

CLINICAL AND LABORATORY OBSERVATIONS

Cerebral syncope in children

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We report a child with recurrent syncopal episodes who underwent head-up tilt testing according to a protocol that includes continuous and noninvasive measurement of brain oxygen saturation. We demonstrated significant cerebral hypoxemia during syncope without concomitant systemic hemodynamic disturbances. This response to head-up tilt test suggested the diagnosis of cerebral syncope. (J Pediatr 2000;136:542-4)

Neurocardiogenic syncope is frequent; however, the pathophysiology is unknown.¹ The development of head-up tilt testing for the evaluation of patients with syncope has improved evaluation and has allowed further studies of the pathophysiologic disturbances triggered during the test.²⁻⁷

The proposed mechanism for syncope is that the critical decrease in cerebral blood flow that leads to faint-

ing is secondary to a hemodynamic collapse mediated by a sudden change in autonomic nervous system activity.^{1,7} However, new evidence suggests that, at least in some patients, the main and primary event is a failure in cerebral vascular self-regulation.⁸⁻¹¹

See editorial, p. 431.

An entity named *cerebral syncope*, in which loss of consciousness occurs in the absence of systemic hemodynamic disturbances, has been recently reported.¹² This singular state has been studied in adult patients,¹² but not in children.

CASE REPORT

An 8-year-old boy was evaluated because of recurrent episodes of sudden loss of consciousness. Findings on general and cardiologic examinations were normal. A head-up tilt test was performed according to the previously de-

scribed protocol,¹¹ which includes the continuous and noninvasive monitoring of cerebral oxygen saturation by means of a near-infrared spectroscopy device.

Briefly, the patient was monitored by means of standard electrocardiography, continuous (beat-to-beat) noninvasive blood pressure measurements (Finapres; Ohmeda, Louisville, Colo), pulse-oximetry and nasal capnography (Ohmeda 4700 OxiCap, Ohmeda), and noninvasive near-infrared cerebral spectrophotometry (Invos 3100 Cerebral Oximeter; Somanetics Corp, Troy, Mich) with the sensor placed on the child's forehead. After the baseline period, during which the child had been supine for 10 minutes, he was positioned at 80° from horizontal on a tilt table with a weight-bearing footboard. The test continued for a maximum of 40 minutes; if no response (cardio-inhibitory, vasodepressor, or mixed) was observed, a threat of venipuncture and eventual procedure were performed. If no response was elicited in 5 minutes, the test result was deemed negative. If a response was present, the patient was repositioned in Trendelenburg's position until total resolution of symptoms occurred. The patient was then placed in the supine position.

For the patient in this case, after 10 minutes in the supine position, heart rate was 72 beats/min, blood pressure 111/60 mm Hg, peripheral oxygen sat-

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uration 98%, end-tidal carbon dioxide 40 mm Hg, and cerebral oxygen saturation 56%. The head-up tilt test was performed without clinical manifestations or significant changes in monitored variables during a period of 40 minutes. At that time and according to our usual protocol, "venipuncture threat" was performed. Thirty seconds later, the patient complained of dizziness, lightheadedness, and feeling cold; the patient turned pale and finally experienced impaired consciousness. Simultaneously, a sudden decrease in cerebral oxygen saturation was noted, as low as 40%; at that time, heart rate was 129 beats/min, blood pressure 100/64 mm Hg, peripheral oxygen saturation was 97%, and end-tidal carbon dioxide, 38 mm Hg. The patient was placed in Trendelenburg's position, resulting in rapid recovery of clinical status to baseline (within 1 minute) and showed a "rebound" increase in cerebral oxygen saturation up to 62%, maintained over 4 minutes before returning to baseline (Figure). At the end of the test, the patient was questioned about the symptoms, which he found to be similar to the previous events that had led to our consultation.

DISCUSSION

This patient showed clinical symptoms and alterations in vital signs consistent with the diagnosis of cerebral syncope, which has previously been described by Grubb et al,¹² who demonstrated the presence of cerebral hemodynamic disturbances in 5 adult patients with syncope by using transcranial Doppler ultrasonography; each patient demonstrated a paradoxical cerebral vasoconstriction during the course of syncope, without concomitant significant systemic hemodynamic changes. We have achieved the same conclusion by measuring, in a noninvasive manner, the cerebral oxygen saturation with a near-infrared spectroscopy device.^{13,14} The measurement of cere-

bral oxygen saturation reveals the equilibrium between delivery and consumption of oxygen by neurons in a given time (that means cerebral tissue oxygenation). A decrease in cerebral oxygen saturation indicates a relative decrease in blood flow and

hypoxia or a change in oxygen consumption or a combination of both, whereas an increase in cerebral oxygen saturation points to a relative hyperemia. Here we assume that desaturation relates to decreased flow. Under neuronal metabolic stability, those

