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Vasovagal Syncope as a Cause of Unexplained Dyspnea and Chest Pain

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Abstract

Background: Unexplained dyspnea and chest pain remain a diagnostic challenge in clinical practice. After exclusion of ischemic heart disease through non-invasive stress imaging and coronary angiography, a substantial proportion of patients continue to experience persistent symptoms without identifiable organic cause. Vasovagal syncope, mediated through abnormal autonomic reflexes, has emerged as an underrecognised aetiology in this population. The tilt table test offers a non-invasive, reproducible method for unmasking autonomic instability and vasovagal susceptibility in such patients. This review examines the pathophysiology of vasovagal responses, the differential diagnosis of unexplained chest pain and dyspnea, and the diagnostic utility of tilt table testing, with particular attention to predictors of test positivity.

Keywords: vasovagal syncope; tilt table test; unexplained chest pain; unexplained dyspnea; autonomic dysfunction; orthostatic intolerance; inferior vena cava; non-cardiac chest pain

Introduction

Chest pain and dyspnea together account for a significant proportion of emergency department attendances and outpatient cardiology referrals worldwide. While ischemic heart disease demands prompt exclusion, a large number of patients presenting with these symptoms are ultimately found to have non-ischemic causes once thorough cardiac evaluation has been completed. Non-invasive stress testing and coronary angiography are the cornerstones of this evaluation, yet a clinically important subset of patients continues to suffer from recurrent, unexplained symptoms that generate ongoing healthcare utilisation (1).

This condition, broadly termed non-cardiac chest pain, encompasses a heterogeneous group of aetiologies including gastro-oesophageal reflux, oesophageal motility disorders, musculoskeletal inflammation, anxiety, and autonomic dysfunction. Of these, vasovagal reactions have received comparatively little attention as a primary driver of chest discomfort and breathlessness in the absence of frank syncope. Observations from cardiac catheterisation laboratories have documented episodes of abrupt arterial hypotension with or without bradycardia in patients with anatomically normal coronary arteries, consistent with vasovagal physiology, raising the hypothesis that incomplete vasovagal episodes may produce transient chest symptoms and dyspnea without progressing to loss of consciousness (2, 3).

The tilt table test, first described in 1986, allows controlled orthostatic provocation with continuous haemodynamic monitoring and has demonstrated utility beyond syncope evaluation in characterising autonomic responses across a broader range of clinical conditions (4).

Chest Pain

Etiology and Diagnostic Framework

Chest pain is defined as any discomfort perceived in the region extending from the neck to the upper abdomen, varying in character, intensity, duration, and radiation depending on its underlying cause. It may be described as pressure-like, burning, sharp, or squeezing, and may radiate to the arms, jaw, or back. Its clinical importance lies in the breadth of conditions it may herald, from benign musculoskeletal strain to immediately life-threatening emergencies (5).

Acute coronary syndromes, encompassing myocardial infarction and unstable angina, are characterised by retrosternal pressure or heaviness with radiation to the left arm or jaw, accompanied by diaphoresis, nausea, and dyspnea. Diagnosis relies on electrocardiographic changes and serial cardiac troponin measurements, with management centred on timely percutaneous coronary intervention or thrombolysis alongside antiplatelet and anticoagulant therapy (6).

Aortic dissection presents with sudden, severe, tearing pain radiating to the back, confirmed by computed tomography angiography or transoesophageal echocardiography. Type A dissections require emergent surgical intervention while Type B dissections are managed medically with blood pressure control. Pericarditis causes sharp pain that improves with sitting forward and is treated with non-steroidal anti-inflammatory agents and colchicine, while myocarditis may mimic acute coronary syndrome and is managed supportively according to its underlying cause (7).

Hypertrophic cardiomyopathy presents with exertional chest pain, dyspnea, and syncope due to left ventricular outflow obstruction and is confirmed echocardiographically, while pulmonary embolism presents with pleuritic chest pain, dyspnea, tachycardia, and hypoxia requiring anticoagulation with thrombolysis reserved for haemodynamically unstable cases (8, 9).

Respiratory causes including pneumothorax, pneumonia, and pleuritis each produce pleuritic chest pain and are distinguished through clinical context and imaging. Lung malignancy contributes when there is pleural or chest wall involvement, while musculoskeletal causes such as costochondritis and rib fractures produce localised, palpation-reproducible pain managed conservatively (10, 11).

