SUMMARY Patients with recurrent, unheralded syncope are often suspected of having intermittent cardiac bradycardia or tachyarrhythmias. However, syncopal episodes may be infrequent and investigations may yield few or non-specific etiological clues. Therapy may be entirely empirical (pacing or antiarrhythmic drugs) or guided by an abnormality detected that suggests a particular diagnosis. Intracardiac electrophysiological studies may detect conduction abnormalities or provoke arrhythmias but the relationship between these findings and clinical symptoms may be difficult to establish. The recording of the ECG during syncope by repeated ambulatory monitoring or other methods remains the only unequivocal diagnostic technique to establish an arrhythmic etiology.

PATIENTS with frequently recurring episodes of syncope or near syncope rarely pose diagnostic difficulties as ample opportunity exists for the physician to witness or document physiological functions during a spell. A greater challenge is posed by the patient with infrequent episodes who provides few clues after a careful history, physical examination and laboratory assessment. Although a "classical" description of the spell by a literate patient or witness can be virtually diagnostic, realities dictate that many patients will have unwitnessed spells or be unable to provide a reliable account of the surrounding events. The problem is compounded by the fact that syncope and presyncope of diverse etiologies may present in very similar ways with overlap of features considered characteristic of one or more mechanisms. For example, tachycardia is not an infrequent prodrome prior to vasodepressor syncope and seizures may occur after a period of cardiac asystole.

Clinical Assessment

The fundamental cardiac assessment of unexplained syncope should include a careful history and physical examination and laboratory testing to determine the nature of any heart disease. The latter would include echocardiography, exercise testing and ambulatory monitoring. Ideally, ambulatory monitoring should be repeated as often as possible until a typical spell occurs at the time of monitoring. If this initial assessment does not reveal a cause for the syncopal spell, several courses may be followed.

Follow-up and periodic ambulatory monitoring without any intervention is reasonable, especially if only one or two syncopal spells have occurred. The frequency of spells may increase allowing for an opportunity to monitor a spell or a primary disease process may evolve and make the etiology more apparent. Conversely, many patients have only one or a cluster of spells with no recurrence during follow-up and any extrapolation of these findings to an etiology for syncope is more tenuous. The problem is frequently compounded by the occurrence of several abnormalities in the same patient, each of which suggests a different etiology. The patient whose ambulatory ECG record is shown in figure 1 is a case in point. This patient was noted to have frequent ventricular extrasystoles, atrial arrhythmias, sinus bradycardia and sinus pauses are not uncommon in normal individuals and any extrapolation of these findings to an etiology for syncope is more tenuous. The problem is frequently compounded by the occurrence of several abnormalities in the same patient, each of which suggests a different etiology. The patient whose ambulatory ECG record is shown in figure 1 is a case in point. This patient was noted to have frequent ventricular extrasystoles, and short runs of supraventricular tachycardia, both suggesting possible etiologies of syncope, until the symptomatic episode of complete heart block illustrated in the figure was recorded. The finding of a prolonged QT interval in a patient with a family history of "seizure" or sudden cardiac death suggests a diagnosis of atypical ventricular tachycardia or "torsades des pointes" as an etiology even if the spells cannot be documented. An acquired form of the QT syndrome can occur in certain patients as an idiosyncrasy to quinidine-like drugs, phenothiazines, tricyclic antidepressants and other agents. Such patients will frequently present with syncope or seizures and may not experience a sensation of rapid heart beating (fig. 2).

Patients with fascicular blocks on the electrocardiogram, especially patients with bifascicular block, obviously have manifest abnormalities of cardiac conduction and have a greater probability of having or developing complete heart block than the general pop-
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FIGURE 1. Ambulatory ECG tracings from a patient with recurrent syncope. The strips are not continuous. The upper strips show ventricular extrasystoles, a short episode of supraventricular tachycardia and an atrial premature systole. All suggest potential causes of syncope. No symptoms were recorded in the diary until the episode of heart block was observed (lower strip) and the true cause of syncope was established.

However, when documentation of the spell is obtained in such patients, a whole spectrum of etiologies other than heart block can be found, making it evident that even a diagnosis of episodic heart block based on a finding of bifascicular block on the ECG is tenuous.