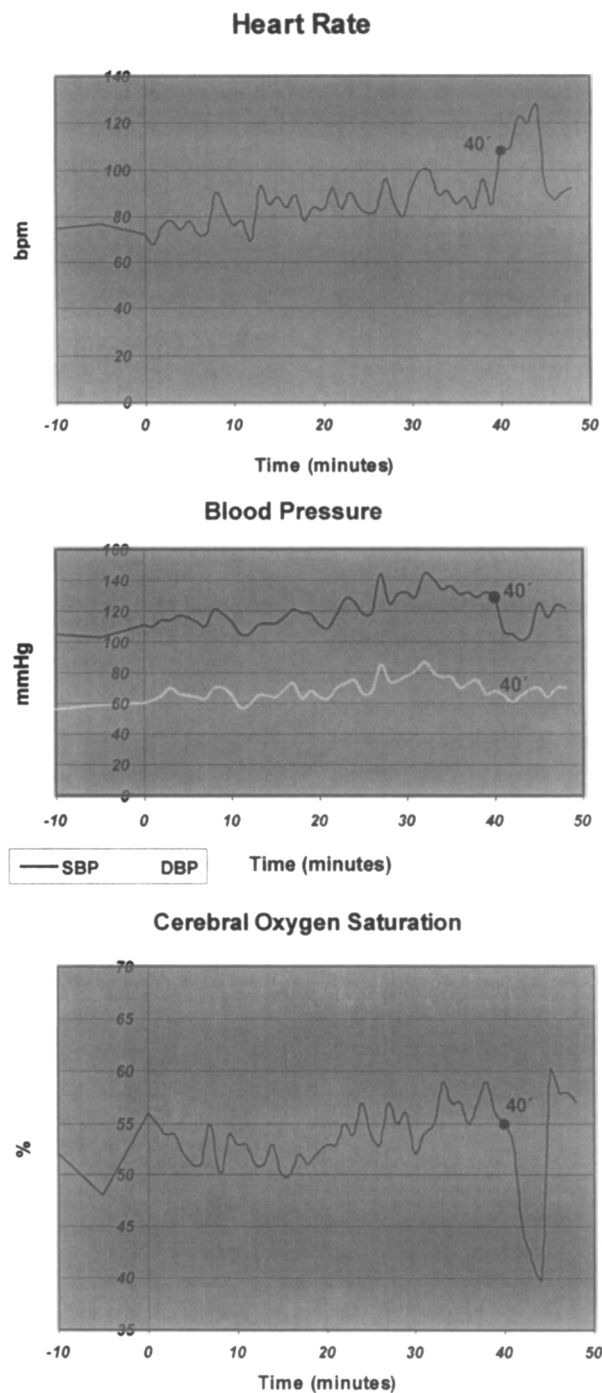


Figure. Heart rate (beats per minute), blood pressure (mm Hg), and cerebral oxygen saturation (%) modifications during head-up tilt test in our patient. SBP, Systolic blood pressure; DBP, diastolic blood pressure.

disturbances are related to cerebral arteriolar vasoconstriction and vasodilatation, respectively.^{13,14}

The introduction into the head-up tilt test of new monitoring devices that permit assessment of the brain's hemodynamic and/or metabolic status has improved the knowledge of the pathophysiology of syncope, showing previously unknown response patterns and allowing a more accurate diagnosis. If we had not monitored a "cerebral function" parameter during the head-up tilt testing, our patient would have been labeled as "simulator" or as having a psychogenic disorder because of the lack of objective and significant systemic hemodynamic disturbances. Taking into account that head-up tilt test protocols usually do not include methods to evaluate cerebral hemodynamic or metabolic status, the incidence of cerebral syncope has probably been underestimated. However, in our experience with children, its incidence seems to be very low, because among 295 patients who have been studied, only the patient in this case report was found to have this kind of syncope.

This response supports the hypothesis that the presence of primary disturbances in cerebral vascular regulation can be involved in the pathogenesis of

neurocardiogenic syncope.^{9,11,12} It is possible that a spectrum of clinical responses may occur during a head-up tilt test, ranging from pure cerebral syncope to pure hemodynamic syncope. Patients may present with more or less influence of cerebral or hemodynamic alterations. It seems clear that further investigation is needed to better define the multiple ways that syncope may occur in children and adults.

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