT syndrome with symptoms closely related to an identifiable cause or mechanism
Wolff-Parkinson-White syndrome	Patients being evaluated for catheter ablation or surgical ablation of an accessory pathway Patients with ventricular preexcitation who have survived cardiac arrest or who have unexplained syncope Symptomatic patients in whom determination of the mechanism of arrhythmia or knowledge of the electrophysiologic properties of the accessory pathway and normal conduction system would help in determining appropriate therapy	Asymptomatic patients with a family history of sudden cardiac death or with ventricular preexcitation but no spontaneous arrhythmia who engage in high-risk occupations or activities and in whom knowledge of the electrophysiologic properties of the accessory pathway or inducible tachycardia may help determine recommendations for further activities or therapy Patients with ventricular preexcitation who are undergoing cardiac surgery for other reasons	Asymptomatic patients with ventricular preexcitation, except those in class II
Ventricular premature complexes, couplets, and nonsustained VT	None	Patients with other risk factors for future arrhythmic events, such as a low ejection fraction, positive signal-averaged electrocardiogram, and nonsustained VT on ambulatory ECG recordings in whom electrophysiologic studies will be used for further risk assessment and for guiding therapy in patients with inducible VT Patients with highly symptomatic, uniform-morphology premature ventricular complexes, couplets, and nonsustained VT who are considered potential candidates for catheter ablation	Asymptomatic or mildly symptomatic patients with premature ventricular complexes, couplets, and nonsustained VT without other risk factors for sustained arrhythmias

VT = ventricular tachycardia.

symptoms and the findings from electrophysiologic studies are unlikely to alter management (e.g., documentation of His bundle conduction rarely improves the management of a patient whose other clinical data indicate that placement of a permanent pacemaker is warranted because of symptomatic advanced atrioventricular [AV] block). Similarly, electrophysiologic studies are not appropriate for asymptomatic patients with mild degrees of AV block who are not likely to warrant pacemaker implantation. According to these guidelines, electrophysiologic studies of AV conduction should be performed when a relationship between symptoms and AV block is a reasonable possibility but has not been proved.

Chronic Intraventricular Delay

According to the ACC/AHA guidelines, the main role of electrophysiologic testing in patients with prolonged H-V intervals is not to predict

future complications but to determine whether the symptoms of arrhythmia are caused by conduction delay or block versus some other arrhythmia. The only class I (clearly appropriate) indication for electrophysiologic testing is symptomatic patients for whom the cause of symptoms is not known. The guidelines specifically discourage such testing of asymptomatic patients and provide only equivocal support for asymptomatic patients with bundle branch block in whom treatment with drugs that might increase conduction delay is being considered.

Narrow- and Wide-QRS Complex Tachycardia

The ACC/AHA guidelines define different roles for electrophysiologic testing in patients with narrow- and wide-complex tachycardias. In narrow-QRS tachycardia, the site of abnormal impulse formation or

the reentry circuit can often be determined from information on the 12-lead electrocardiogram. Thus electrophysiologic testing was considered more appropriate as a guide to therapy in this setting than as a tool for diagnosis. Class I indications for electrophysiologic testing include patients with recurrent tachycardia for whom data from testing may help clinicians choose among drug therapy, catheter ablation, pacing, and surgery. However, testing is not considered useful for patients whose tachycardias are controlled by vagal maneuvers or medications and who are not candidates for nonpharmacologic therapy.

In wide-complex tachycardias, correct diagnosis is occasionally not possible from ECG tracings alone. However, electrophysiologic testing permits accurate diagnosis in virtually all patients. Because knowledge of the mechanism of the arrhythmia is essential for selection of optimal therapy, electrophysiologic testing was considered appropriate (class I) for the diagnosis of wide-complex tachycardias in these guidelines. However, when the diagnosis is clear from other data and electrophysiologic testing is not likely to influence therapy, the guidelines consider it inappropriate.

Prolonged QT Intervals

The ACC/AHA guidelines do not consider routine use of electrophysiologic testing appropriate for any indications in patients with prolonged QT intervals. Whether catecholamine infusion during testing is useful for revealing patients who are at high risk for complications or whether electrophysiologic testing can be used to evaluate proarrhythmic effects in this population is considered uncertain.