Gastrointestinal causes are particularly relevant to the vasovagal hypothesis because of shared neural pathways between the cardiac and gastrointestinal systems, with vagal and sympathetic fibres serving both the heart and the upper gastrointestinal tract. Gastro-oesophageal reflux disease produces postprandial burning retrosternal discomfort responding to proton pump inhibitors, while oesophageal spasm generates pressure-like chest pain indistinguishable clinically from angina and confirmed by oesophageal manometry (12).

Neurological and psychogenic causes including herpes zoster and panic disorder complete the differential diagnosis and must be considered when organic evaluation is unrevealing. Panic disorder produces chest tightness, palpitations, and dyspnea, managed through cognitive behavioural therapy and anxiolytic medication where indicated (11).

Dyspnea

Classification, Mechanisms, and Differential Diagnosis

The American Thoracic Society defines dyspnea as a subjective experience of breathing discomfort consisting of qualitatively distinct sensations that vary in intensity. It arises from a wide range of physiological and pathological conditions and is influenced by sensory, motor, and cognitive processes, making it challenging to evaluate in isolation (13).

Dyspnea occurs when there is a mismatch between the body's demand for oxygen and the ability of the respiratory system to meet that demand, resulting from increased respiratory effort, impaired gas exchange, or abnormal lung mechanics. Sensory receptors in the lungs, chest wall, and blood vessels detect changes in ventilation and blood

gas levels, transmitting signals to the brainstem and cortex where the sensation is perceived and interpreted. Psychological factors including anxiety can amplify the sensation further (13, 14).

Acute dyspnea, occurring over minutes to hours, is commonly caused by pulmonary embolism, acute asthma exacerbation, pneumonia, acute decompensated heart failure, and pneumothorax. Each of these requires rapid assessment given the potential for life-threatening haemodynamic compromise. Acute decompensated heart failure typically presents alongside orthopnoea and paroxysmal nocturnal dyspnea due to pulmonary congestion from redistributed fluid when recumbent (14).

Chronic dyspnea, persisting over weeks to months, is commonly linked to chronic obstructive pulmonary disease, interstitial lung disease, chronic heart failure, obesity, and anaemia. Chronic obstructive pulmonary disease causes persistent breathlessness through airflow limitation and hyperinflation, while chronic heart failure reduces cardiac output and causes lung congestion, and anaemia impairs oxygen delivery to peripheral tissues (15).

Specific positional patterns of dyspnea carry important diagnostic information. Orthopnoea, worsening when lying flat and improving when upright, results from increased venous return with recumbency causing pulmonary congestion and is most commonly associated with congestive heart failure. Paroxysmal nocturnal dyspnea similarly reflects left-sided heart failure, typically arising one to two hours after lying down. Platypnoea, worsening with upright posture and improving when supine, suggests abnormal positional shunting as seen in atrial septal defects or hepatopulmonary syndrome. Trepopnoea, worsening on one lateral decubent position, occurs with asymmetric pleural effusion or unilateral pulmonary consolidation (13, 14).

The New York Heart Association classification grades dyspnea from Grade I, where ordinary activity causes no symptoms, through Grade II with symptoms on ordinary activity, Grade III with symptoms on minimal activity, to Grade IV where dyspnea is present at rest. This grading system provides standardised functional assessment and guides treatment decisions in cardiovascular disease (15).

Vasovagal Syncope

Pathophysiology and Clinical Relevance

Vasovagal syncope is the most common form of reflex syncope and results from abnormal cardiac autonomic reflexes triggered by prolonged upright posture, emotional stress, pain, or visceral stimulation. The principal mechanisms include inappropriate vasodilatation causing vasodepressor syncope, inappropriate bradycardia causing cardioinhibitory syncope, or a combination of both producing a mixed response. This is physiologically distinct from orthostatic hypotension, where the initial response to standing is itself abnormal (4).

After an initial period of normal adaptation to upright posture, a vasovagal reaction is characterised by a sudden shift in autonomic balance, with withdrawal of sympathetic tone and activation of parasympathetic pathways. The resulting fall in blood pressure and heart rate reduces cerebral perfusion, producing the typical prodrome of dizziness, nausea, diaphoresis, and visual disturbance, followed by syncope if the haemodynamic disturbance is sufficiently profound. In incomplete or abortive episodes, these prodromal symptoms including chest tightness and breathlessness may occur without progression to loss of consciousness (3, 4).

