Invited Review

Head-up tilt test – a rationale for using in diagnosis of syncope

Adam Stanczyk
Ward of Internal Diseases, Norbert Barlicki Memorial Teaching Hospital N 1, Medical University of Lodz, Lodz, Poland

Abstract
Fainting and loss of consciousness (including syncope) are one of the most common causes of medical consultations. A detailed history is of particular importance in their diagnosis. It allows to select a group of patients with a high risk of life-threatening condition and to direct further interdisciplinary process with the participation of a cardiologist, neurologist, psychiatrist and psychologist. The head-up tilt test (HUTT) has a special place in the diagnosis of neurocardiogenic syncope. The main purpose of this review is to highlight the indications for HUTT and indicate situations in which it should be avoided due to the lack of added value of the applied therapy.

Key words: syncope, loss of consciousness, head-up tilt test
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Introduction

Syncope - why the definition is important

According to current guidelines of the European Society of Cardiology (ESC)(1), syncope is defined as a transient loss of consciousness due to a generalized decrease in cerebral perfusion, characterized by rapid onset, short duration, and spontaneous complete recovery (1). The same document draws attention to the essence of distinguishing syncope from conditions without loss of consciousness, as well as differentiating situations in which loss of consciousness does not result from a reduction in central nervous system perfusion. It should be emphasized that every syncope is a loss of consciousness, but not every loss of consciousness is a syncope.

Syncope is not a disease, but a symptom that can be inscribed in the clinical picture of various disease entities, or may be the result of a healthy organism's hyperactivity to some external and internal stimuli. Classification of syncope is presented in Table 1. Fainting and loss of consciousness are common problems in the general population. Moreover, the situation is complicated by the fact that the main causes of syncope differ among age groups and depends on comorbidities (2, 3). Reflex syncope is predominant in the general population as well as in young people (3, 4), with the first episode between the ages of 10 and 30, with a peak around the age of 25 (5). The proportion of arrhythmias and organic diseases of the cardiovascular system increases with age (6). The second peak of syncope is after age 65, however, in this group of patients, syncope secondary to cardiovascular diseases prevail (4). In this group, the share of reflex vasovagal mechanisms is estimated at 31-34% of the causes of syncope. The observed episodes of orthostatic hypotension are most often caused by drugs and polypharmacy (4). The largest source of information on the epidemiology of syncope is the Framingham study, with all limitations resulting from the studied population, in which 3% of men and 3.5% of women experienced at least one syncope episode in their lifetime, and the incidence of syncope increased significantly after age 70 (7).

Address for Correspondence: Adam Stanczyk, Ward of Internal Diseases, Norbert Barlicki Memorial Teaching Hospital N 1, Medical University of Lodz, Lodz, Poland
Email: stanczykpl@yahoo.com
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Table 1. Classification of syncope
Reflex syncope (neurocardiogenic)
- Vasovagal (orthostatic, emotional) - Situational - Carotid sinus syndrome - Atypical (without prodromes and/or without apparent triggers and/or atypical presentation
Orthostatic hypotension - OH
- Primary autonomic failure (pure autonomic failure, multiple system atrophy, Parkinson's disease, dementia with Lewy bodies) - Secondary autonomic failure (diabetes, amyloidosis, spinal cord injuries, auto-immune autonomic neuropathy, paraneoplastic autonomic neuropathy, kidney failure) - Drug-induced orthostatic hypotension most common cause of OH (e.g. vasodilators, diuretics, phenothiazine, antidepressants, alcohol) - Volume depletion (hemorrhage, diarrhea, vomiting, etc.)
Cardiovascular syncope
- Bradycardia-dependent (sinus node dysfunction - including bradycardia/tachycardia syndrome, atrioventricular conduction system disease) - Tachycardia-dependent (supraventricular, ventricular) - Structural cardiac diseases (aortic stenosis, acute myocardial infarction/ischemia, hypertrophic cardiomyopathy, cardiac masses -atrial myxoma, tumors, etc., pericardial disease/tamponade, congenital anomalies of coronary arteries, prosthetic valve dysfunction) - Cardiopulmonary and great vessels diseases (pulmonary embolus, acute aortic dissection, pulmonary hypertension)

Diagnosis of loss of consciousness - is always the same?

