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Randomized Assessment of Syncope Trial: Conventional Diagnostic Testing Versus a Prolonged Monitoring Strategy

To the Editor:

The effort of Krahn et al1 to provide a direct, albeit invasive, monitoring strategy to ascertain the nature of spontaneous syncope follows Sir Thomas Lewis’s worthy tradition of direct observation.2 But terminology complicates their research design. “Vasovagal Syncope” was the name that Lewis cribbed from Gowers in his original description. In 1991, Sra et al renamed this condition Neurocardiogenic Syncope (reviewed in references 3 and 4). Because Krahn et al excluded patients with “presentation typical of neurally mediated syncope,” exactly what case descriptions were left for “randomized assessment” is unknown.

Although syncope almost always occurs when the patient is standing, human primates, especially the elderly, assume this position only when awake during a small fraction of the day. The logical inference is that deficient vasopressor mechanism must contribute to this postural liability. Conversely a primary cardiac etiology is likely for sedentary or recumbent symptoms. Having observed that atropine blocked bradycardia, but not syncope, Lewis concluded, “the cause of syncope is mainly vasomotor and not vagal.”2 Sharpey-Schafer5 noted that heart failure patients (increased blood volume) do not faint. Krahn et al fail to describe the clinical circumstances of their monitored syncopeal episodes.

Transient bilateral myoclonic limb movements as a consequence of transient brain ischemia are commonplace. But an acquired temporal lobe seizure focus as the cause of asystole is exceptionally rare and unrelated to posture. The features of Krahn et al’s “neurological consultation” that led to presumably successful anticonvulsant medication for 2 patients are likewise unexplained.

Clinical label also relates to the validity of the “diagnostic” tilt table routine.3, 4 Any proposed diagnostic laboratory test for any clinical condition can be validated only by carefully controlled blind comparative studies of unaffected and “gold standard” patients whose diagnosis is certainly established by independent criteria. The tilt table literature fails to meet this reference standard. Furthermore, the tilting routine, especially with the addition of isoproterenol, fails to simulate the real life patterns of spontaneous syncope. The procedure also fails to control for variations of lower extremity muscular contraction that pump venous blood into the vena cava as the subject assumes a standing posture.

Pending better resolution of these syncopeal controversies, all parties may concur with editorialist Olshansky’s assertion6 of the primary importance of “a thoughtful history and complete physical examination, performed by an astute clinician.”

Response

We would like to thank Dr Landau for pointing out that the investigation of syncope is often in need of a gold standard. There is frequent difficulty with establishing a diagnosis, making it all the more important to establish the physiological state of the patient during spontaneous symptoms. Clearly patients in whom a clinical diagnosis of vasovagal syncope can be made (based on a thoughtful history and complete physical examination) are not candidates for an invasive clinical trial of unexplained syncope.

As indicated in the RAST study, this presentation included upright posture with a prodrome, including warmth and diaphoresis, with post-episode fatigue.1 The patients who had seizures had motion artifact on their recorded ECG in keeping with tonic-clonic movements. This has been referenced and published in the article. Finally, Dr Landau’s comments regarding the utility of tilt testing are in many ways borne out by the results of this study. Clearly, the absence of a gold standard makes the utility of tilt testing at best modest. Without question, we agree with both Dr Olshansky and Landau that a thoughtful history and physical examination remain the cornerstone of diagnosing patients with syncope.


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