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Cerebral syncope: New insights into an emerging entity

Only if one knows the causes of syncope will be able to recognize its onset and combat the cause.

Maimonides, 1135-1204 CE

Syncope, the sudden loss of consciousness and postural tone with spontaneous recovery, is a common clinical complaint for which patients are frequently referred for evaluation. Recurrent unexplained syncope can provoke great anxiety among patients, their families, and physicians because, as one observer succinctly observed, "Syncope and sudden death are the same thing, except in one you wake up."¹ For years it was postulated that many episodes of syncope occurred because of periods of decompensation in the autonomic nervous system that would lead to hypotension (with or without bradycardia) profound enough to lead to cerebral hypoperfusion and loss of consciousness, a phenomenon known as neurocardiogenic or vasova-

gal syncope. In the mid 1980s, head-up tilt table testing became available as a means of provoking autonomic nervous system decompensation in a laboratory setting.² This not only introduced a long-sought-after diagnostic modality but also provided a controlled setting in which detailed observations and measurements could be made during syncope itself. As a result, there has been a virtual explosion in our knowledge about these disorders. It has become increasingly apparent that classic neurocardiogenic syncope is but one aspect of a broad, heterogeneous group of disturbances in the autonomic nervous system, which can affect normal homeostatic mechanisms to such an extent that loss of consciousness ensues.³

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Almost at the same time as tilt table testing was introduced, transcranial Doppler ultrasonography became available, allowing for the first highly accurate noninvasive assessments of cerebral blood flow to be easily performed. In the early 1990s, several

groups of investigators began performing TCD assessments of cerebral blood flow during tilt-induced neurocardiogenic syncope.⁴⁻⁷ The existing theories of cerebral blood flow autoregulation would have predicted a sudden cerebral arteriolar vasodilation at the time of syncope, in order to help preserve cerebral perfusion. Instead, what was uniformly reported was a sudden sig-

TCD Transcranial Doppler ultrasonography

nificant increase in cerebral vascular resistance (signifying arteriolar vasoconstriction) as measured by TCD, which occurred concomitant with the loss of consciousness. These observations suggest that this apparently "paradoxical" increase in cerebral arteriolar vasoconstriction in the face of increasing systemic hypotension may significantly decrease cerebral perfusion and help produce cerebral hypoxia and loss of consciousness.⁴ Similar degrees of paradoxical vasoconstriction have also been noted during hypotension associated with polymorphic ventricular tachycardia induced during

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implantable defibrillator placement.⁸ Shortly thereafter, it was reported that cerebral blood flow changes alone, at normal systemic pressures, could produce severe dizziness and vertigo.⁹

In many research laboratories, TCD measurements, as well as electroencephalographic recordings, were routinely made during every head-up tilt test. Gomez et al¹⁰ reported sudden cerebral vasoconstriction alone (as measured by TCD) in the absence of systemic hypoperfusion or bradycardia, causing loss of consciousness during head-up tilt table testing. Njemanze⁶ reported identical observations in 10 patients during tilt-induced syncope. Daffertshafer et al¹¹ made similar observations of syncope caused by cerebral blood flow alterations alone in 4 patients, calling it "normotensive orthostatic syncope," and referred to the phenomenon in general as "cerebrovascular dysautoregulation syndrome." Fredman et al¹² and Obara et al¹³ reported cases of tilt-induced syncope with TCD-measured cerebral vasoconstriction in the absence of systemic hypotension. Grubb et al¹⁴ presented 5 patients with tilt-induced loss of consciousness, confirmed by electroencephalographic monitoring, associated with TCD-recorded vasoconstriction without systemic hypotension, and referred to the entity as "cerebral syncope."

In this issue of *The Journal*, Rodríguez-Núñez et al¹⁵ describe the novel approach of using noninvasive near-infrared cerebral spectrophotometry to document this same phenomenon in a child with recurrent syncope. This insightful observation, taken together with the aforementioned studies, suggests that in some patients, a disturbance in normal cerebrovascular autoregulation results in an inappropriate degree of cerebral arteriolar constriction. The resultant reduction in cerebral blood flow can be so profound as to produce cerebral hypoxia and loss of consciousness, even in the absence of systemic hypotension. This mechanism may be similar to those that sometimes produce

syncope in patients who have basilar artery migraines. It appears that there may be a significant degree of overlap between these 2 disorders.^{16,17} Indeed, disorders of cerebral autoregulation may be as diverse and varied as are autonomic disorders themselves. Although the study by Rodríguez-Núñez et al¹⁵ helps to confirm the existence of cerebrovascular syncope in younger children, there is perhaps an equally important message. Patients who have experienced normotensive syncope in the past (whether tilt-induced or spontaneous) were diagnosed as having a psychogenic condition.¹⁸ Although cerebral syncope would seem an uncommon disorder, something not looked for is seldom found. These data suggest that either TCD, electroencephalography, or near-infrared cerebral spectrophotometry monitoring may be needed during tilt-induced normotensive syncope to ensure that a central physiologic cause is not responsible before a person's condition can be labeled psychogenic. Further, more in-depth studies will be necessary to better understand the phenomenon of "cerebral syncope."

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