A New Guideline for Diagnosis and Treatment of Syncope in Children and Adolescents That Stimulates Further Thought and Discussion

Julian M. Stewart

New York Medical College

Follow this and additional works at: https://touroscholar.touro.edu/nymc_fac_pubs

Part of the Medicine and Health Sciences Commons


This Response or Comment is brought to you for free and open access by the Faculty at Touro Scholar. It has been accepted for inclusion in NYMC Faculty Publications by an authorized administrator of Touro Scholar. For more information, please contact touro.scholar@touro.edu.
A new guideline for diagnosis and treatment of syncope in children and adolescents that stimulates further thought and discussion

Julian M. Stewart
Physiology and Medicine, Center for Hypotension, The New York Medical College, Valhalla, NY 10595, USA

A new guideline for diagnosis and treatment of syncope in children and adolescents was published by Chinese Pediatric Cardiology Society (CPCS) [1]. This guideline admirably outlines a differential diagnosis of syncope in the Chinese population, placing appropriate emphasis on vasovagal syncope (VVS, simple faint), the most common cause. The authors appropriately discuss other more ominous causes of syncope such as cardiogenic syncope and neurogenic orthostatic hypotension. In particular, cardiogenic syncope can have lethal consequences if not promptly treated. The paper provides a useful algorithm for diagnostic evaluation of syncope patients placing primary emphasis on history, physical exam, and ECG, and further discussing testing procedures such as standing tests used in screening patients, and upright tilt table testing which remains the reference standard for orthostatic testing in younger patients. Indeed, most vasovagal and other forms of reflex or situational syncopes can be best diagnosed on clinical history [2]. The authors further consider other forms of orthostatic intolerance such as orthostatic hypertension [3], postural tachycardia syndrome (POTS), and “initial orthostatic hypotension” experienced as transient lightheadedness upon rapid standing [4]. The authors regard POTS as a cause of vasovagal syncope. This supposition is controversial and deserves further comment.

POTS has been reported to be associated with vasovagal syncope to varying degree [5,6]. Postural Tachycardia Syndrome was a term devised by workers at Mayo clinic to describe patients with symptoms of chronic orthostatic intolerance synchronous with excessive upright tachycardia but who had no postural hypotension as characterizes neurogenic orthostatic hypotension and vasovagal syncope [7]. POTS is best considered “a heterogeneous and multifactorial disorder” [8] of diverse etiology rather than a disease or causal entity. POTS can be clinically distinguished from typical recurrent vasovagal syncope in that POTS is chronic with day-to-day symptoms while VVS is episodic; between episodes VVS patients are generally well.

Tilt table studies may add confusion to this etiological scheme. In brief, tilt table results need not correspond to real world syncope. More than 40% of recurrent VVS patients have

julian_stewart@nymc.edu.

Conflict of interest
The author declare that he has no conflict of interest.
heart rates exceeding POTS thresholds during tilt tests [9], while prolonged orthostatic testing of POTS patients beyond the original standard 10-minute tilt produces ‘false positive tests” including increasing numbers of false positive POTS [10] and a relatively high incidence of vasovagal syncope in healthy young volunteers (and POTS patients) with no real world history of syncope [11]. Specificity is further reduced by nitroglycerin [12]. However, recent reconsideration of such false positive VVS suggests that they represent a subpopulation with a hypotensive susceptibility [13]. Moreover, the vasovagal syncope response can be induced in almost everyone at different thresholds of orthostatic stress [14,15] and may be an evolutionary reflex response to hemorrhage [16].

In summary, the guideline provides a useful review of syncope and orthostatic intolerance in general while stimulating further thought and discussion.

**Acknowledgments**

This work was supported by RO1 HL 134674 and RO1 HL 112736 from the National Heart Lung and Blood Institute.

**Biography**

Julian Stewart graduated from Cornell University with a degree in physics, receiving M.D. and Ph.D. (cardiac physiology) from the University of Chicago, and internship and residency at New York University. He completed a Pediatric Cardiology fellowship at Cornell University-New York Hospital. He is currently Professor of Pediatrics, Physiology, and Medicine, and Associate Chairman of Pediatrics at New York Medical College. He directs the Center for Hypotension, funded by the National Heart, Lung and Blood Institute of the National Institutes of Health, and has studied the circulatory bases of orthostatic intolerance in the young for more than 18 years with a focus on mechanisms of vasovagal syncope and postural tachycardia syndrome.

**References**