Wolff-Parkinson-White Syndrome

Electrophysiologic testing is useful for patients with this syndrome for both diagnosis and planning of therapy. The ACC/AHA guidelines consider electrophysiologic testing appropriate for patients who are candidates for catheter or surgical ablation, for those who have had cardiac arrests or unexplained syncope, or for patients whose management might be altered by knowledge of the electrophysiologic properties of the accessory pathway and normal conduction system. For asymptomatic patients, however, electrophysiologic studies are deemed inappropriate except in special situations, such as patients with high-risk occupations or those with a family history of sudden cardiac death. More recently recognized entities, such as Brugada

syndrome, catecholaminergic tachycardia, and right ventricular cardiomyopathy, were not considered.

Nonsustained Ventricular Tachycardia

For patients with ventricular premature complexes, couplets, and nonsustained ventricular tachycardia, the usefulness of electrophysiologic testing is compromised by the lack of therapeutic strategies that have been shown to improve outcomes. There are no clearly appropriate indications for electrophysiologic studies in these patients, and the guidelines discourage testing in patients without other risk factors for sustained arrhythmias. Research published since these guidelines suggests that exceptions would include patients who fit the MADIT (Multicenter Automatic Defibrillator Implantation Trial) or MUSTT (Multicenter Unsustained Tachycardia Trial) criteria. For certain patients with other data suggesting an adverse prognosis, electrophysiologic testing is thought to have possible but unproven appropriateness (class II).

Unexplained Syncope

In patients with unexplained syncope and structural heart disease (see Chapter 40), recent ACC/AHA guidelines on the evaluation of syncope¹⁰ recommend a low threshold for the use of electrophysiologic testing (Table 34G-5). In patients without structural heart disease, the yield of electrophysiologic testing is low. Thus the guidelines recommend a higher threshold for use of electrophysiologic studies in such patients and suggest that head-up tilt testing may be a more useful test. However, given the low risk associated with electrophysiologic testing and the high risk for potentially harmful recurrent syncope, electrophysiologic testing may be beneficial for patients with a malignant episode of syncope.¹³

Survivors of Cardiac Arrest

The ACC/AHA guidelines consider electrophysiologic testing appropriate for patients who are survivors of cardiac arrest (see Chapter 39) other than in the earliest phase of acute myocardial infarction (see Table 34G-5). Since publication of these guidelines, acceptance of the usefulness of ICDs has become more widespread, and many of these patients receive such a device without electrophysiologic testing or undergo limited electrophysiologic testing at device

TABLE 34G-5 ACC/AHA Guidelines on Clinical Intracardiac Electrophysiologic Studies for Evaluation of Clinical Syndromes

INDICATION	CLASS I (APPROPRIATE)	CLASS II (EQUIVOCAL)	CLASS III (INAPPROPRIATE)
Unexplained syncope	Patients with suspected structural heart disease and syncope that remain unexplained after appropriate evaluation	Patients with recurrent unexplained syncope but without structural heart disease and a negative head-up tilt test result	Patients with a known cause of syncope for whom treatment will not be guided by electrophysiologic testing
Survivors of cardiac arrest	Patients surviving cardiac arrest without evidence of acute Q wave MI Patients surviving cardiac arrest occurring more than 48 hr after the acute phase of MI in the absence of recurrent ischemic events	Patients surviving cardiac arrest caused by bradyarrhythmia Patients surviving cardiac arrest thought to be associated with a congenital repolarization abnormality (long-QT syndrome) in whom the results of noninvasive diagnostic testing are equivocal	Patients surviving a cardiac arrest that occurred during the acute phase (<48 hr) of MI Patients with cardiac arrest resulting from clearly definable specific causes, such as reversible ischemia, severe valvular aortic stenosis, or noninvasively defined congenital or acquired long-QT syndrome
Unexplained palpitations	Patients with palpitations who have their pulse rate documented by medical personnel as inappropriately rapid and in whom ECG recordings fail to document the cause of the palpitations Patients with palpitations preceding a syncopal episode	Patients with clinically significant palpitations, suspected to be of cardiac origin in whom the symptoms are sporadic and cannot be documented; studies performed to determine mechanisms of arrhythmias, direct or provide therapy, or assess prognosis	Patients with palpitations documented to be due to extracardiac causes (e.g., hyperthyroidism)

MI = myocardial infarction.

implantation. The guidelines consider electrophysiologic studies inappropriate when cardiac arrest has occurred within the first 48 hours of myocardial infarction or when the cardiac arrest results from clearly definable specific causes.