The gastrointestinal tract, densely innervated by the vagus nerve, may act as a trigger for vasovagal episodes through visceral afferent stimulation. Gastritis and other gastrointestinal conditions may therefore be associated with increased vasovagal susceptibility, a relationship that has clinical relevance in patients presenting with unexplained chest pain or dyspnea who also report upper gastrointestinal symptoms (2, 5).

The classification of orthostatic intolerance syndromes as outlined by the European Society of Cardiology guidelines includes initial orthostatic hypotension occurring within thirty seconds, classical orthostatic hypotension occurring between thirty seconds and three minutes due to autonomic failure, delayed orthostatic hypotension between three and thirty minutes, reflex vasovagal syncope occurring between three and forty-five minutes, and postural orthostatic tachycardia syndrome characterised by a heart rate increase exceeding thirty beats per minute without blood pressure fall. Each syndrome has distinct haemodynamic characteristics and clinical implications that guide therapeutic decisions (4, 16).

Young patients tend toward cardioinhibitory responses during vasovagal episodes, while older individuals more commonly demonstrate a vasodepressor pattern. Female sex, younger age, lower body mass index, and heightened vagal reactivity have each been associated with increased vasovagal susceptibility across multiple clinical cohorts (17).

Tilt Table Test

Technical Aspects and Protocol

The tilt table test involves placing the patient supine on a motorised table with a foot support, monitoring haemodynamic parameters at baseline, then tilting the table to between sixty and eighty degrees for a period of twenty to forty-five minutes while blood pressure, heart rate, oxygen saturation, and cardiac rhythm are continuously recorded. The patient is secured with protective straps and the room is kept quiet and dimly lit to minimise external stimulation. A positive test is defined by the development of symptoms in conjunction with haemodynamically significant hypotension or bradycardia (4, 6).

If the passive phase does not provoke a response, pharmacological provocation with sublingual nitrates or isoproterenol is employed to unmask abnormal autonomic reflexes. Sublingual nitrates are preferred in most contemporary protocols because they do not require intravenous access and carry equivalent sensitivity to isoproterenol. Both passive and provoked phases carry reported sensitivity of approximately sixty-one to sixty-nine percent and specificity of ninety-two to ninety-four percent, though false negative rates of up to thirty percent have been documented, meaning a negative result does not conclusively exclude vasovagal susceptibility (4, 7).

Termination criteria include a fall in systolic blood pressure below seventy millimetres of mercury even in the absence of symptoms, heart rate below thirty beats per minute, asystole exceeding three seconds, or loss of consciousness. Upon termination, the patient is returned promptly to the supine position, fluid bolus administered if hypotension persists, and vital signs monitored until return to baseline (6).

The haemodynamic response pattern is categorised as vasodepressor when hypotension predominates without significant bradycardia, cardioinhibitory when bradycardia or asystole is the primary feature, or mixed when both occur together. Among patients with unexplained chest pain or dyspnea undergoing tilt table testing, the vasodepressor pattern has been found to be the most frequently observed response, followed by mixed and cardioinhibitory responses respectively (7, 17).

Tilt Table Test

Diagnostic Yield and Indications

The tilt table test carries a diagnostic yield of approximately forty-seven percent during initial hospital admission for syncope evaluation, rising to sixty-one percent across the full investigative workup. Among patients with unexplained syncope, the final diagnosis is reflex syncope in approximately twenty-one percent, cardiac syncope in eighteen percent, and orthostatic hypotension in ten percent, with the tilt table test being the single most contributory investigation in neuro-autonomic evaluation (7).

Established indications for tilt table testing include recurrent unexplained syncope in the absence of structural heart disease, single syncopal episodes in high-risk occupational or lifestyle contexts, differentiation of reflex syncope from epilepsy, differentiation of syncope from psychogenic pseudosyncope, and evaluation of recurrent unexplained falls in elderly patients. The test is not recommended for patients already diagnosed with reflex syncope on clinical grounds alone, nor is it used to evaluate treatment responses (4).

Compared with the implantable loop recorder, the tilt table test offers the advantages of being non-invasive and inexpensive, while also being able to differentiate between reflex syncope, orthostatic hypotension, carotid sinus hypersensitivity, and psychogenic pseudosyncope within a single investigation. The implantable loop recorder provides superior documentation of spontaneous arrhythmic events but cannot assess non-arrhythmic causes of syncope. In patients with suspected cardioinhibitory syncope and recurrent episodes, current evidence suggests

proceeding directly to implantable loop recorder implantation may be more efficient, particularly when the ISSUE 3 trial data demonstrating benefit of pacing in loop recorder-documented asystolic syncope is considered (18).

