Syncope Evaluation at a Crossroad
For Which Patients?

Brian Olshansky, MD

Syncope is one of the most common, alarming, and challenging symptoms with which cardiologists, and most other physicians, grapple. It can cause injury and disability, affect lifestyle and quality-of-life, and be an expensive management nightmare. Causes range from isolated, benign, situational, and “dysautonomic” events to life-threatening ventricular arrhythmias.

A thoughtful history and complete physical examination, performed by an astute clinician, will provide diagnostic clues to guide management. Unfortunately, the approach often undertaken includes low-yield testing (EEG, CT scan, carotid Dopplers, Holter monitor, and cardiac enzymes), yet even “proper” testing (electrophysiology testing and tilt table testing) can be fruitless. In nearly half of all patients, no diagnosis is secured. Although an implantable loop recorder (ILR) may be useful when all else fails, no randomized trial has provided evidence that it is the best initial approach when the history does not provide a diagnosis... until now.

Krahn et al report the first prospective, randomized trial of the ILR as the initial approach when the cause for syncope could not be gleaned from a circumspect evaluation. They address an important clinical problem and provide new insight into methods to assess syncope. The best way to identify the cause is to monitor the episode. The ILR can do just that: it can provide a diagnosis efficiently, apparently without injury, and blood sugar changes. In the future, perhaps, this will be possible.

The population selected here was at low risk for death, but it is clear that some syncope patients have imminent demise from cardiovascular death. How these patients were excluded is unknown. Consider the following example. A patient with ischemic heart disease, mildly impaired ventricular function, and a bundle branch block who has had coronary revascularization is admitted for recurrent syncope and collapse. Should this patient get an ILR as a first-line approach? Is it possible that the ILR will record the cause of the next event for posterity: ventricular fibrillation. What about the patient with dilated cardiomyopathy and abrupt, unexplained syncope? The benefit of the ILR in this population remains unknown.

So, if patients with extremely benign causes for syncope should not be considered, yet those with serious, but undiagnosed, causes should also not be included, then who should be considered for an ILR as a first-line diagnostic approach?

The evaluation before ILR was not standardized and, therefore, the diagnosis of heart disease may have been missed. Criteria for echocardiography, cardiac catheterization, and treadmill testing are vague. A need to perform electrophysiology testing in patients with preserved left ventricular function and those without ischemic heart disease is not supported.

The ILR cannot distinguish bradycardia by mechanism (neurocardiogenic from intrinsic disease). Not all bradycardia in a syncope patient requires a pacing device. Treating bradycardia with a pacemaker in a patient with neurocardiogenic syncope may not necessarily prevent recurrent syncope. One patient in this trial treated with a pacemaker still had recurrent syncope.

The extraordinarily low recurrence rate of syncope in a substantial segment of the patients with a negative diagnostic evaluation suggests that (1) these patents may not require an aggressive assessment (perhaps there is a way to exclude such patients); (2) those with a positive evaluation had a treatment that exacerbated or did not treat their syncope; (3) neither approach was capable of arriving at all diagnoses in the time allotted; and/or (4) the diagnosis of syncope is incorrect.

A role for the ILR in syncope evaluation exists, but for which patients? One crossroad in syncope evaluation has arrived. Others roads must be crossed. Carefully controlled, clinical trials clearly enunciating the population will define the exact role of ILR. A device that can measure heart rate and hemodynamic response would be more accurate to define the cause and mechanism for syncope. Even better would be a device that measures these parameters along with an electroencephalogram, cerebral blood flow, and hormonal and blood sugar changes. In the future, perhaps, this will be possible.

The opinions expressed in this editorial are not necessarily those of the editor or of the American Heart Association.

From the Division of Cardiology, University of Iowa Hospital, Iowa City.

Correspondence to Brian Olshansky, MD, Division of Cardiology, University of Iowa Hospital, 200 Hawkins Drive, 4426 B, JCP, Iowa City, IA 52242-1081. E-mail brian-olshansky@uiowa.edu

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