

REVIEW | SJIP 2026

Tilt Table Testing in Migraine: Pathophysiological Insights, Clinical Applications, and Future Perspectives

Ananna Rani Sen* 

Department of Physiology, Ad-din Momin Medical College, South Keraniganj, Dhaka, Bangladesh

Abstract

Migraine is a common neurovascular disorder, increasingly recognized to involve cardiovascular autonomic dysfunction. Abnormal autonomic regulation may contribute to orthostatic intolerance, impaired cardiovascular responses to postural stress, dizziness, presyncope, and syncope. Tilt table testing is a standardized, non-invasive investigation that evaluates integrated cardiovascular responses to orthostatic stress and may detect both overt and subclinical autonomic abnormalities. Current evidence indicates that patients with migraine have a higher prevalence of orthostatic intolerance, abnormal heart rate and blood pressure regulation, impaired cardiovascular autonomic responses, and neurally mediated syncope than healthy individuals. These abnormalities may reflect dysfunction of the central autonomic network, altered sympathetic-parasympathetic balance, interactions between trigeminovascular and autonomic pathways, and broader neurovascular dysregulation. The procedure allows objective assessment of cardiovascular autonomic function and assist in the evaluation of selected patients with migraine who experience orthostatic symptoms, presyncope, or syncope. Further multicenter studies using standardized protocols and continuous hemodynamic monitoring are needed to establish the diagnostic and prognostic utility of the test and clarify its potential role in individualized migraine management.

Keywords: Autonomic dysfunction, cardiovascular regulation, migraine, orthostatic intolerance, tilt table testing

1. Introduction

Migraine is one of the most prevalent neurological disorders worldwide, affecting approximately 14-15% of the global population and more than one billion individuals across all age groups¹. It is a leading cause of years lived with disability (YLD), particularly among individuals younger than 50 years. Women are affected nearly three times more frequently than men, resulting in a substantial socioeconomic burden through impaired quality of life (QoL), reduced productivity, and increased healthcare utilization².

Beyond its characteristic recurrent headache, migraine is increasingly recognized as a complex neurovascular disorder in which cardiovascular autonomic dysfunction plays an important role. Traditionally, migraine was considered primarily a vascular disorder. Current evidence, however, recognizes migraine as a complex neurovascular disease involving interactions among the trigeminovascular system, cortical and subcortical pain-processing pathways, and the central autonomic network (CAN). Activation of the trigeminovascular pathway stimulates the release of calcitonin gene-

related peptide (CGRP) and other vasoactive neuropeptides. This process initiates neurogenic inflammation, promotes central sensitization, and alters nociceptive transmission, thereby contributing to migraine pathophysiology^{3,4}.

Increasing evidence suggests that autonomic nervous system (ANS) dysfunction is an integral component of migraine rather than merely an associated clinical manifestation^{5,6}. Patients frequently experience autonomic symptoms, including nausea, vomiting, diaphoresis, palpitations, dizziness, gastrointestinal (GI) disturbances, and orthostatic intolerance during both ictal and interictal periods. Functional abnormalities within the CAN, particularly involving the hypothalamus, insular cortex, anterior cingulate cortex, amygdala, and brainstem autonomic nuclei, are believed to underlie these manifestations⁷. These interconnected structures play a fundamental role in regulating cardiovascular homeostasis and visceral autonomic function.

This narrative review summarizes the current evidence regarding cardiovascular autonomic dysfunction in migraine, with particular emphasis on the physiological mechanisms underlying abnormal

*Address for correspondence:

Dr. Ananna Rani Sen
Department of Physiology, Ad-din Momin Medical College
Container Port Road, Dholeswar, Konda
South Keraniganj, Dhaka-1311, Bangladesh
E-mail: ananya16rani@gmail.com

How to cite this article:

Sen AR. Tilt table testing in migraine: pathophysiological insights, clinical applications, and future perspectives. SAAP J Integr Physiol. 2026;1(3):19-23.

orthostatic responses, methodological variations in tilt table testing (TTT), its clinical applications, current limitations, and future research priorities.

2. Search Strategy

A comprehensive literature search was conducted on PubMed/MEDLINE, Scopus, Web of Science, and Google Scholar to identify relevant articles published in English up to June 2026. The search strategy combined Medical Subject Headings (MeSH) and free-text keywords, including migraine, autonomic dysfunction, tilt table testing, cardiovascular autonomic regulation, orthostatic hypotension, postural orthostatic tachycardia syndrome, vasovagal syncope, baroreflex sensitivity, heart rate variability, and orthostatic intolerance. Appropriate Boolean operators were used to maximize the retrieval of relevant studies.

Inclusion criteria: Original research, systematic reviews/meta-analyses, consensus statements, and international clinical guidelines.

Exclusion criteria: Conference abstracts, editorials, letters to the editor, duplicate publications, non-English articles, and clinically irrelevant studies.

Due to methodological variability (Table 1), quantitative synthesis was considered inappropriate, so a narrative approach was adopted. Notably, the methodological variability could have resulted from differences in study design, participant selection bias, small sample size, migraine subtype, autonomic assessment methods, and TTT protocols.