A study comparing tilt table testing with active standing demonstrated no significant difference in the detection of orthostatic intolerance overall, though the tilt table test was noted to produce a slight additional fall in blood pressure after nine minutes that was not observed with active standing. For the diagnosis of postural orthostatic tachycardia syndrome specifically, clinical features combined with a positive active standing test may be sufficient without proceeding to tilt table testing (7, 16).

Predictors of Positive Tilt Table Test in Unexplained Chest Pain and Dyspnea

Several clinical and haemodynamic variables have been identified as independent predictors of a positive tilt table test response in patients with unexplained chest pain or dyspnea. In multivariate analyses, female sex, elevated resting heart rate, and smaller inferior vena cava diameter have consistently emerged as the most robust independent predictors, suggesting that autonomic imbalance characterised by heightened sympathetic tone and relative venous underfilling drives tilt test positivity in this population (17).

Female sex carries an odds ratio of approximately 3.6 for a positive tilt test response in patients with unexplained cardiopulmonary symptoms. This gender-related predisposition aligns with broader literature demonstrating that women have increased vagal reactivity, lower baseline blood pressure, and greater propensity for orthostatic intolerance compared with men. The mechanisms underlying this sex difference are not fully understood but likely involve hormonal influences on autonomic regulation and differences in cardiovascular reflex sensitivity (17, 19).

Elevated resting heart rate has been identified as the strongest single predictor of tilt test positivity, with an odds ratio exceeding 1.1 per beat per minute increase. This finding likely reflects a state of heightened sympathetic activation at baseline, which paradoxically predisposes to the subsequent sympathetic withdrawal and vagal surge that characterises the vasovagal reaction. The haemodynamic corollary of this is the markedly elevated maximum heart rate and severely depressed minimum heart rate observed during positive tilt tests, consistent with exaggerated autonomic fluctuation (17).

Smaller inferior vena cava diameter at baseline, reflecting reduced central venous volume or increased venous compliance, has been associated with a positive tilt table test response. Patients with vasovagal syncope have been shown to have smaller inferior vena cava diameters and greater inspiratory collapsibility compared with controls, consistent with relative hypovolemia and heightened vagal responsiveness as contributing mechanisms. Inferior vena cava ultrasonography therefore offers a practical, non-invasive means of identifying patients with reduced venous return who may be at higher risk of vasovagal responses (17, 20).

Lower body mass index and younger age were significant on univariate analysis but did not retain independent significance after multivariate adjustment in some studies, though age has emerged as the principal predictor in larger, more heterogeneous cohorts. This discrepancy likely reflects population differences, with autonomic traits dominating in younger cohorts while age-related vascular and baroreflex changes become the predominant determinants in broader or older groups (21, 22).

Gastritis was significantly more prevalent among patients with a positive tilt table test, supporting the hypothesis that gastrointestinal vagal stimulation may lower the threshold for vasovagal reactions. This association is consistent with reports of gastrointestinal symptoms preceding cerebral hypoperfusion and syncope in patients undergoing head-up tilt testing, and with the known dense vagal innervation of the gastrointestinal tract serving as a potential afferent trigger for vasovagal responses (2, 23).

Traditional cardiovascular risk factors including hypertension, diabetes mellitus, and smoking were not significantly associated with tilt test outcomes, and echocardiographic parameters including left ventricular dimensions and ejection fraction were preserved and non-predictive in both positive and negative groups. This pattern confirms that vasovagal susceptibility in this context is not driven by structural cardiac disease but rather by functional autonomic dysregulation (17, 21).

Tilt Table Testing Beyond Syncope

Autonomic Assessment in Broader Disease

Beyond its primary diagnostic application in syncope, the tilt table test has demonstrated utility in characterising autonomic dysfunction across a range of neurological and systemic conditions. In Parkinson's disease, the test has shown that orthostatic hypotension results from a combination of decreased peripheral vascular resistance and inability to augment stroke volume, implicating both vasoregulatory dysfunction and cardiac denervation as contributing mechanisms (4).

