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Vasovagal Syncope: Current Concepts of Pathogenesis, Diagnosis and Clinical Significance

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Abstract

Background: Vasovagal syncope (VVS) is a common cause of transient loss of consciousness, particularly among young adults. Its diagnosis remains challenging because of overlapping manifestations with cardiac, neurological and functional disorders. This study aimed to evaluate diagnostic approaches, haemodynamic characteristics and preventive strategies in patients with suspected VVS.

Materials and Methods: Clinical and instrumental findings were analysed in 78 patients with recurrent transient loss of consciousness, including 69 women (88.5%). Assessment comprised clinical history, neurological and cardiovascular examinations, electrocardiography, orthostatic testing and, where indicated, head-up tilt testing, ambulatory ECG monitoring, echocardiography and electroencephalography. Recurrence frequency, precipitating factors and preventive interventions were evaluated.

Results: VVS was confirmed in 57 patients (73.1%), functional disorders in 8 (10.3%), epilepsy in 3 (3.8%) and cardiac causes in 7 (9.0%); the aetiology remained undetermined in 3 (3.8%). Annual syncope frequency was 3 [2; 5] episodes in VVS versus 2 [1; 4] in other diagnostic groups ($p = 0.13$). During active standing, heart rate increased by 18 [12; 25] versus

12 [8; 18] beats/min ($p = 0.028$), while systolic blood pressure decreased by 16 [10; 23] versus 8 [5; 14] mmHg ($p = 0.005$). Head-up tilt testing demonstrated greater hypotensive responses in VVS ($p < 0.001$). Coughing was identified as a potential situational trigger requiring separate diagnostic consideration.

Conclusions: Accurate VVS diagnosis requires integrated clinical and haemodynamic assessment and exclusion of potentially serious alternative causes. Individualised trigger management, physical counter-pressure manoeuvres and appropriate preventive interventions are important components of patient care.

Keywords: vasovagal syncope; reflex syncope; orthostatic intolerance; head-up tilt test; autonomic dysfunction; cough syncope; differential diagnosis.

Introduction

Syncope is a common clinical syndrome encountered by primary care physicians, neurologists, cardiologists and emergency medicine specialists [1, 2]. Syncope is defined as a transient loss of consciousness caused by brief global cerebral hypoperfusion, characterised by a sudden onset, short duration and spontaneous complete recovery of consciousness. Among the various pathophysiological types of syncope, reflex syncope, particularly vasovagal syncope, accounts for a substantial proportion of transient loss-of-consciousness episodes in young and middle-aged individuals. Despite its generally benign prognosis in terms of survival, recurrent syncopal episodes may considerably impair quality of life, restrict occupational and social activities, increase the risk of injury and contribute to anxiety disorders associated with anticipation of another episode of unconsciousness [1, 3].

The contemporary concept of vasovagal syncope (VVS) encompasses a complex interaction between central and peripheral mechanisms of autonomic cardiovascular regulation [1, 2, 4, 5]. The pathophysiological basis of a syncopal episode involves a reflex reduction in systemic arterial blood pressure resulting from a vasodepressor response, which may be accompanied by a cardioinhibitory component manifested as bradycardia or asystole. The relative contribution of these mechanisms determines the haemodynamic phenotype of syncope, its clinical characteristics and the choice of therapeutic strategy.

Current research into the pathogenesis of vasovagal syncope focuses on the mechanisms of baroreflex regulation, central integration of autonomic signals, and changes in venous return, cardiac output and peripheral vascular resistance [4, 5]. Individual characteristics of autonomic reactivity, susceptibility to orthostatic intolerance, emotional and nociceptive triggers,

dehydration, prolonged standing and exposure to high ambient temperatures are also important. However, the presence of typical precipitating factors does not always permit reliable identification of the mechanism underlying loss of consciousness, particularly in patients with concomitant cardiovascular disease, cardiac arrhythmias or atypical clinical manifestations.

One of the most challenging issues in clinical practice remains the differential diagnosis of vasovagal syncope and other causes of transient loss of consciousness [1, 2, 6]. Particular attention should be paid to excluding arrhythmogenic and structural cardiac causes of syncope, which may be associated with an increased risk of sudden cardiac death [6, 7]. Equally important is the differentiation of vasovagal syncope from orthostatic hypotension, postural orthostatic tachycardia syndrome, epileptic seizures and psychogenic non-syncopal episodes. The occurrence of myoclonic movements, brief tonic phenomena or autonomic manifestations during loss of consciousness may complicate clinical interpretation and lead to an erroneous diagnosis of epilepsy [8]. Accordingly, the diagnostic algorithm should be based on a careful assessment of the circumstances surrounding the episode, the characteristics of the prodromal period, the duration of unconsciousness, the pattern of recovery and the findings of targeted cardiological and neurological examinations.