The key stage in the diagnosis of syncope is to distinguish syncope from situations imitating transient loss of consciousness, such as metabolic disorders (including hypoglycemia, hypoxia) as well as epilepsy, intoxication or transient ischemia of the central nervous system, often mistakenly referred to as "syncope" (1). Accurate physical examination is of key importance, as it often allows for initial diagnosis, prognosis and indicates the need for additional tests. As part of the extended diagnostics of loss of consciousness, we have, among others, echocardiographic examination, extended ECG monitoring using the Holter method (24-48-hour examination, event Holter, extended electrocardiographic telemonitoring, implantable event recorders), 24-hour ambulatory blood pressure monitoring (ABPM), electrophysiological examination, and exercise test. Provocative tests include carotid sinus massage, active standing test, and tilt-test (head-up tilt test - HUTT). However, not all patients need all of them. As mentioned above, the primary element in the diagnosis of patients with syncope is initial assessment (1). Based on the exact history taken from the patient and witnesses of loss of consciousness, physical examination, measurement of arterial pressure in the supine / sitting position and after standing up and analysis of 12-lead resting electrocardiographic (ECG) examination, it is possible to make a preliminary diagnosis and assess the risk of serious cardiovascular events, or sudden cardiac death (1, 2, 8, 9). It is important to determine the situation in which fainting occurred, prodromal symptoms, accompanying symptoms with particular emphasis on the feeling of palpitations, chest pain, dizziness, visual disturbances, pale skin or the occurrence of seizures.

Table 2. Data from initial anamnesis related to the mechanism of syncope*	
Type of the syncope	**Data from the anamnesis**
Vasovagal	syncope caused by emotional stress or orthostatic stress accompanied by typical prodromal symptoms
Situational	syncope during or immediately after the appearance of specific triggers
Orthostatic	syncope after standing up when orthostatic hypotension is documented
Cardiogenic related to ischemia	syncope with ECG documented acute ischemia with or without myocardial infarction
Cardiovascular	syncope in a patient with: - moving atrial myxoma - severe aortic stenosis - pulmonary hypertension - pulmonary embolism - acute aortic dissection
Arrhythmogenic	syncope in a patient with: - persistent bradycardia <40 / min in awaken state or repeated episodes of sinoatrial block or sinus pauses > 3 seconds, - Mobitz II type AV block or III degree AV block, - an alternating block of the right or left branch of HIS bundle, - VT or fast paroxysmal SVT, - episodes of no sustained polymorphic VT - extended or shortened QT interval, - malfunction of the cardiac stimulating system or ICD with the presence of ventricular pauses
AV – atrioventricular, ECG – electrocardiography, ICD – implantable cardio-defibrillator, SVT – supraventricular tachycardia, VT – ventricular tachycardia *Reproduced under CCC license from reference 1.	

Preliminary assessment allows the diagnosis of vasovagal, situational, orthostatic syncope, myocardial ischemia and arrhythmia-dependent syncope (but only in the case of ECG documentation) in up to 23-50% of patients (10, 11).

When the diagnosis of a particular type of syncope on the basis of a preliminary assessment alone is almost certain or highly probable, further investigations can be discontinued (1). In other cases, the initial evaluation may suggest a diagnosis when the features listed in Table 2 are present. In all other situations, or in the case of a family history of sudden cardiac death, it is necessary to extend the diagnostic process.

By using the algorithm for distinguishing patients with low and high risk of events (1), it is possible to reduce hospital admissions from Emergency Department by as much as 29% (12). Proposed examinations in relations to suspected cause of loss of consciousness are presented in Table 3.

The most developed diagnostic options are in the case of arrhythmic syncope. Electrocardiographic monitoring is recommended if there are clinical or ECG data suggesting arrhythmias as a cause of syncope (1). The type of monitoring method and its duration should be appropriate to the frequency of recurrences of syncope; however, the most popular standard 24-hour ECG monitoring using the Holter method is only indicated when syncope occurs more often than once a week. At present, we can successfully use extended remote telemonitoring systems with real-time evaluation of ECG recording in patients with fewer syncope incidents, with observation time limited only by the patient's tolerance, without any barriers in terms of device memory capacity or battery performance. In other cases we can also use implantable event recorders (1, 13).