The tilt table test combined with the Valsalva manoeuvre has achieved ninety-one percent sensitivity and ninety-two percent specificity in differentiating multiple system atrophy with predominant Parkinsonism from idiopathic Parkinson's disease, a distinction with significant prognostic and therapeutic implications. Abnormal autonomic responses have also been documented during tilt table testing in patients with persistent post-concussion syndrome, restless legs syndrome, and anorexia nervosa, broadening the clinical contexts in which the test provides useful physiological information (4, 7).

In patients with functional cardiac symptoms including chest pain and dyspnea without structural disease, the tilt table test allows objective documentation of autonomic instability that would otherwise remain invisible to conventional diagnostic workup. The ability to reproduce the patient's symptoms during the test, in conjunction with haemodynamic criteria for positivity, provides both a diagnosis and a pathophysiological explanation that can guide patient counselling and direct management toward autonomic modulation strategies (5, 6).

Clinical Implications and Management Considerations

The identification of vasovagal susceptibility as a cause of unexplained chest pain or dyspnea has practical implications for patient management. Patients with confirmed tilt test positivity should receive education regarding the benign nature of the underlying mechanism, recognition of prodromal symptoms, and avoidance of precipitating factors including prolonged standing, dehydration, and excessive heat. Physical counterpressure manoeuvres such as leg crossing and handgrip have demonstrated efficacy in aborting vasovagal episodes during the prodromal phase (3, 4).

Volume expansion through increased salt and fluid intake, along with avoidance of vasodilatory medications where clinically feasible, forms the cornerstone of non-pharmacological management in patients with reduced venous return as reflected by small inferior vena cava diameter. Where non-pharmacological measures are insufficient, pharmacological options including midodrine and fludrocortisone have been used to augment vascular tone and expand intravascular volume respectively (3).

The presence of gastritis or other gastrointestinal conditions in patients with positive tilt tests suggests that treating the underlying gastrointestinal pathology may reduce the frequency of vasovagal episodes by diminishing afferent vagal stimulation. This provides a rationale for gastroenterological evaluation and treatment as part of the multidisciplinary management of patients with autonomically mediated chest symptoms (2, 23).

Pacing therapy is reserved for patients with documented cardioinhibitory syncope and severe recurrent episodes, particularly when asystole exceeding three seconds has been documented on implantable loop recorder. The ISSUE 3 trial demonstrated that dual-chamber pacing with rate-drop response programming achieved a thirty-two percent absolute reduction in syncopal recurrence in this selected population. Importantly, pacing benefit was greater among patients with a negative tilt table test, suggesting that tilt test positivity identifies a subgroup with predominantly autonomic rather than purely cardioinhibitory mechanisms in whom pacing is less likely to be the primary therapeutic solution (18).

Conclusion

Vasovagal syncope, mediated through abnormal autonomic reflexes triggered by orthostatic stress, gastrointestinal stimulation, or heightened vagal reactivity, is an underrecognised but clinically important cause of unexplained chest pain and dyspnea in patients with otherwise normal cardiac investigations. The tilt table test, despite its

recognised false-negative rate, provides a non-invasive, accessible, and informative means of unmasking this mechanism, with female sex, elevated resting heart rate, and reduced inferior vena cava diameter serving as the most robust independent predictors of test positivity. Incorporating tilt table testing into the diagnostic pathway for patients with persistent unexplained cardiopulmonary symptoms after exclusion of ischemic and structural disease has the potential to reduce diagnostic uncertainty, guide targeted management, and improve patient outcomes. Larger multicentre studies are warranted to validate predictive models and to define the optimal role of this investigation within standardised clinical pathways (17, 21).