Orthostatic testing, particularly the head-up tilt test, plays an important role in the diagnosis of reflex syncope by allowing characteristic haemodynamic responses to be reproduced and susceptibility to reflex hypotension and bradycardia to be assessed [9]. Additional diagnostic information may be obtained from prolonged electrocardiographic monitoring, implantable cardiac monitors and assessment of autonomic cardiovascular reflexes, where clinically indicated [6, 10].

Despite considerable advances in understanding the pathophysiology of reflex syncope, the optimal management of patients with recurrent episodes remains a matter of debate. In particular, discrepancies between haemodynamic responses observed during provocative testing and the mechanisms underlying spontaneous syncopal episodes complicate the individualisation of treatment strategies.

The aim of the study was to analyse contemporary diagnostic and therapeutic approaches to vasovagal syncope.

Materials and Methods

A clinical and instrumental analysis was conducted using examination data from 78 patients with recurrent episodes of transient loss of consciousness initially considered to represent probable reflex (vasovagal) syncope. Young women predominated in the study population, accounting for 69 (88.5%) participants. The diagnostic assessment was designed to

establish the nature of syncopal episodes, identify possible cardiac, neurological and functional causes of loss of consciousness, evaluate recurrence frequency and assess the effectiveness of therapeutic and preventive interventions.

The examination included a detailed review of medical history, the circumstances and duration of episodes, precipitating factors, prodromal symptoms, characteristics of recovery of consciousness, comorbidities and current medication. The frequency of syncopal and presyncopal episodes during the preceding 12 months, their monthly distribution, injuries resulting from falls, effects on daily activities and the need for emergency medical care were assessed separately. Longitudinal follow-up was intended to document recurrent episodes, changes in their frequency and the severity of prodromal manifestations.

Clinical assessment included neurological and cardiological examinations, measurement of blood pressure and heart rate in supine and standing positions, and 12-lead electrocardiography. Depending on clinical indications, 24-hour or extended ambulatory ECG monitoring, echocardiography, active standing tests, head-up tilt testing and electroencephalography were performed [6, 9]. During orthostatic testing, changes in systolic and diastolic blood pressure, heart rate, the occurrence of typical symptoms and the pattern of haemodynamic response were analysed. Vasovagal syncope was diagnosed on the basis of combined clinical and historical findings and additional investigations, in accordance with contemporary approaches to the evaluation of transient loss of consciousness.

In patients with confirmed vasovagal syncope, both non-pharmacological and pharmacological treatment and prevention strategies were analysed. Non-pharmacological interventions included patient education regarding the generally benign nature of typical reflex syncope, recognition of prodromal symptoms, avoidance of prolonged standing, overheating and dehydration, optimisation of fluid intake, and individual adjustment of dietary salt intake in the absence of contraindications. The use of physical counter-pressure manoeuvres (leg crossing with muscle tensing, isometric handgrip and squatting), compression garments and gradual orthostatic training was evaluated. In patients with frequent recurrences or an inadequate response to non-pharmacological measures, prescribed pharmacotherapy was analysed, particularly agents intended to support blood pressure and alleviate orthostatic intolerance. Treatment effectiveness was assessed through changes in the frequency of syncopal and presyncopal episodes, the duration of recurrence-free intervals, orthostatic tolerance and patients' subjective assessment of daily functioning.

The study was conducted in accordance with the principles of the World Medical Association Declaration of Helsinki, Good Clinical Practice standards and applicable Ukrainian

legislation concerning personal data protection and patients' rights [11]. Medical records were analysed with appropriate safeguards for confidentiality and anonymisation of individual patient data. Information regarding approval by the local ethics committee and procedures for obtaining informed consent should be specified in accordance with the actual approved study protocol.

Statistical analysis was performed using descriptive and comparative statistical methods [12]. Differences were considered statistically significant at $p < 0.05$.

Results and Discussion

Differential diagnostic assessment confirmed vasovagal syncope in 57 (73.1%) patients. Functional disorders presenting with psychogenic non-syncopal episodes of apparent loss of consciousness were identified in 8 (10.3%) individuals, while epilepsy was diagnosed in 3 (3.8%). Cardiac causes of loss of consciousness were established in a further 7 (9.0%) patients. These included cardiac rhythm and conduction disturbances (paroxysmal tachyarrhythmias, clinically significant bradyarrhythmias, sinoatrial or atrioventricular block), as well as structural heart disease potentially affecting systemic haemodynamics. A cardiac origin was assessed on the basis of clinical and electrocardiographic findings, the temporal association between arrhythmias and symptoms, and echocardiographic results. In 3 (3.8%) patients, the aetiology of the episodes remained undetermined.

Among patients with VVS, episodes were predominantly associated with prolonged standing, emotional stress, overheating and painful stimuli. The frequency of syncopal episodes during the preceding year was 3 [2; 5], compared with 2 [1; 4] episodes among patients with other established causes of loss of consciousness (n = 18; U = 394.0; p = 0.13). Prodromal autonomic symptoms, including nausea, sweating, sensations of warmth and visual dimming, were more characteristic of VVS than of other diagnostic categories (Figure 1).