Unexplained Palpitations

The procedure of choice to determine the cause of palpitations is ambulatory ECG according to the ACC/AHA guidelines. The guidelines suggest that electrophysiologic testing should be reserved for patients with palpitations that are associated with syncope or for those in whom electrocardiograms have failed to capture a cause of the palpitations but who have been noted to have a rapid pulse rate by medical personnel (see Table 34G-5). Electrophysiologic testing is considered to be of equivocal value in patients with symptoms so sporadic that they cannot be documented while ambulatory ECG is performed.

ELECTROPHYSIOLOGIC STUDIES FOR THERAPEUTIC INTERVENTION

The 1995 ACC/AHA guidelines on the appropriateness of electrophysiologic studies for guidance of drug therapy and implantable electrical devices do not reflect the decline in the role of oral antiarrhythmic therapy and the rise in the use of ICDs for the treatment of patients who have experienced cardiac arrest (Table 34G-6). However, the guideline recommendations for the role of catheter ablation remain valid. Characteristics that are common among appropriate indications include supraventricular arrhythmias, including atrial fibrillation, that are symptomatic; that cannot be controlled with medications because of limited effectiveness, side effects, or inconvenience; or that have caused sudden cardiac death.¹⁵ Catheter ablation is also useful for the same reasons in some patients with ventricular tachycardia when it occurs in the absence of structural heart disease, and

TABLE 34G-6 ACC/AHA Guidelines on Clinical Intracardiac Electrophysiologic Studies for Therapeutic Intervention

INDICATION	CLASS I (APPROPRIATE)	CLASS II (EQUIVOCAL)	CLASS III (INAPPROPRIATE)
Guidance of drug therapy	Patients with sustained VT or cardiac arrest, especially those with prior MI Patients with AVNRT, AV reentrant tachycardia using an accessory pathway, or atrial fibrillation associated with an accessory pathway for whom chronic drug therapy is planned	Patients with sinus node reentrant tachycardia, atrial tachycardia, atrial fibrillation, or atrial flutter without ventricular preexcitation syndrome for whom chronic drug therapy is planned Patients with arrhythmias not inducible during controlled electrophysiologic study for whom drug therapy is planned	Patients with isolated atrial or ventricular premature complexes Patients with ventricular fibrillation with a clearly identified reversible cause
Patients who are candidates for or who have implantable electrical devices	Patients with tachyarrhythmias before and during implantation and final (predischarge) programming of an electrical device to confirm its ability to perform as anticipated Patients with an implanted electrical antitachyarrhythmia device in whom changes in status or therapy may have influenced the continued safety and efficacy of the device Patients who have a pacemaker to treat a bradyarrhythmia and receive a cardioverter-defibrillator to test for device interactions	Patients with previously documented indications for pacemaker implantation to test for the most appropriate long-term pacing mode and sites to optimize symptomatic improvement and hemodynamics	Patients who are not candidates for device therapy
Indications for catheter ablation procedures	Patients with symptomatic atrial tachyarrhythmias who have inadequately controlled ventricular rates unless primary ablation of the atrial tachyarrhythmia is possible Patients with symptomatic atrial tachyarrhythmias such as those above but in whom drugs are not tolerated or the patient does not wish to take them, even though the ventricular rate can be controlled Patients with symptomatic nonparoxysmal junctional tachycardia that is drug resistant or the patient is drug intolerant or does not wish to take it Patients resuscitated from sudden cardiac death caused by atrial flutter or atrial fibrillation with a rapid ventricular response in the absence of an accessory pathway	Patients with a dual-chamber pacemaker and pacemaker-mediated tachycardia that cannot be treated effectively by drugs or by reprogramming the pacemaker	Patients with atrial tachyarrhythmias responsive to drug therapy acceptable to the patient
Radiofrequency catheter ablation for AVNRT	Patients with symptomatic sustained AVNRT that is drug resistant or the patient is drug intolerant or does not desire long-term drug therapy	Patients with sustained AVNRT identified during electrophysiologic study or catheter ablation of another arrhythmia Finding of dual-AV nodal pathway physiology and atrial echoes but without AVNRT during electrophysiologic study in patients clinically suspected of having AVNRT	Patients with AVNRT responsive to drug therapy that is well tolerated and preferred by the patient over ablation Finding of dual-AV nodal pathway physiology (with or without echo complexes) during electrophysiologic study in patients in whom AVNRT is not suspected clinically

