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Review

SYNCOPE (NMS) IN ADOLESCENTS

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Syncope is a common, potentially dangerous symptom among children and adolescents who usually undergo numerous and expensive tests with low diagnostic yield offering little insight into the clinical or diagnostic management. A basic understanding of pathophysiology is helpful in counseling these patients and, in selected cases, finding a possible therapeutic approach. Clearer insights have become available with non-invasive head-up tilt table testing with measurement of numerous hemodynamic regulatory responses. Classification of test-reactions are helpful information for the physician and patient.

Descriptors: NEUROCARDIOGENIC SYNCOPE; ADOLESCENTS

INTRODUCTION

Syncope is defined as transient loss of consciousness with loss of postural tone that resolves spontaneously and without medical intervention. It must be kept in mind that syncope is merely a symptom that by itself does not give any key to the underlying etiology. There are multiple potential causes and long lists of differentials to be worked up. Significant morbidity may result if syncope recurs or coincides with injuries especially from certain high-risk occupations. The recurrence-rate is estimated high at 35% to >50% with a high proportion of recurrences within the first two years (1,2). Syncope is a common, potentially dangerous symptom among adolescents who usually undergo numerous and expensive tests with low diagnostic yield offering little insight into the clinical or diagnostic management. According to the literature syncope accounts for 1-3% of hospital admissions in the US and the incidence in youths up to 18 years of age is estimated at about 15 % (3,4). Prognosis is generally felt to be good if patients have no heart disease, are under 45 years of age, suffer from neurally mediated syncope, or from orthostatic hypotension (5,6).

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PATHOPHISIOLOGY

Cerebral perfusion is mostly dependant on arterial blood pressure. Brain tissue cannot store energy. Therefore, a cessation of 3 to 5 seconds of cerebral perfusion results in loss of consciousness/syncope (7). Cerebral perfusion is kept constant by intrinsic and complex feedback systems involving cardiac output, systemic vascular resistance, arterial blood pressure, cerebrovascular resistance with intrinsic autoregulation, metabolic regulation. Cardiac output and total peripheral vascular resistance play the main role in sustaining postural tone and consciousness. With regard to cardiac output the most important determinant is venous filling, preload. Thus, venous pooling in the dependent parts of the body or diminished blood volume will predispose to syncope. Other factors influencing cardiac output should also be considered: cardiac rhythm, valvular heart disease, outlet tract obstruction, myocardial disease. Impaired capacity to increase peripheral vascular resistance during standing is the cause of orthostatic hypotension. Autonomic dysfunction with excessive vasodilatation may play a role in decreasing arterial blood pressure resulting in fainting in the reflex syncopal syndromes.

CLASSIFICATION

Syncope must be differentiated from other conditions associated with real or apparent loss of consciousness (8). The latter include:

- disorders resembling syncope with impairment or loss of consciousness: seizures, intoxication, metabolic disorder (hypoglycemia, hypoxemia, hyperventilation), vertebro-basilar transitoric ischemic attack and others;
- disorders resembling syncope without true loss of consciousness: somatisation disorder, hysteric attack, drop attack, and others.

Syncope itself may be classified as follows:

1. Neurally-mediated reflex syncopal syndromes (NMS). This refers to a reflex that gives rise to vasodilatation and bradycardia;
2. Orthostatic syncope which occurs with impaired autonomic regulation resulting in inadequate reaction of vasoconstriction. (The author feels that this subgroup should actually be summarised into NMS because it includes neural malfunction);
3. Cardiac arrhythmias which usually occur without circulatory demands;

4. Structural heart disease may result in diminished cardiac output on demand;
5. Cerebrovascular disease like steal syndromes can cause syncope.

A limitation of this classifications lies in the fact that more than one pathophysiologic factor may contribute to the symptom. Causes of syncope are summarised in Table 1.

Table 1
Causes of syncope

Tablica 1.
Uzroci sinkope

Neurally mediated reflex syncope Vasovagal faint ("common faint") Carotid sinus syncope Situational syncope Acute haemorrhage Cough, sneeze Gastrointestinal stimulation Micturition, post-micturition Post-exercise Others (weight-lifting, post-prandial) Glossopharyngeal and trigeminal neuralgia
Orthostatic Autonomic failure Primary autonomic failure (pure autonomic failure, multi system atrophy, Parkinson) Secondary autonomic failure (diabetic neuropathy, amyloid neuropathy) Drugs, Alcohol Volume depletion (diarrhoea, Addison)
Cardiac Arrhythmia Sinus node dysfunction Atrioventricular conduction system disease Paroxysmal supraventricular and ventricular tachycardias Inherited syndromes (long-QT, Brugada) Implanted device malfunction (pacemaker, ICD) Drug-induced proarrhythmias
Structural cardiac or cardiopulmonary disease Cardiac valvular disease Acute myocardial infarction/ischemia Obstructive cardiomyopathy Atrial myxoma Acute aortic dissection Pericardial disease Pulmonary hypertension/embolus
Cerebrovascular Vascular steal syndromes (e.g. post coarctation repair)

DIAGNOSTICS

The first important issue is to find out if the patient had syncope or was he suffering from other possible symptoms. Several clinical features suggest specific causes of syncope (8-11).