Carotid sinus hypersensitivity or elicitation of prolonged (greater than 3 seconds) asystole and/or hypotension with carotid sinus massage is often presumed to be the problem when found in patients with unexplained syncope.16, 17 Permanent pacing in patients with undocumented syncope and carotid sinus hypersensitivity would appear to be generally effective in terminating symptoms.17 However, carotid sinus hypersensitivity may be found in asymptomatic patients, especially elderly patients and those with organic heart disease, and this finding alone must still be accepted with some reservation as a cause for unexplained syncope.

Role of Cardiac Electrophysiological Studies

In recent years, the techniques of intracardiac recording and programmed electrical cardiac stimulation have been applied to patients with possible arrhythmic causes of syncope.1, 7, 9, 18-22 Intracardiac recording can identify abnormalities of atrioventricular nodal and His-Purkinje conduction (AH and HV interval). Programmed electrical stimulation can provide various tests of sinus node function (sinus node recovery time, sino-atrial conduction time) and assess conduction and refractory properties of cardiac muscle and the conduction system. In addition, programmed electrical stimulation can frequently reproduce arrhythmias identical to those experienced spontaneously by many patients with paroxysmal tachycardia.23 Although one would generally expect a history of rapid palpitations in patients with paroxysmal tachycardia, some patients may experience syncope or presyncope during tachycardia and not be aware of a sensation of rapid heart beating.1 The tracing of such a patient is shown in figure 3.

Cardiac electrophysiological studies in patients with syncope and a spectrum of cardiac disease have yielded a high frequency of abnormalities and treatment directed at the abnormalities resulted in elimination of spells in many patients.18-21 However, these studies included many patients with manifest abnormalities of conduction on the electrocardiogram and patients with

FIGURE 2. ECG monitor strips from a patient admitted with syncopal spells. The QT interval of sinus beats is prolonged and late-coupled ventricular extrasystoles initiate short episodes of atypical ventricular tachycardia. Longer episodes were associated with symptoms of presyncope. This patient had a drug-induced (quinidine) QT abnormality.
ventricular ectopic activity. It is not surprising that the diagnostic yield was high in such a patient population. On the other hand, when electrophysiologic testing was evaluated in selected syncopeal patients without any evidence of heart disease or conduction abnormalities, the diagnostic yield was considerably less. Although there is little doubt that cardiac electrophysiology testing can detect conduction abnormalities and elicit arrhythmias, these abnormalities must still be shown to cause the patient’s clinical symptoms and a convincing reproduction of the patient’s symptoms is only infrequently demonstrated in the laboratory. The fundamental limitations of electrophysiology testing in syncope assessment are self-evident: 1) Multiple abnormalities with a potential for causing syncope may be present. 2) Conduction abnormalities may be intermittent and not apparent at the time of testing and many forms of tachycardia cannot be reproduced by programmed stimulation. 3) Asymptomatic patients may have abnormal conduction intervals or sinus node function test results may be found to have symptoms from an unrelated cause when spells are finally monitored. 4) Treatment directed at the discovered abnormality is not uniformly successful. In fact, spontaneous remission and variable periodicity of symptoms make the evaluation of treatment directed at discovered abnormalities difficult. In short, we feel that electrophysiological testing should be reserved for patients with continued syncope after repeated attempts at documentation have failed. Even then, such testing may only uncover yet other abnormalities with no proven relationship to the spontaneous symptoms.

Conclusion

The only direct and unequivocal method for assessing a possible arrhythmic disturbance is the recording of an electrocardiogram during the event. Ambulatory monitoring is a valuable tool for this purpose and its only essential limitation is the expense and inconvenience of repeated ambulatory recordings in patients with infrequent symptoms. Since the interpretation of a 24 hour ECG record is a major component of cost and time, it has been suggested that patients could have repeated examinations until symptoms occur and only those records that occur during symptoms be read. In patients with presyncope or some warning prior to syncope, a patient-activated monitoring device may be useful. Ultimately, the development of monitoring devices that can be used conveniently over long time periods will simplify the assessment of patients with dizzy spells and syncope.

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