

Syncope in children and adolescents: Evaluation and treatment

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Syncope in children is most often neurally mediated and usually has a natural history of spontaneous resolution or improvement. Syncope is defined as the temporary loss of consciousness resulting from a reversible disturbance of cerebral function. It is characterized by a loss of consciousness due to a lack of cerebral blood flow, rapid or sudden onset, falling of the patient, if not supported, and transiency of the attack. In children, it is most often benign, but may sometimes herald a more serious, potentially life-threatening cause. The main purpose of the present paper is to propose an evaluation scheme that will allow the physician involved in the care of children to differentiate the life-threatening causes of syncope with potential for injury or sudden death from the common, more benign neurally mediated syncope. Secondly, the present article facilitates the identification of the patient with neurally mediated syncope who may benefit from medical therapy and distinguishes syncope from the more frequent noncardiac 'spells' of childhood.

Key Words: *Evaluation; Syncope*

Syncope is defined as the temporary loss of consciousness resulting from a reversible disturbance of cerebral function. It is characterized by a loss of consciousness because of a lack of cerebral blood flow; rapid or sudden onset; falls by the patient if he or she is not supported; and transient attacks. In children, it is most often benign, but may sometimes herald a more serious and potentially life-threatening cause.

Syncope is not an uncommon problem because it is estimated that 20% of all children will experience at least one episode of fainting before the end of adolescence (1). Before the age of six years, syncope is unusual except in patients with seizure disorders, breath-holding episodes and primary cardiac dysrhythmias.

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La syncope chez les enfants et les adolescents : L'évaluation et le traitement

RÉSUMÉ : Règle générale, la syncope chez les enfants est à médiation neurale et présente une résolution ou une amélioration spontanée. La syncope se définit comme une perte de conscience temporaire causée par une perturbation réversible de la fonction cérébrale. Elle se caractérise par une perte de conscience secondaire à une carence du débit sanguin cérébral, une apparition rapide ou subite, une chute du patient s'il n'est pas soutenu et le caractère transitoire de la crise. Chez les enfants, elle est généralement bénigne, mais elle cache parfois une cause plus grave, au potentiel fatal. Le présent article vise à proposer un schéma d'évaluation qui permettra au médecin qui participe aux soins des enfants de distinguer les causes potentiellement fatales de syncope avec possibilités de lésion ou de mort subite d'une syncope à médiation neurale fréquente, plus bénigne. De plus, il facilite le dépistage du patient atteint d'une syncope à médiation neurale qui peut profiter d'une pharmacothérapie et distingue la syncope des épisodes non cardiaques plus courants dans l'enfance.

death from the common, more benign, neurally mediated syncope (NMS). Secondly, this paper facilitates the identification of the patient with NMS who may benefit from medical therapy and distinguishes syncope from the more frequent noncardiac 'spells' of childhood.

CAUSES OF SYNCOPE

Syncope can be classified into three major categories: NMS, cardiovascular syncope and noncardiovascular syncope (Table 1). The second category represents only a small proportion of the cases of syncope, but it is responsible for most of the anxiety on the part of parents that surrounds the diagnosis of syncope due to its potentially life-threatening causes.

NMS, or vasovagal syncope, is the most common cause of syncope in young patients. It seems to be related to the beta-adrenergic hypersensitivity of baroreceptors in the

TABLE 1 : Causes of syncope**Neurally mediated syncope****Cardiovascular syncope**

Primary

- Left ventricular outflow obstruction
- Right ventricular outflow obstruction
- Pulmonary hypertension
- Eisenmenger
- Cardiomyopathy

Arrhythmias

- Tachyarrhythmias
 - Long QT syndrome
 - Brugada syndrome (familial ventricular fibrillation)
 - Wolff-Parkinson-White syndrome
- Supraventricular tachycardia
- Ventricular tachycardia
 - postoperative
 - idiopathic
 - right ventricular dysplasia

Bradycardias

- Sick sinus syndrome
- Heart block

Noncardiovascular syncope

- Basilar migraine
- Seizures
- Vertigo
- Hyperventilation
- Situational (cough, micturition, stretch, hair grooming, defecation)
- Breath-holding spells

vessels and the mechanoreceptors of the left ventricle after subtle changes in postural tone, circulating volume or the direct release of catecholamine from higher cerebral centres. This hypersensitivity results in an efferent response consisting of peripheral alpha-adrenergic withdrawal and enhanced parasympathetic tone.

NMS is recognized by a constellation of signs and symptoms, beginning with a prodrome that lasts several seconds to minutes, progressing to a brief period of unconsciousness. The episode may be initiated by one of many provocative events that usually consist of emotional stress such as fear, anxiety or sudden change in posture. Other precipitating states include anemia, dehydration, hunger, physical exhaustion and a crowded or poorly ventilated environment. Premonitory symptoms include lightheadedness, dizziness, nausea, shortness of breath, pallor, diaphoresis and visual changes. Vasodepressor syncope that is associated with exercise has been well described in paediatric patients and most

TABLE 2: Risk factors that suggest cardiac syncope

- Little or no prodrome
- Prolonged loss of consciousness (longer than 5 min)
- Exercise-induced syncope
- Chest pain or palpitations
- History of cardiac disease
- Familial history of long QT syndrome, cardiomyopathy or sudden death

commonly occurs immediately after the termination of an activity.