Table 3. Examination performed in diagnosis of different kind of syncope and loss of consciousness	
Suspected cause of loss of consciousness	**Proposed examinations**
Vasovagal syncope	anamnesis head-up tilt test
Situational syncope	anamnesis provocative test including treadmill test
Sick sinus syndrome	anamnesis carotid sinus massage
Orthostatic hypotension	anamnesis active standing test ABPM neurological consultation
Arrhythmogenic syncope	12-leads resting ECG 24 -hours Holter ECG telemetry implantable arrhythmia recorders (ILR/ICM) electrophysiological study adenosine test
Structural heart disease	echocardiography (TTE, TEE) heart CT/MRI
Psychogenic pseudosyncope	psychiatric consultation head-up tilt test with simultaneous EEG
Cerebrovascular disorders	neurologic consultation cerebrovascular USG /CT angiography head CT/MRI
Epilepsy	neurologic consultation EEG
ABPM – ambulatory blood pressure measurement, CT – computer tomography, ECG – electrocardiography, EEG – electroencephalography, ICM – implantable cardiac monitor, ILR – implantable loop recorder, MRI - magnetic resonance imaging, TTE – transthoracic echocardiography, TEE – transesophageal echocardiography, USG – ultrasonography	

In the case where a structural cause (valve disease, heart tumors, thrombi etc.) of loss of consciousness is suspected, it is advisable to perform echocardiography. However, routine echocardiography in all patients is not cost effective, as it confirms the diagnosis in a small percentage of patients with syncope. In a study of Gana et al. (14) patients with abnormal physical examination and ECG had abnormal findings on echocardiography in only 36%, while less than 1% of patients with normal physical examination group had positive findings on echocardiography.

Tilt test – should not be ordered for everyone

Because diagnostics of broadly defined loss of consciousness should be planned individually for each patient, the indications for echocardiography or ECG monitoring should always be carefully analyzed. It is no different in the case of the tilt test.

HUTT (head-up tilt test) is a test aimed at triggering the neurocardiogenic reflex in predisposed persons. HUTT in the diagnosis of syncope of unknown etiology was introduced in 1986 by Kenny and colleagues (15). Since then, the method of performing the test has changed many times - the tilt angle, methods of pharmacological challenge and the duration of individual phases of the study have been modified (16). The protocol originally described by Ammirati et al. (17), with later modifications known as the "Italian protocol"(18) seems to be the most popular.

The passive phase of the test according to the Italian protocol lasts 20 minutes, after which the active phase (with sublingual trinitroglycerin (TNG, 300-400ug) lasting up to 15 minutes. Other less frequently used test methods are the Westminster protocol including only the passive phase lasting 45 minutes (16), the test with isoproterenol (19, 20) or other possible modifications of the duration of individual phases or the used provocations.

HUTT, like any test, also has drawbacks. These include frequent false positive results in patients with carotid sinus syndrome (21) and other forms of reflex reactions (22). In addition, patients with sinus node disease or paroxysmal atrioventricular block may develop a reflex reaction during HUTT despite no history of syncope (23). The table's angle of inclination is of particular importance. The 60^{o} angle is characterized by the highest sensitivity and specificity, while the reduction of the angle is associated with a reduction in sensitivity, and the slope above 60^{o} is associated with an increase in the number of false positive results (24). Another important determinant affecting HUTT sensitivity (without a relationship to specificity) is the length of the passive phase - the longer the passive phase of the test, the greater the sensitivity (25).

The most significant limitation of HUTT seem to be poor repeatability of the results, estimated at 31-92% with a result confirming the neurocardiogenic reflex and 85-94% with a negative result (26), and the lack of exclusion of the diagnosis of reflex syncope with a negative result of HUTT (1).

Also, do not forget about possible complications of the study. During HUTT, asystole may occur that requires resuscitation in patients with cardiodepressive syncope. After the examination, arrhythmias may occur, mainly attacks of atrial fibrillation (27, 28).

Tilt test - when “YES” and when “NOT”

HUTT is indicated in patients with suspected reflex syncope when initial assessment does not allow to find a diagnosis (1, 15, 19, 24). In a subject with a typical history of vasovagal syncope, preceded by typical predictive symptoms, or with a history of only one syncope, there is no need for a test to provoke neurogenic syncope. When cardioinhibitory syncope is suspected, HUTT could be useful to select candidates to pacemaker implantation when pure cardioinhibitory response is provoked. It should be remembered, that if a mixed or vasodepressor reaction is observed during HUTT, a cardioinhibitory reaction could occur in spontaneous syncope.

