symptoms with tachycardia and hypotension. The patient described by Kosinski et al.[25] was taking fluoxetine, methylbarbital, and clonazepam for depression and seizure disorder. Exercise provoked tachycardia and then hypotension.

This mixed bag of exercise-associated symptoms may be related to decreased basal blood volume, cardiac vagal sensitivity, or both. Such patients require critical study, because they do not suffer from classic vasovagal syncope. Healthy subjects can develop postexercise asystole[39] and hypotension.[40] In regard to the risk of death with exercise, a recent report of autopsy findings in 134 cases of sudden death in young competitive athletes discovered only three who showed "absence of structural heart disease on standard [not carefully focused on heart pathology] autopsy examination."[41]

Kosinski and Grubb's last paragraph is a reprise of interspecialty conflict, which we shall not join. They cite an abstract[28] that described components of what is defined as a "neurologic workup" of patients with syncope. We agree, no matter who orders them, that such studies as carotid ultrasound, EEG, head CT, brain MRI, transcranial Doppler, brainstem potentials, and carotid arch angiogram are practically never indicated. The tilt-table venture is the most frequent extravagant procedure. Our criticism concerns the quasireligious diagnostic dependence on magical gadgetry. We concur with a prominent cardiologist, M. C. Petch, whose concise review of this field was aptly entitled "Syncope: an accurate history tells all."[42]

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Correction

The article "Long-term follow-up after temporal lobe resection for lesions associated with chronic seizures" by Eliashiv et al. was inadvertently published twice in *Neurology* (Neurology 1997;48:621–626 and Neurology 1997;48:1383–1388.) We apologize for any inconvenience or confusion this may have caused.