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Original Article

The Clinical and Electroencephalographic Spectrum of Tilt-Induced Syncope and "Near Syncope" in Youth

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ABSTRACT

BACKGROUND: The aim of the study was to characterize the clinical and electroencephalographic (EEG) patterns associated with tilt-induced reflex syncope and delayed orthostatic hypotension without syncope in youth. **METHODS:** We conducted a prospective observational study of 95 patients referred to a pediatric neurology clinic for head-upright tilt testing. Clinical signs, symptoms, video EEG, and continuous blood pressure and heart rate were monitored. **RESULTS:** Eighty patients had reflex syncope, and 15 had delayed-onset hypotension without syncope. The mean age was 15.3 (standard deviation ±2.3) years; 75 (78.9%) were female. All patients with hypotension only had corresponding signs and symptoms; 13 (86.7%) had corresponding EEG slowing. The duration of EEG slowing with hypotension far exceeded the presyncope interval from onset of slowing to loss of consciousness among patients with syncope ($P < 0.001$). Although prior near-syncope and presyncope episodes were reported commonly in both groups, patients with delayed hypotension without syncope were less likely to have experienced loss of consciousness during episodes of orthostatic intolerance ($P < 0.001$). Patients with syncope had either slow-flat-slow (n = 23) or slow-only (n = 57) EEG patterns. Compared to those with slow-only EEG patterns, patients with the slow-flat-slow pattern had greater rates of asystole ($P < 0.001$), myoclonic movements ($P < 0.001$), facial grimace ($P = 0.003$), vocalizations ($P = 0.002$), and arm flexion ($P < 0.001$) or extension ($P = 0.006$) during tilt-induced syncope. **CONCLUSIONS:** Among otherwise healthy youth, orthostatic signs and symptoms vary across the spectrum of tilt-induced reflex syncope and delayed hypotension without syncope. Delayed hypotension without syncope may represent the poorly defined phenomenon of "near syncope" in some patients.

Keywords: delayed orthostatic hypotension, orthostatic hypotension, pediatric, adolescent, electroencephalography, syncope, near syncope

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Introduction

Reflex (neurally mediated) syncope is a common cause of transient loss of consciousness among children and adults, with a lifetime cumulative incidence estimated as high as 35% to 40%.[1-3] Syncope ultimately results from insufficient cerebral blood flow, but the precise afferent nerve pathways and central nervous system mechanisms underlying the process remain largely unknown.[4] Electroencephalography (EEG) allows real-time assessment of dynamic cerebral changes during the syncope episode. Two EEG patterns have been described from predominantly adult studies of tilt-induced reflex syncope: a slow-flat-slow pattern and a slow-only pattern.[5-13] The slow-flat-slow pattern represents a greater degree of cerebral hypoperfusion than the slow-only pattern,[14] but why two patterns exist is not clear. Prospective studies of otherwise healthy youth with reflex syncope are lacking.

We sought to further characterize the clinical and EEG patterns associated with tilt-induced reflex syncope in

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youth. As part of the research protocol, we asked that patients remain in the tilted position until loss of consciousness occurred. Some patients developed substantial hypotension with corresponding signs and symptoms of imminent syncope, but they did not experience loss of consciousness. These patients were also included in this study. A better understanding of the clinical and physiologic changes that occur with the spectrum of tilt-induced hypotension and syncope may help to elucidate the elusive neural mechanism(s) underlying these common clinical events and to aid clinicians in distinguishing syncope from epileptic seizures and other forms of transient loss of consciousness.

Patients and Methods

Subjects

We conducted a prospective observational study of sequential patients referred to a pediatric neurology–based clinic for head-upright tilt (HUT) testing between June 2014 and October 2015. Reasons for referral included refractory syncope, transient loss of consciousness of unclear etiology, and persistent orthostatic symptoms (e.g., lightheadedness, blackout spells, "near syncope") with or without loss of consciousness. Study inclusion criteria were normal cardiac evaluation (minimally including 12-lead electrocardiogram and cardiac examination) and syncope or substantial orthostatic hypotension (OH) during HUT. The protocol allowed for all patient ages given their referral to a pediatric neurology–based clinic and their ability to tolerate HUT testing. Patients were excluded from study if they had normal HUT tests; if their procedures were aborted prematurely for any reason; if the captured event was consistent with a psychogenic form of collapse; or if the patient had a genetic, autoimmune, metabolic, or neuropathic cause of OH. Patients with typical features of presyncope who requested lowering before loss of consciousness or less than 20 seconds after onset of hypotension were also excluded. To help distinguish delayed hypotension without syncope (see the later discussion) from presyncope, we chose 20 seconds based on our prior experience that most patients with syncope will lose consciousness within 20 seconds of symptomatic hypotension.

