Evaluation of the patient with syncope

SYNCOPE is a common symptom whose cause is often elusive. While the clinical history and physical examination are important,[1,2] their findings are of limited value in the routine evaluation and care of the vast majority of patients with syncope. Except when syncopal episodes are related to obvious severe obstructive valvular lesions, the usual clinical techniques lack diagnostic precision. The clinician requires a test that reliably provokes the syncopal episode, which allows testing of various therapeutic modalities under control circumstances.

ARRHYTHMIC CAUSES OF SYNCOPE

The usual modes of establishing whether bradyarrhythmias or tachyarrhythmias are the cause of syncope have serious limitations. Since syncopal events are usually isolated and punctuated by long asymptomatic intervals, ordinary 24-hour continuous ambulatory recordings often fail to provide the required diagnostic accuracy. Similarly, trans-telephonic monitoring systems are of little value because the patient is unable to transmit at the critical time. A number of studies have emphasized the value of invasive electrophysiologic testing for patients with syncope. A positive test is most often found in patients with organic cardiac disease, particularly those with prior myocardial infarction. In contrast, the yield of these studies is quite low when applied to patients with no structural heart disease.[3–5]

■ *See Maloney et al (pp 542–548)*

Invasive studies are used to obtain evidence of sinus node or arterioventricular conduction disturbances by atrial pacing and/or drug challenge. Standard pacing protocols are also used to test whether the patient has inducible tachycardia. The latter group may experience syncope owing to inordinately rapid heart rates. Provocation of tachycardia allows for sequential drug testing and/or use of the automatic internal defibrillator when appropriate.

HEAD-UP TILT

The cardiology group at The Cleveland Clinic Foundation has provided important pioneering observations confirming the value of head-up tilting in the evaluation of patients with syncope.[6–8] This noninvasive test allows the clinician to establish a rational approach to the management of patients with hypovolemia and autonomic insufficiency, as well as neurally mediated syncope. On the basis of hemodynamic responses during tilting, the clinician can select specific treatment modalities and check their efficacy.

Maloney et al[9] have documented the serious potential consequences of vasovagal episodes by documenting 73 seconds of cardiac asystole in a patient with syncope. This response was provoked by the head-up tilt test. The authors further document evidence of pan-cerebral hypoperfusion by the classic EEG changes of diffuse brainwave slowing followed by attenuation. Of interest is that myoclonic jerks occurred during the syncopal episode. Patients with arrhythmic causes of syncope often show tonic motor activity, with flexion of extremities or irregular clonic contractions of the extremities.

The motor activity accompanying pan-cerebral hypoperfusion may be confused with true seizure activity. In studies from our laboratory,[10] we found several clinical clues of value in differentiating true seizures from motor activity accompanying pan-cerebral hypoperfusion. First, the clonic activity observed in patients with arrhythmia-provoked syncope is always coarse and irregular. Second, full loss of consciousness is transient and related to the duration of the arrhythmia. This clinical presentation is different from true seizure activity, in which clonic movements are regular and the seizure is

followed by a prolonged postictal state. A clear description of the syncopal event by observers often leads to more appropriate laboratory testing.

The report by Maloney et al demonstrates the clinical value of head-up tilt testing. Broader use of this technique holds promise of both a better understanding of the pathophysiology of syncope and better care for these patients.

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