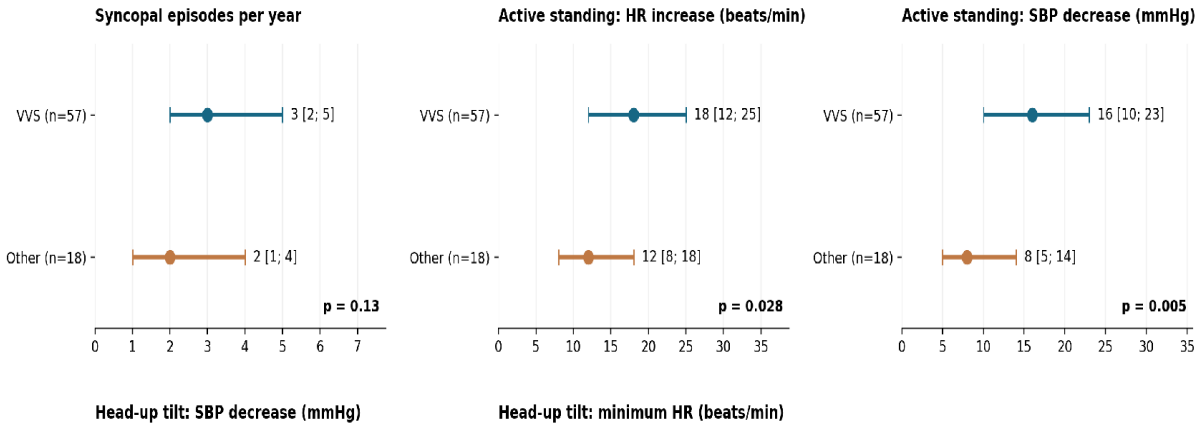


Figure 1 Clinical characteristics by syncope diagnosis

During active standing tests, the median maximum increase in heart rate was 18 [12; 25] beats/min in the VVS group, compared with 12 [8; 18] beats/min in the comparison group ($U = 337.0$; $p = 0.028$). The maximum reduction in systolic blood pressure was 16 [10; 23] and 8 [5; 14] mmHg, respectively ($U = 289.0$; $p = 0.005$). Nevertheless, individual haemodynamic measurements obtained during active standing lacked sufficient specificity to confirm VVS and were therefore interpreted in conjunction with clinical history.

During head-up tilt testing, a more pronounced reduction in systolic blood pressure was registered in patients with VVS: 42 [30; 55] mmHg, compared with 14 [8; 22] mmHg in the comparison group ($U = 168.0$; $p < 0.001$). The minimum heart rate was 48 [41; 57] and 69 [61; 77] beats/min, respectively ($U = 194.0$; $p < 0.001$). The time to the development of typical symptoms during provocative testing in the VVS group was 17 [12; 24] minutes. These relationships reflect the combined contribution of vasodepressor and cardioinhibitory mechanisms; however, their clinical interpretation requires consideration of the testing protocol and whether the patient's habitual symptoms were reproduced.

Particular attention was paid to coughing as a potential precipitating factor for transient loss of consciousness. Coughing causes a sharp increase in intrathoracic pressure, a transient reduction in venous return and cardiac output, and changes in systemic blood pressure and cerebral perfusion. In susceptible individuals, these haemodynamic disturbances may contribute to the development of a syncopal episode. However, cough syncope is classified as situational reflex syncope and is not synonymous with classical vasovagal syncope [6]. Its mechanism is frequently related primarily to the haemodynamic consequences of increased intrathoracic pressure rather than predominantly vagally mediated cardioinhibition. Therefore, episodes of loss of consciousness occurring directly during intense coughing require separate clinical characterisation and differentiation from arrhythmogenic causes, particularly in the absence of a typical vasovagal prodrome.

Analysis of therapeutic and preventive approaches highlighted the importance of identifying individual triggers, teaching physical counter-pressure manoeuvres, optimising fluid intake and providing behavioural recommendations [4-6, 9, 10]. Where coughing acts as a trigger, additional attention should be directed towards identifying and treating the underlying causes of chronic cough, including bronchopulmonary disorders and medication-induced cough. The effectiveness of preventive interventions should be assessed according to recurrence frequency, the number of injury-associated episodes and changes in daily activities.

Conclusions

1. The greatest diagnostic value in vasovagal syncope is achieved through the combined assessment of characteristic clinical history, presenting manifestations and documented haemodynamic responses.

2. Orthostatic measurements and provocative tests complement clinical evaluation but do not replace the need to exclude cardiac and neurological causes of loss of consciousness.

3. Coughing should be considered a distinct situational trigger requiring clarification of the underlying syncopal mechanism and appropriate modification of diagnostic and preventive strategies.

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