TABLE 34G-6 ACC/AHA Guidelines on Clinical Intracardiac Electrophysiologic Studies for Therapeutic Intervention—cont'd

INDICATION	CLASS I (APPROPRIATE)	CLASS II (EQUIVOCAL)	CLASS III (INAPPROPRIATE)
Ablation of atrial tachycardia, flutter, and fibrillation: atrium/atrial sites	Patients with atrial tachycardia that is drug resistant or the patient is drug intolerant or does not desire long-term drug therapy Patients with atrial flutter that is drug resistant or the patient is drug intolerant or does not desire long-term drug therapy	Atrial flutter or atrial tachycardia associated with paroxysmal atrial fibrillation when the tachycardia is drug resistant or the patient is drug intolerant or does not desire long-term drug therapy Patients with atrial fibrillation and evidence of a localized site of origin when the tachycardia is drug resistant or the patient is drug intolerant or does not desire long-term drug therapy	Patients with atrial arrhythmia responsive to drug therapy that is well tolerated and preferred by the patient over ablation Patients with multifocal atrial tachycardia
Ablation of atrial tachycardia, flutter, and fibrillation: accessory pathways	Patients with symptomatic AV reentrant tachycardia that is drug resistant or the patient is drug intolerant or does not desire long-term drug therapy Patients with atrial fibrillation (or other atria tachyarrhythmia) and a rapid ventricular response through the accessory pathway when the tachycardia is drug resistant or the patient is drug intolerant or does not desire long-term drug therapy	Patients with AV reentrant tachycardia or atrial fibrillation with rapid ventricular rates identified during electrophysiologic study for another arrhythmia Asymptomatic patients with ventricular preexcitation whose livelihood or profession, important activities, insurability, or mental well-being or the public safety would be affected by spontaneous tachyarrhythmias or the presence of the ECG abnormality Patients with atrial fibrillation and a controlled ventricular response through the accessory pathway Patients with a family history of sudden cardiac death	Patients who have accessory pathway-related arrhythmias responsive to drug therapy that is well tolerated and preferred by the patient over ablation
Ablation of VT	Patients with symptomatic sustained monomorphic VT when the tachycardia is drug resistant or the patient is drug intolerant or does not desire long-term drug therapy Patients with bundle branch reentrant VT Patients with sustained monomorphic VT and an ICD who are receiving multiple shocks not manageable by reprogramming or concomitant drug therapy	Nonsustained VT that is symptomatic when the tachycardia is drug resistant or the patient is drug intolerant or does not desire long-term drug therapy	Patients with VT responsive to drug, ICD, or surgical therapy that is well tolerated and preferred by the patient over ablation Asymptomatic and clinically benign nonsustained VT

AVNRT = AV nodal reentrant tachycardia; MI = myocardial infarction; VT = ventricular tachycardia.

ablation is often useful as an adjunct to ICD implantation to limit episodes of ventricular tachycardia requiring ICD treatment.¹⁶ Left ventricular dysfunction develops in some patients from frequent premature ventricular complexes, with reversal occurring after ablation of the premature ventricular complex.