Age gives clues to the most common etiologies: Pallid syncope in an infant should always be aggressively investigated for myocardial tumors, structural heart disease, myocarditis, long- QT - syndrome, seizure disorder, structural cerebral disease. The most common cause of syncope in adolescents is vasovagal syncope or orthostatic hypotension but differentials work-up should include possible causes like pulmonary hypertension, coronary artery abnormalities, mitral valve prolaps, pregnancy, psychological problems, drug abuse and others.

At any age dysrhythmias, sick sinus syndrome, atrioventricular block, hypoxia, hypoglycemia, volume depletion should be considered. The circumstances of a syncopal attack should be reviewed: syncope that occurs with exercise, in an athlete, or is recurrent could be the first symptom of cardiomyopathy. A family history of sudden death in younger adults should alert for long-QT-syndrome and cardiomyopathy. Therefore, initial evaluation should include:

- careful history from patient and witness about circumstances, attack onset and termination;
- physical examination including orthostatic blodd pressure measurements;
- standard Electrocardiogram.

Answers to the three key questions must be sought: is loss of consciousness attributable to syncope or not, are there important clinical features suggesting the diagnosis, and is heart disease present or not? Features that suggest a non-syncopal attack are e.g.: confusion after attack for more than five minutes, prolonged tonic-clonic movements at the onset of attack, frequent attacks with somatic complaints, association with vertigo. Cardiac causes should be suspected if attack occurred supine, during exercise, preceeded by palpitation, presence of heart disease. Vasovagal syncope may be diagnosed if precipitating events such as fear, severe pain, emotional distress, instrumentation, or prolonged standing are associated with typical symptoms. Orthostatic syncope may be diagnosed if there is documentation of orthostatic hypotension (i.e. decrease of systolic blood pressure = 20 mmHg or to below 90 mmHg in adults) associated with syncope or "presyncope". Features that suggest neurally-mediated syncope are: attack following unexpected unpleasant sight/ sound/ smell, prolongend standing in crowded or warm places, nausea or vomiting associated with the attack, within one hour of a meal, after exertion. Without doubt a patient should be hospitalised if there is suspicion of cardiac cause, if syncope caused severe injury or if there are recurrent syncopes. Head-up tilt table testing is feasable and rewarding in children and adolescents because it may reveal the underlying mechanism of syncope, although one must be aware that a negative test does not exclude the diagnosis of NMS (12-14). Different protocols of head-up tilt table testing may be adopted (8, 15) baring in mind that there is no gold standard to compare results, specifity and sensitivity with.

TREATMENT

Treatment of syncope depends on the type of syncope or better: on the underlying mechanism if this can be found out (e.g. tilt table). There are several reports with a confusing amount of possible treatment options for syncope. Generally, syncope in the adolescent will not need specific treatment as long as somatic and psychological disorders have been ruled out. In neurally mediated syncope options include behaviour modification, salt and and increased fluids (16, 17). Pharmacologic therapy should be reserved for patients with continued symptoms despite behaviour modification. Uncontrolled studies suggest help from beta-adrenergic blockers, alpha-fludrocortisone and alpha-adrenergic agonists (17-20). Pacemaker implantation should be reserved for the very occasional patient with symptomatic cardioinhibitory sncope.

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Sažetak

NEUROKARDIOGENA SINKOPA U ADOLESCENATA

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Sinkopa je čest, potencijalno opasni simptom u djece i adolescenata zbog koje oni budu podvrgnuti mnogobrojnim i skupim testovima niske dijagnostičke i prediktivne vrijednosti glede kliničkog i dijagnostičkog menadžmenta ovih pacijenata. Osnovno poznavanje patofiziologije sinkope pomaže u savjetovanju ovih pacijenata, a u selektiranih slučajeva i u svrhu pronalaženja mogućeg terapijskog pristupa. Jasniji uvid se dobio u ovo stanje primjenom neinvazivnog "head-up tilt table" testom i mjerenjem brojnih hemodinamskih nadzornih odgovora. Klasifikacija test-reakcija je značajna informacija kako za liječnika tako i za pacijenta.

Deskriptori: NEUROKARDIOGENA SINKOPA; ADOLESCENTI