Letters to the Editor must not exceed 400 words in length and must be limited to three authors and five references. They should not have tables or figures and should relate solely to an article published in Circulation within the preceding 12 weeks. Authors of letters selected for publication will receive prepublication proofs, and authors of the article cited in the letter will be invited to reply. Replies must be signed by all authors listed in the original publication. Please submit three typewritten, double-spaced copies of the letter to Herbert L. Fred, MD, % the Circulation Editorial Office. Letters will not be returned.

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

To the Editor:

The effort of Krahn et al 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. 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 a 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.” Sharpey-Schafer noted that heart failure patients (increased blood volume) do not faint. Krahn et al fail to describe the clinical circumstances of their monitored syncopal 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 presump-
tively successful anticonvulsant medication for 2 patients are likewise unexplained.

Clinical label also relates to the validity of the “diagnostic” tilt table routine. 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 syncopal controversies, all parties may concur with editorialist Olshansky’s assertion of the primary importance of “a thoughtful history and complete physical examination, performed by an astute clinician.”

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