However, there are situations when the first episode of syncope is an indication for the provocation of a vasovagal reaction. Patients, whose subsequent loss of consciousness threatens themselves or their surroundings, belonging to professional groups such as bus and truck drivers, working at heights, pilots should be examined in every case of syncope or presyncope. Another, although controversial, exception to this rule is the willingness (of the doctor and patient) to demonstrate to the patient the mechanism of reported ailments, avoid fear of subsequent loss of consciousness and ensure about the harmless nature of the "disease" by provoking symptoms during HUTT under controlled conditions (1). HUTT should not be performed in patients with syncope of unclear etiology whose epilepsy, neurological disorders or other cardiovascular diseases have not been ruled out, as well as to assess the effectiveness of treatment or patients with fainting without loss of consciousness.

If during the diagnosis of syncope an organic heart disease was found, HUTT can be performed only after excluding it as a cause of loss of consciousness. In the elderly, in whom syncope is often caused by orthostatic hypotension, HUTT may be useful in differentiating the mechanism of syncope, then the passive phase of the test becomes the equivalent of a standing test, or to differentiate syncope from “drop attacks”(29). HUTT is the only examination enabling confirmation of postural-orthostatic tachycardia syndrome (POTS), a rare form of orthostatic intolerance, most common in young women (30, 31).

Although HUTT is a relatively safe test, one should remember about contraindications. Neurocardiogenic reactions should not be provoked in pregnant women, patients with severe cerebral atherosclerosis, as well as those in whom syncope is caused by structural heart diseases, confirmed by e.g. echocardiography (1). Depending on the pharmacological challenge used, a protocol with isoproterenol cannot be performed in patients with coronary artery disease (32) or sinus node disease (33, 34).

Contraindications to the test must be remembered not only by the persons performing the examination, but first and foremost by doctors referring patients to diagnostic laboratories, including family doctors, primary care physicians, also cardiologists and neurologists, which would often reduce the waiting time for a specialist`s consultation or hospitalization and would allow to avoid unnecessary patients' disappointments resulted from prolonging the whole diagnostic and therapeutic process.

Do you always need a neurological consultation?

In a group of patients with loss of consciousness, it should be remembered that not everyone requires a full neurological assessment - including ultrasound of the head arteries, EEG, head CT. Such extensive diagnostics is only necessary in cases of suspected loss of consciousness, which is not syncope. In addition, in patients with orthostatic hypotension, one should think about neurological diagnostics if primary autonomic failure is suspected in the course of diseases such as multi-system atrophy, Parkinson's disease, dementia with Lewy bodies.

In the differentiation of reflex and neurological causes of loss of consciousness (including patients with suspected psychiatric disorders and pseudo-syncope) HUTT can be performed with simultaneous EEG monitoring and video recording (35). Detailed psychiatric and psychological assessment is recommended in these patients (36).

It should be remembered that syncope with prolonged bradycardia and hypotension may be accompanied by involuntary urination and stool, asynchronous myoclonus, eye movement, which are an expression of central nervous system hypoperfusion, and can incorrectly be considered convulsions; then a positive HUTT result can protect patients from long-term use of antiepileptic drugs and further consequences resulting from the diagnosis of epilepsy (37, 38).

Conclusion

Head-up tilt test can be useful non-invasive examination for the diagnosis of syncope of unclear etiology when vasovagal mechanism is suspected. Considering the disadvantages and limitations, it should not be performed in every patient, but only in strictly defined situations. It is the only study enabling the diagnosis of postural-orthostatic tachycardia, a form occurring mainly in young women. In addition, HUTT can be useful in the population of elderly patients, in whom, after ruling out cardiogenic syncope, allows for the diagnosis of vasovagal syndrome, orthostatic hypotension and to distinguish syncope from falls. Restrictions of HUTT should not lead to the cessation of testing, but should only encourage more careful and thoughtful qualification, which would allow maintaining the satisfactory sensitivity and specificity of this test. Full diagnosis of loss of consciousness should be carried out in an individualized manner, and the selection of tests and diagnostic procedures should be made rationally to minimize the costs and duration of the entire diagnostic process.