Cardiac syncope is less common than NMS, but a thorough evaluation is required to ensure the detection of potentially life-threatening diagnoses and to provide accurate prognostic information. Cardiac causes can be separated into two categories: primary cardiac anomalies (right or left heart obstruction, pulmonary hypertension, cyanotic spells in cyanotic heart defects, hypertrophic or congestive cardiomyopathies); and arrhythmias that can lead to syncope in many conditions (long QT syndrome, Brugada syndrome [familial ventricular fibrillation], postoperative atrial flutter or ventricular tachycardia VT, Wolff-Parkinson-White syndrome [WPW] associated with atrial tachycardia [atrial fibrillation], and rarely, idiopathic VT or VT associated with arrhythmogenic right ventricular dysplasia) (2-4).

Clinical features that suggest an underlying cardiac problem are listed in Table 2.

CLINICAL EVALUATION AND INVESTIGATION

A thorough clinical evaluation, including history and physical examination, is the most important part of the investigation. The history usually indicates that the diagnosis is related to cardiac causes (Table 2), NMS (prodrome, associated symptoms, triggering event) or noncardiac syncope (migraine, history of seizures, etc). A family history of syncope, cardiomyopathy or sudden death will direct the diagnosis towards potentially severe cardiac causes such as long QT syndrome and familial hypertrophic cardiomyopathy. As stated above, NMS that is associated with exercise does exist, but a more serious cardiac cause should always be eliminated in exercise-related syncope.

The physical examination should include supine and standing blood pressures, and a complete cardiac examination with emphasis on the pulse rate, and clicks, gallops and murmurs that would reveal a cardiac pathology.

An electrocardiogram (ECG) should always be included in all initial evaluations of syncope in children. Emphasis should be placed on the QT interval and T wave morphology for evidence of long QT syndrome; voltage criteria that are consistent with left or right ventricular hypertrophy for the evaluation of obstructive cardiac

lesions or cardiomyopathies; manifest pre-excitation of WPW; and bradycardia, pauses, conduction disturbances or ectopy.

In cases in which an arrhythmia is suspected, a 24 h ambulatory monitor should be used to eliminate frequent ectopy, VT, supraventricular tachycardia, bradycardia, intermittent WPW, heart block or pauses.

In most cases, no further diagnostic tests are needed when the history, physical examination and ECG suggest NMS. However, further diagnostic work-up is required if the diagnosis is unclear or if any of the following conditions exist: exercise-induced syncope that occurs during exertion; chest pain that precedes an episode of fainting; seizure activity; recurrent syncope (more than two or three episodes); and an abnormal cardiac examination. An echocardiogram should be performed to rule out malformations or cardiomyopathies. The tilt-table test came into use as a method for the evaluation of syncope of unknown cause. Its use is controversial in paediatrics because several groups have demonstrated sensitivities of 43% to 57% and specificities of 83% to 100% (5-8). The test usually confirms the diagnosis of NMS that is detected simply on the basis of history, physical examination and ECG. In cases of syncope of unknown origin, the goal of investigation is primarily to rule out life-threatening causes rather than to diagnose NMS; therefore, echocardiography is more useful than the tilt-table test. The author reserves this test for suspected NMS in situations in which the first-line medical therapy failed to relieve symptoms. For exercise-induced syncope, an exercise stress test is mandatory to look for st-T wave changes that are associated with coronary insufficiency and catecholamine-sensitive dysrhythmias (9,10).

MANAGEMENT

In NMS, the first line of treatment is behaviour modification. Conservative therapy is mandatory because there is a tendency for children to grow out of the propensity for syncope. It has been suggested that resolution is related to the recognition of prodromal symptoms and early interventions. The patient is advised to avoid dehydration, long periods of standing and irregular mealtimes. Other simple measures include water and table salt intake to increase plasma volume. When syncope persists despite behavioural changes, medical therapy can be used and usually includes a beta-blocker or fludrocortisone. Fur-

ther therapy is rarely required. Avoidance of potentially toxic drugs is the rule; however, disopyramide has been used with success in some cases (11). Pacemaker therapy is rarely used.

For cardiac syncope, the therapy is usually more clearly defined and directed to the underlying cause. It includes beta-blocker therapy in cases of long QT syndrome, medical therapy for some forms of cardiomyopathy, surgical resection for cardiac obstructive lesions and pacemaker therapy for a sick sinus or heart block. An implantable defibrillator is rarely required in cases of resuscitated cardiac arrest or high risk familial disease (such as long QT syndrome, Brugada syndrome and hypertrophic cardiomyopathy).

CONCLUSIONS

Syncope in children is most often neurally mediated and usually has a natural history of spontaneous resolution or improvement. Conservative measures should be tried before introducing pharmacotherapy. Cardiac syncope is potentially life-threatening and can usually be suspected after the use of simple evaluative measures, including history, physical examination and ECG.

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