

PEDIATRIC NEUROLOGY BRIEFS

A MONTHLY JOURNAL REVIEW

J. GORDON MILLICHAP, MD. EDITOR
JOHN J. MILLICHAP, MD. ASSOCIATE EDITOR

Vol. 28, No. 4

April 2014

SYNCOPE

SYMPTOMS, SIGNS, AND EEG CHANGES DURING REFLEX SYNCOPE

Investigators at Leiden University Medical Centre, The Netherlands, studied the symptoms, video data of signs, and EEG changes during tilt-induced vasovagal syncope in 69 subjects, age range 12-84 years (mean 46 years). Reflex syncope occurred during 92 (12.8%) of a total of 720 tilt-table tests, presyncope in 101 (14.1%), and orthostatic hypotension in 84 (11.7%). The average duration of loss of consciousness was 22.4 s (range 4-55 s). EEG slowing preceded the onset of loss of consciousness. Flattening of the EEG indicates more profound circulatory changes and cerebral hypoperfusion than EEG slowing alone. 'Slow-flat-slow'-EEG is associated with a lower minimum blood pressure, longer maximum RR-interval, more frequent asystole, and longer duration of loss of consciousness than the 'slow'-EEG group.

Clinical signs during syncope were of 4 types, based on their relation to the EEG: Type A signs include loss of consciousness, eye opening and general stiffening and occur during the first slow and flat phases in the EEG and end in the second slow phase. Type B signs (myoclonic jerks) occur when the EEG is slow, and are abolished with EEG flattening. Type C signs (making sounds, roving eye movements, and stertorous breathing) occur only in the EEG flat phase, whereas type D signs (dropping the jaw and snoring) occur either in slow or flat phases. The occurrence of specific clinical signs depends on whether the EEG shows flattening. Events occurring before syncope included sweating, pallor and yawning; during syncope, eyes open, dilated pupils, oral automatisms, head and jaw dropping, and arm raising; and after syncope, sweating and pallor. Whether the pattern of signs may be used to infer cause of the syncope remains to

PEDIATRIC NEUROLOGY BRIEFS © 1987-2014, ISSN 1043-3155 (print) 2166-6482 (online), is published monthly and covers selected articles from the world literature. The Editor is Pediatric Neurologist and the Associate Editor, Pediatric Epileptologist and Neurologist at the Ann & Robert H. Lurie Children's Hospital of Chicago; Northwestern University Feinberg School of Medicine, Chicago, IL. PNB is a continuing education service designed to expedite and facilitate the review of current scientific information for physicians and other health professionals. Apply to PediatricNeurologyBriefs.com for Subscriptions (12 issues, January-December). Digital Edition PDF: \$72; Print + Free Digital: \$96 within US/UK, \$128 outside US/UK. Institutions: Digital Edition IP Access \$188, Print + Free Digital \$228. Mailing address for subscription: Pediatric Neurology Briefs Publishers, PO Box 11391, Chicago, IL 60611

be determined. Syncope resulting in short, shallow hypoperfusion will elicit signs corresponding to the slow EEG pattern, whereas a cause resulting in a quick circulatory standstill will result in signs typical of slow-flat-slow EEG pattern. Whereas eyes are open in syncope and in seizures, they are commonly closed in psychogenic attacks [1]. (van Dijk JG, Thijs RD, van Zwet E, et al. The semiology of tilt-induced reflex syncope in relation to electroencephalographic changes. *Brain* 2014 Feb;137(Pt 2):576-85).

COMMENTARY. Syncope is defined as a transient loss of consciousness resulting from cerebral hypoperfusion/hypoxia. Vasovagal reflex syncope is the most common form of syncope, sometimes called neurocardiogenic or neurally mediated syncope. The autonomic nervous system is activated, resulting in low blood pressure, decreased cardiac output, vasodilatation and bradycardia. Recognized usually by the pattern of symptoms and signs only, the present study provides a correlation between symptoms, signs, video and EEG data, leading toward a more definite diagnosis. Vasovagal syncope is differentiated from other, rare causes of syncope, including cardiac (ventricular tachycardia, long QT syndrome, Wolff-Parkinson-White syndrome, and atrioventricular block), and non-cardiovascular pseudo-syncope (reflex anoxic seizures or psychogenic causes) [2].

The differentiation of syncope from seizure (faint from fit) [3] is often difficult. One authority finds in patients presenting with anoxic-epileptic seizures the epileptic component is usually clonic, whereas the nonepileptic convulsive syncope is an arrhythmic tonic extension or spasm [4]. Opisthotonus is common in asystolic syncope in young children but was not seen in the present cohort of older patients [5]. In a study of 141 children referred for evaluation of syncope, 78% had simple neurocardiogenic syncope, 38% had syncopal convulsions, and 2.8% had concurrent epilepsy. The EEG performed in 91 (64%) subjects was diagnostic for epilepsy in 1 (1.4%). MRI, and/or EKG/Holter monitoring/stress testing were primarily normal or nondiagnostic. A detailed medical history was the most useful diagnostic tool [6].

References.

1. Chung SS, et al. Ictal eye closure is a reliable indicator for psychogenic nonepileptic seizures. *Neurology*. 2006 Jun 13;66(11):1730-1.
2. McLeod KA. Syncope in childhood. *Arch Dis Child*. 2003 Apr;88(4):350-3.
3. Stephenson JB. Fits and Faints. London: Blackwell Scientific Publications; 1990:202.
4. Stephenson JB. Syncopes and other paroxysmal events. *Handb Clin Neurol*. 2013;112:861-6.
5. van Dijk JG, et al. *Brain*. 2014 Feb;137(Pt 2):576-85.
6. DiMario FJ Jr, et al. *J Child Neurol*. 2011 May;26(5):548-51.

HEADACHE DISORDERS

POST-LUMBAR PUNCTURE HEADACHE AND NEEDLE GAUGE

Investigators from the Oncology Unit, Royal Children's Hospital, Melbourne, Australia, compared the frequency of headache and procedure time following lumbar puncture (LP) in a randomized crossover trial using a 25-gauge compared to a 22-gauge needle. LP headache occurs within 7 days after the procedure, becomes worse within 1.5 min of standing up and improves within 30 min of lying down. As part of their treatment