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References

1. Brignole M, Moya A, de Lange FJ, Deharo JC, Elliott PM, Fanciulli A, et al.: 2018 ESC Guidelines for the diagnosis and management of syncope. Eur Heart J 2018; 39: 1883–948. https://doi.org/10.1093/eurheartj/ehy037
2. Alboni P, Brignole M, Menozzi C, Raviele A, Del Rosso A, Dinelli M, et al. Diagnostic value of history in patients with syncope with or without heart disease. J Am Coll Cardiol 2001; 37: 1921-8.
3. Soteriades ES, Evans JC, Larson MG Chen MH, Chen L, Benjamin EJ, et al. Incidence and prognosis of syncope. N Eng J Med 2002; 374: 878-85.
4. Colman N, Nahm K, Ganzeboom KS, Shen WK, Reitsma JB, Linzer M, et al. Epidemiology of reflex syncope. Clin Auton Res 2004; 14 (Suppl 1): 9-17.
5. Sheldon RS, Sheldon AG, Connolly SJ, Morillo CA, Klingenheben T, Krahn AD, et al. Age of first faint in patients with vasovagal syncope. J Cardiovasc Electrophysiol 2006; 17: 49-54.

6. Romme JJ, van Dijk N, Boer KR, Dekker LR, Stam J, Reitsma JB, et al. Influence of age and gender on the occurrence and presentation of reflex syncope. Clin Auton Res 2008; 18: 127-33.
7. Savage DD, Corwin L, McGee DL, Kannel WB, Wolf PA. Epidemiologic features of isolated syncope The Framingham Study. Stroke 1985; 16: 626-9.
8. Piepoli MF, Hoes AW, Agewall S, Albus C, Brotons C, Catapano AL, et al. 2016 European Guidelines on cardiovascular disease prevention in clinical practice. Eur Heart J 2016; 37: 2315–81. Doi: 10.1093/eurheartj/ehw106
9. Priori SG, Blomström-Lundqvist C, Mazzanti A, Blom N, Borggrefe M, Camm J, et al. 2015 ESC Guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death. Eur Heart J 2015; 36: 2793–867. doi: 10.1093/eurheartj/ehv316
10. Crane SD: Risk stratification of patients with syncope in an accident and emergency department. Emerg Med J 2002; 19: 23-7.
11. Croci F, Brignole M, Alboni P, Menozzi C, Raviele A, Del Rosso A, et al. The application of a standardized strategy of evaluation in patients with syncope referred to three Syncope Units. Europace 2002; 4: 351-6.
12. Ungar A, Tesi F, Chisciotti VM, Pepe G, Vanni S, Grifoni S, et al. Assessment of a structured management pathway for patients referred to the Emergency Department for syncope: results in a tertiary hospital. Europace 2016; 18: 457–62.
13. Stańczyk A, Kaczmarek K. Usefulness and safety of implantable loop recorders – initial data.(article in Polish) Lek Wojsk 2009; 87: 73-9.
14. Ghani AR, Ullah W, Abdullah HMA, Sattar Y, Sarwar U, Ahsan I, et al. The role of echocardiography in diagnostic evaluation of patients with syncope-a retrospective analysis. Am J Cardiovasc Dis 2019; 9: 78-83.
14. Kenny RA, Ingram A, Bayliss J, Sutton R. Head-up tilt: a useful test for investigating unexplained syncope. Lancet. 1986; 1: 1352-5.
15. Fitzpatrick AP, Theodorakis G, Vardas P, Sutton R. Methodology of head-up tilt testing in patients with unexplained syncope. J Am Coll Cardiol 1991; 17: 125-30.
16. Ammirati F, Colivicchi F, Biffi A, Magris B, Pandozi C, Santini M. Head-up tilt testing potentiated with low-dose sublingual isosorbide dinitrate: a simplified time-saving approach for the evaluation of unexplained syncope. Am Heart J 1998; 135: 671-6.
17. Bartoletti A, Alboni P, Ammirati F, Brignole M, Del Rosso A, Foglia Manzillo G, et al.: 'The Italian Protocol': a simplified head-up tilt testing potentiated with oral nitroglycerin to assess patients with unexplained syncope. Europace 2000; 2: 339 - 42. Doi: 10.1053/eupc.2000.0125.
18. Almquist A, Goldenberg IF, Milstein S, Chen MY, Chen X, Hansen R, et al. Provocation of bradycardia and hypotension by isoproterenol and upright posture in patients with unexplained syncope. N Engl J Med 1989; 320: 346-51.
19. Waxman MB, Yao L, Cameron DA, Wald RW, Roseman J. Isoproterenol induction of vasodepressor-type reaction in vasodepressor-prone person. Am J Cardiol 1989; 63: 58-65.
20. Brignole M, Menozzi C, Gianfranchi L, Oddone D, Lolli G, Bertulla A. Neurally mediated syncope detected by carotid sinus massage and head-up tilt test in sick sinus syndrome. Am J Cardiol 1991; 68: 1032-6.
21. Accurso V, Winnicki M, Shamsuzzaman AS, Wenzel A, Johnson AK, Somers VK. Predisposition to vasovagal syncope in subjects with blood/injury fobia. Circulation 2001; 104: 903-7.
22. Krzesiński P, Wierzbowski R, Stańczyk A, Gielerak G. Cardiodepressive neurocardiogenic reaction in response to intravenous injection in healthy young men without a prior history of syncope – report of two cases. (article in Polish) Lek Wojsk 2012; 90: 361–6.
23. Sheldon R, Koshman ML. A randomized study of tilt test angle in patients with undiagnosed syncope. Can J Cardiol 2001; 17: 1051-7.
24. Bartoletti A, Gaggioli G, Menozzi C, Bottoni N, Del Rosso A, Mureddu R, et al. Head-up tilt testing potentiated with oral nitroglycerin: a randomized trial of the contribution of a drug-free phase and a nitroglycerin phase in the diagnosis of neurally mediated syncope. Europace 1999; 1: 183-6.
25. Foglia-Manzillo G, Giada F, Beretta S, Corrado G, Santarone M, Raviele A. Reproducibility of head-up tilt testing potentiated with sublingual nitroglycerin in patients with unexplained syncope. Am J Cardiol 1999; 84: 284-8.
26. Leitch J, Klein G, Yee R, Murdick C, Teo WS. Neurally-mediated syncope and atrial fibrillation. N Eng J Med 1991; 324: 495-6.