Clinical Competence

The ACC/AHA statement on clinical competence⁸ describes three levels of training: level 1 for every cardiology trainee, level 2 for those wishing to acquire advanced training in the management of arrhythmia, and level 3 for those intending to specialize in invasive diagnostic and therapeutic cardiac electrophysiology. The level 3 guidelines recommend a minimum of 1 year of specialized training in electrophysiologic studies, during which the physician should be the primary operator and analyze 100 to 150 initial diagnostic studies, at least 50 of which should involve patients with supraventricular arrhythmias. Because antiarrhythmic devices constitute a major part of current electrophysiology practice, the guidelines suggest that a trainee should be the primary operator during at least 25 electrophysiologic evaluations of implantable antiarrhythmic devices. For maintenance of competence, a minimum of 100 diagnostic electrophysiologic studies per year is recommended. The statement also recommends that specialists in electrophysiology attend at least 30 hours of formal continuing medical education every 2 years to remain abreast of changes in knowledge and technology.

For physicians who perform catheter ablation, the NASPE Ad Hoc Committee on Catheter Ablation (now the Heart Rhythm Society) has recommended that training should include at least 75 catheter ablations, at least 10 of which are accessory pathway ablations and 30 to 50 are mentored ablations.⁸ The ACC/AHA statement recommends that physicians who perform ablations carry out at least 20 to 50 ablations per year.

Individuals receiving training in pacemaker implantation must participate as the primary operator (under direct supervision) in at least 50 primary implantations of transvenous pacemakers and 20 pacemaker system revisions or replacements. At least half of the implantations should involve dual-chamber pacemakers. The trainee must also participate in the follow-up of at least 100 pacemaker patient visits and acquire proficiency in advanced pacemaker electrocardiography, interrogation, and programming of complex pacemakers.⁸

References

- Knoebel SB, Crawford MH, Dunn MI, et al: Guidelines for ambulatory electrocardiography. A report of the American College of Cardiology/American Heart Association Task Force on Assessment of Diagnostic and Therapeutic Cardiovascular Procedures (Subcommittee on Ambulatory Electrocardiography). *Circulation* 79:206, 1989.
- Crawford MH, Bernstein SJ, Deedwania PC, et al: ACC/AHA guidelines for ambulatory electrocardiography. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Revise the Guidelines for Ambulatory Electrocardiography). Developed in collaboration with the North American Society for Pacing and Electrophysiology. *J Am Coll Cardiol* 34:912, 1999.
- Kadish AH, Buxton AE, Kennedy HL, et al: ACC/AHA clinical competence statement on electrocardiography and ambulatory electrocardiography: A report of the ACC/AHA/ACP-ASIM task force on clinical competence (ACC/AHA Committee to Develop a Clinical Competence Statement on Electrocardiography and Ambulatory Electrocardiography) endorsed by the