Syncope was defined as a transient loss of consciousness associated with hypotension, with or without bradycardia. Bradycardia was defined as heart rate slowing $\leq$40 bpm for $\geq$10 seconds. Asystole was defined as an interval $\geq$3 seconds between QRS complexes. We defined delayed-onset hypotension without syncope as a symptomatic drop in systolic blood pressure (SBP, $\geq$20 mm Hg), persisting $\geq$20 seconds without loss of consciousness, and occurring beyond three minutes of HUT.

Protocol

All medicines that could affect orthostatic tolerance were discontinued $\geq$5 half-lives. Video EEG (Comet AS-40; GRASS systems, Warwick, RI) was synchronized with continuous heart rate and blood pressure (Portapres; Finapres Medical Systems, Amsterdam, The Netherlands) monitoring at baseline and during HUT testing. After 30 minutes of recumbency, patients were tilted upright (70°), up to 45 minutes. Patients with syncope were lowered with onset of loss of consciousness. Patients who developed hypotension without syncope were encouraged to remain upright until symptoms became intolerable. They underwent simple cognitive testing during the hypotensive period (e.g., What is the name of this hospital? Where were you born?). Four patients with syncope were given sublingual nitroglycerin (0.3 mg) as part of their clinical protocol to provoke syncope. Medicine provocation was not given to any patient with hypotension only.

Patients were asked to report all symptoms immediately upon symptom onset. Clinical signs were recorded in real time and confirmed by video review. On recovery, several individuals recalled prodromal symptoms that were not reported in real time, so precise timing of symptom onset could not be established. Amnesia of prodromal symptoms did not occur in any patient. EEGs were interpreted by a pediatric neurologist trained in neurophysiology (M.P.I.).

Standard protocol approvals

The study was approved by the Institutional Review Board at Nationwide Children's Hospital. Informed consent (parents and subjects $\geq$18 years of age) and assent (subjects 9 to 17 years) were obtained before all testing.

Statistical analysis

Descriptive statistics were calculated for clinical and HUT characteristics. Baseline and nadir blood pressure data were calculated using mean values from 20-second epochs for patients with hypotension only. Sustained movement artifacts from convulsions prevented accurate and consistent assessments of nadir blood pressures in syncope patients. One-way analysis of variance with Bonferroni post hoc testing was used to compare variables between the three groups. The chi-square test or

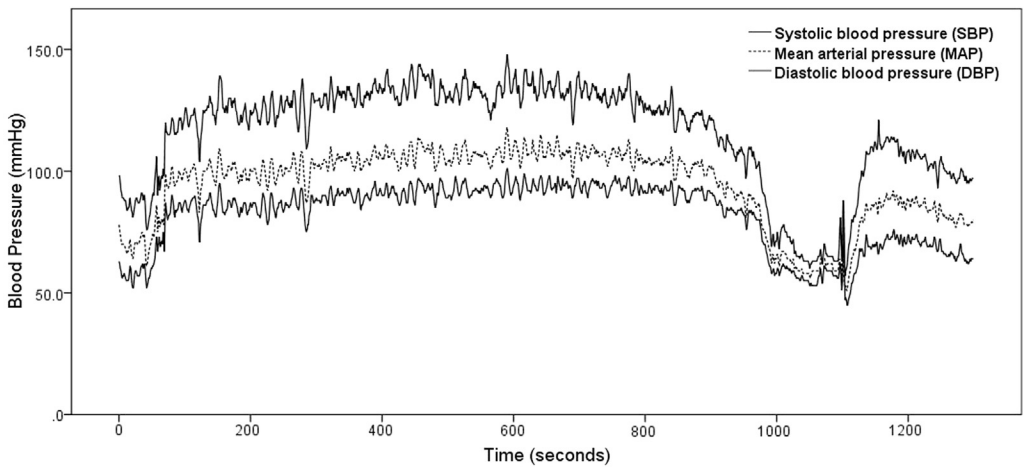

FIGURE 1.
Blood pressure changes with delayed orthostatic hypotension without syncope. An abrupt blood pressure drop (greater than 20 mm Hg) occurred at 992 seconds of head-upright tilt and persisted through table lowering at 1087 seconds. Table lowering takes an additional 12 seconds. Corresponding electroencephalographic changes were present for 51 seconds.

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