27. De Castro RR, Mesquita ET, da Nobrega AC. Parasympathetic-mediated atrial fibrillation during tilt test associated with increased baroreflex sensitivity. Europace 2006; 8: 349-51.
28. Heitterachi E, Lord SR, Meyerkort P, McCloskey I, Fitzpatric P. BP changes on upright tilting predict falls in older people. Age Ageing 2002; 31: 181-6.
29. Raj SR. The Postural Tachycardia Syndrome (POTS): pathophysiology, diagnosis & management. Indian Pacing Electrophysiol J 2006; 6: 84-99.
30. Freeman R, Wieling W, Axelrod FB, Benditt DG, Benarroch E, Biaggioni I, et al. Consensus statement on the definition of orthostatic hypotension, neurally mediated syncope and the postural tachycardia syndrome. Auton Neurosci 2011; 161: 46-8. Doi: 10.1016/j.autneu.2011.02.004.
31. Leman RB, Clarke E, Gillette P. Significant complications can occur with ischemic heart disease and tilt table testing. PACE 1999; 22: 675-7.
32. Gatzoulis KA, Mamarelis IE, Apostlopulos T, Dilaveris P, Gialafos J, Toutouzas P. Polymorphic ventricular tachycardia induced during tilt table testing in a patient with syncope and probable dysfunction of the sinus node. PACE 1995; 18: 1075-9.
33. Kenny RA, O`Shea D, Parry SW. The Newcastle protocols for head-up tilt table testing in the diagnosis of vasovagal syncope, carotid sinus hypersensitivity, and related disorders. Heart 2000; 83: 564-9.
34. Petersen ME, Williams TR, Sutton R: Psychogenic syncope diagnosed by prolonged head-up tilt testing. QJM 1995; 88: 209-13.
35. Andrighetto AG, John AB, Barbisan JN, Taborda JG. Medically unexplained syncope and its relationship to psychiatric disorders. Arq Bras Cardiol 1999; 72: 751-60.
36. Zaidi A, Clough P, Cooper P, Scheepers B, Fitzpatric AP. Misdiagnosis of epilepsy: many seizure-like attack have a cardio-vascular cause. J Am Coll Cardiol 2000; 36: 181-4.
37. Giada F, Silvestri I, Rossillo A, Nicotera PG, Manzillo GF, Raviele A: Psychiatric profile, quality of life and risk of syncopal recurrence in patients with tilt-induced vasovagal syncope. Europace 2005; 7: 465-71.

Central Slovakia, Summer 2020. Ljuba Bacharova, Bratislava, Slovakia