- International Society for Holter and noninvasive electrocardiology. *Circulation* 104:3169, 2001.
4. Akhtar M, Fisher JD, Gillette PC, et al: NASPE Ad Hoc Committee on Guidelines for Cardiac Electrophysiological Studies. North American Society of Pacing and Electrophysiology. *Pacing Clin Electrophysiol* 8:611, 1985.
 5. Zipes DP, DiMarco JP, Gillette PC, et al: Guidelines for clinical intracardiac electrophysiological and catheter ablation procedures. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Clinical Intracardiac Electrophysiologic and Catheter Ablation Procedures), developed in collaboration with the North American Society of Pacing and Electrophysiology. *J Am Coll Cardiol* 26:555, 1995.
 6. Tracy CM, Akhtar M, DiMarco JP, et al: American College of Cardiology/American Heart Association clinical competence statement on invasive electrophysiology studies, catheter ablation, and cardioversion. A report of the American College of Cardiology/American Heart Association/American College of Physicians-American Society of Internal Medicine Task Force on Clinical Competence. *J Am Coll Cardiol* 36:1725, 2000.
 7. Naccarelli GV, Conti JB, DiMarco JP, Tracy CM: Task Force 6: Training in specialized electrophysiology, cardiac pacing, and arrhythmia management: Endorsed by the Heart Rhythm Society. *J Am Coll Cardiol* 47:904, 2006.
 8. Naccarelli GV, Conti JB, DiMarco JP, Tracy CM: Task force 6: Training in specialized electrophysiology, cardiac pacing, and arrhythmia management endorsed by the Heart Rhythm Society. *J Am Coll Cardiol* 51:374, 2008.
 9. Epstein AE, Miles WM, Benditt DG, et al: Personal and public safety issues related to arrhythmias that may affect consciousness: Implications for regulation and physician recommendations. A medical/scientific statement from the American Heart Association and the North American Society of Pacing and Electrophysiology. *Circulation* 94:1147, 1996.
 10. Epstein AE, Baessler CA, Curtis AB, et al: Addendum to "Personal and Public Safety Issues Related to Arrhythmias That May Affect Consciousness: Implications for Regulation and Physician Recommendations. A medical/scientific statement from the American Heart Association and the North American Society of Pacing and Electrophysiology." Public safety issues in patients with implantable defibrillators. A scientific statement from the American Heart Association and the Heart Rhythm Society. *Heart Rhythm* 4:386, 2007.
 11. Gregoratos G, Abrams J, Epstein AE, et al: ACC/AHA/NASPE 2002 guideline update for implantation of cardiac pacemakers and antiarrhythmia devices: Summary article: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (ACC/AHA/NASPE Committee to Update the 1998 Pacemaker Guidelines). *Circulation* 106:2145, 2002.
 12. Epstein AE, DiMarco JP, Ellenbogen KA, et al: 2012 ACCF/AHA/HRS focused update incorporated into the ACCF/AHA/HRS 2008 guidelines for device-based therapy of cardiac rhythm abnormalities: A report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines and the Heart Rhythm Society. *Circulation* 127:e283, 2013.
 13. Strickberger SA, Benson DW, Biaggioni I, et al: AHA/ACCF scientific statement on the evaluation of syncope: From the American Heart Association Councils on Clinical Cardiology, Cardiovascular Nursing, Cardiovascular Disease in the Young, and Stroke, and the Quality of Care and Outcomes Research Interdisciplinary Working Group; and the American College of Cardiology Foundation: In collaboration with the Heart Rhythm Society: Endorsed by the American Autonomic Society. *Circulation* 113:316, 2006.
 14. Tracy CM, Akhtar M, DiMarco JP, et al: American College of Cardiology/American Heart Association 2006 update of the clinical competence statement on invasive electrophysiology studies, catheter ablation, and cardioversion: A report of the American College of Cardiology/American Heart Association/American College of Physicians Task Force on Clinical Competence and Training developed in collaboration with the Heart Rhythm Society. *J Am Coll Cardiol* 48:1503, 2006.
 15. Calkins H, Kuck KH, Cappato R, et al: 2012 HRS/EHRA/ECAS expert consensus statement on catheter and surgical ablation of atrial fibrillation: Recommendations for patient selection, procedural techniques, patient management and follow-up, definitions, endpoints, and research trial design: A report of the Heart Rhythm Society (HRS) Task Force on Catheter and Surgical Ablation of Atrial Fibrillation. Developed in partnership with the European Heart Rhythm Association (EHRA), a registered branch of the European Society of Cardiology (ESC) and the European Cardiac Arrhythmia Society (ECAS); and in collaboration with the American College of Cardiology (ACC), American Heart Association (AHA), the Asia Pacific Heart Rhythm Society (APHRS), and the Society of Thoracic Surgeons (STS). Endorsed by the governing bodies of the American College of Cardiology Foundation, the American Heart Association, the European Cardiac Arrhythmia Society, the European Heart Rhythm Association, the Society of Thoracic Surgeons, the Asia Pacific Heart Rhythm Society, and the Heart Rhythm Society. *Heart Rhythm* 9:632 e621, 2012.
 16. Aliot EM, Stevenson WG, Almendral-Garrote JM, et al: EHRA/HRS expert consensus on catheter ablation of ventricular arrhythmias: Developed in a partnership with the European Heart Rhythm Association (EHRA), a registered branch of the European Society of Cardiology (ESC), and the Heart Rhythm Society (HRS); in collaboration with the American College of Cardiology (ACC) and the American Heart Association (AHA). *Heart Rhythm* 6:886, 2009.