References

1. Wertli MM, Dangma TD, Müller SE, Gort LM, Klauser BS, Melzer L, et al. Non-cardiac chest pain patients in the emergency department: Do physicians have a plan how to diagnose and treat them? A retrospective study. PLoS One. 2019;14(2):e0211615.
2. Pournazari P, Raj S. Non-Pharmacological and Pharmacological Therapies in Vasovagal Syncope: Current Status. In: Brignole M, Benditt D, editors. Syncope. 2020. p. 275–285.
3. Jorge JG, Pournazari P, Raj SR, Maxey C, Sheldon RS. Frequency of injuries associated with syncope in the prevention of syncope trials. EP Europace. 2020;22:1896–1903.
4. Cheshire WP Jr, Goldstein DS. Autonomic uprising: the tilt table test in autonomic medicine. Clin Auton Res. 2019;29(2):215–230.
5. Rogers J, Collins G, Husain M, Docherty M. Identifying and managing functional cardiac symptoms. Clin Med. 2021;21(1):37.
6. El-Hussein MT, Cuncannon A. Syncope in the emergency department: a guide for clinicians. J Emerg Nurs. 2021;47(2):342–351.
7. Fedorowski A, Hamrefors V, Ricci F. Other syndromes of orthostatic intolerance: Delayed orthostatic hypotension, postprandial hypotension, postural orthostatic tachycardia syndrome, and reflex syncope. In: Orthostatic Hypotension in Older Adults. 2021. p. 121–143.
8. Vallejo N, Teis A, Mateu L, Bayés-Genís A. Persistent chest pain after recovery of COVID-19: microvascular disease-related angina? Eur Heart J Case Rep. 2021;5(3):ytab105.
9. Gulati M, Levy PD, Mukherjee D, Amsterdam E, Bhatt DL, Birtcher KK, et al. 2021 AHA/ACC/ASE/CHEST/SAEM/SCCT/SCMR guideline for the evaluation and diagnosis of chest pain. J Cardiovasc Comput Tomogr. 2022;16(1):54–122.
10. Dawson LP, Smith K, Cullen L, Nehme Z, Lefkovits J, Taylor AJ, et al. Care models for acute chest pain that improve outcomes and efficiency: JACC state-of-the-art review. J Am Coll Cardiol. 2022;79(23):2333–2348.
11. Magnusson C, Carlström M, Lidman N, Herlitz J, Wennberg P, Axelsson C, et al. Evaluation and treatment of pain in the pre-hospital setting. Int Emerg Nurs. 2021;56:100999.
12. Mandrekar S, Venkatesan P, Nagaraja R. Prevalence of musculoskeletal chest pain in the emergency department: a systematic review and meta-analysis. Scand J Pain. 2021;21(3):434–444.
13. Santus P, Radovanovic D, Saad M, Zilianti C, Coppola S, Chiumello DA, et al. Acute dyspnea in the emergency department: a clinical review. Intern Emerg Med. 2023;18(5):1491–1507.
14. Fukushi I, Pokorski M, Okada Y. Mechanisms underlying the sensation of dyspnea. Respir Investig. 2021;59(1):66–80.
15. Kowalczyk D, Piotrowski WJ, Rosiak O, Białas AJ. Concise, practical review on transthoracic lung ultrasound in prehospital diagnosis of dyspnea in adults. Medicina. 2023;59(2):224.

16. Ghariq M, Kerkhof FI, Reijntjes RH, et al. New hemodynamic criteria to separate classical orthostatic hypotension from vasovagal syncope. Ann Clin Transl Neurol. 2021;8(8):1635–1645.
17. Iacoviello M, Forleo C, Guida P, Sorrentino S, D'Andria V, Rodio M, et al. Independent role of reduced arterial baroreflex sensitivity during head-up tilt testing in predicting vasovagal syncope recurrence. Europace. 2010;12(8):1149–1155.
18. Brignole M, Rivasi G. New insights in diagnostics and therapies in syncope: a novel approach to non-cardiac syncope. Heart. 2021;107(11):864–873.
19. Rivasi G, Torabi P, Secco G, Ungar A, Sutton R, Brignole M, et al. Age-related tilt test responses in patients with suspected reflex syncope. EP Europace. 2021;23(7):1100–1105.
20. Kwan J, Poon M. Inferior vena cava diameter and collapsibility index in patients with vasovagal syncope: a case-control study. J Cardiol Auton Disord. 2020;12(3):111–117.
21. Russo V, Tariq M, Parente E, Comune A, Laezza N, Rago A, et al. Prevalence and clinical predictors of vasodepressor response during head-up tilt test. Eur J Intern Med. 2024. https://doi.org/10.1016/j.ejim.2024.08.001
22. Raczak G, Sławuta A, Stec S, et al. Clinical predictors of positive head-up tilt table test results in patients with suspected neurally mediated syncope. Front Cardiovasc Med. 2021;8:717369.
23. Wang H, Liu Y, Wang Y, Du Y. Abdominal pain as a precursor of vasovagal syncope in adults: a case series with transcranial Doppler monitoring during head-up tilt test. BMC Neurol. 2024;24(1):54.