Table 1: Methodological variability in research studies

Parameter	Variation	Impact
General	Participant selection, diagnostic criteria, autonomic evaluation, TTT protocols, outcome measures	Comparability between studies
Tilt angle	Head-up tilt protocol: 60°, 70°, 80°	Orthostatic stress
TTT duration	10-45 minutes	Autonomic response
BP monitoring	Continuous or intermittent	Transient changes
Migraine subtype	Episodic, chronic, or mixed	Severity of autonomic dysfunction
Study population	Mostly adults. Few children and adolescents	Generalizability across age groups

BP: blood pressure

3. Cardiovascular Autonomic Regulation and Orthostatic Adaptation

Maintenance of arterial BP during postural changes depends on rapid and precisely coordinated autonomic cardiovascular regulation. During the transition from supine to upright position, approximately 500-800 ml of blood is redistributed to the lower extremities because of gravitational forces. This transient blood pooling reduces venous return, stroke volume, and cardiac output. If these hemodynamic changes are not promptly compensated, cerebral perfusion decreases, resulting in dizziness, presyncope, or syncope⁸.

These cardiovascular alterations are detected immediately by arterial baroreceptors located in the carotid sinus and aortic arch. Reduced baroreceptor firing activates sympathetic outflow while simultaneously suppressing parasympathetic activity. The resulting physiological responses include tachycardia, increased myocardial contractility, and peripheral vasoconstriction, all of which help restore arterial pressure. Additional support is provided by the skeletal muscle pump and neurohumoral mechanisms, which together

maintain adequate cerebral perfusion during orthostatic stress^{9,10}.

Failure of these compensatory mechanisms results in disorders of orthostatic regulation, including orthostatic hypotension (OH), postural orthostatic tachycardia syndrome (POTS), vasovagal syncope (VVS), and chronic orthostatic intolerance (COI)^{11,12}. These conditions have been reported more frequently in patients with migraine than in the general population, suggesting that autonomic dysregulation contributes to the clinical manifestations of migraine.

Calcitonin gene-related peptide (CGRP) plays a central role in migraine pathophysiology through trigeminovascular activation, neurogenic inflammation, and vascular regulation. Given its role in cardiovascular homeostasis and vascular tone, CGRP signaling has also been implicated in autonomic cardiovascular regulation¹³. Although CGRP-targeted therapies effectively reduce migraine frequency and severity, their effects on cardiovascular autonomic function and orthostatic responses remain uncertain¹⁴. Consequently, it is unclear whether clinical improvement following CGRP-targeted therapy is accompanied by normalization of autonomic dysfunction or TTT responses. Therefore, prospective multi-center studies assessing CGRP-targeted therapy are warranted.

4. Cardiovascular Autonomic Tests

Several complementary methods are available for evaluating cardiovascular autonomic function, including heart rate variability (HRV) analysis, baroreflex sensitivity testing, conventional autonomic function test (CAFT), and TTT. Tilt table testing provides a comprehensive assessment of integrated HR and BP responses during controlled orthostatic stress. Unlike resting autonomic assessments, TTT evaluates dynamic cardiovascular adaptation and can identify abnormalities that may remain undetected under basal conditions¹⁵. The major tests are presented in Table 2.

Table 2. Cardiovascular autonomic tests used in migraine

Test	Physiological Parameter	Clinical Utility	Limitation
CAFT	Sympathovagal balance	Detects autonomic imbalance	Influenced by respiration and medications
Deep breathing test	Parasympathetic function	Evaluates vagal integrity	Requires patient cooperation
Valsalva maneuver	Baroreflex function	Assesses sympathetic and parasympathetic responses	Technique-dependent
HR response to standing	Parasympathetic function	Detects early autonomic adaptation	Limited sensitivity
BP response to standing	Sympathetic vasoconstriction	Detects OH	May miss mild dysfunction
TTT	Integrated cardiovascular autonomic regulation	Detects OH, POTS, VVS, and subclinical autonomic dysfunction	Protocol variability across centers

HR: heart rate; BP: blood pressure; OH: orthostatic hypotension; POTS: postural orthostatic tachycardia syndrome; TTT: tilt table testing; CAFT: conventional autonomic function test; VVS: vasovagal syncope

5. Tilt Table Testing

Tilt table testing is a standardized, non-invasive procedure that evaluates integrated cardiovascular responses to controlled orthostatic stress (Figure 1). It is widely used in autonomic laboratories to investigate unexplained syncope, OH, POTS, neurally mediated syncope, and other disorders of cardiovascular autonomic regulation^{16,17}.

During orthostatic challenge, inadequate compensatory mechanisms may result in abnormal cardiovascular responses, including excessive

reductions in BP, inappropriate heart rate responses (HRR), delayed hemodynamic recovery, or syncope. By continuously assessing HR and BP during dynamic orthostatic stress, TTT provides an integrated evaluation of cardiovascular adaptation that may complement resting autonomic function tests.

Growing evidence suggests that migraine may be associated with impaired orthostatic cardiovascular adaptation. Abnormalities in sympathetic activation, BP regulation, orthostatic tolerance, and susceptibility to vasovagal responses have been reported in patients with migraine¹⁸. Notably, abnormal responses during TTT have been observed in some patients with otherwise normal CAFT, suggesting that orthostatic challenge may reveal subtle or context-dependent abnormalities that are not apparent at rest^{19,20}.

These findings support the potential role of TTT as a complementary investigation in selected patients with migraine in whom cardiovascular autonomic dysfunction is clinically suspected. However, given the variability of the available evidence, TTT should not be considered a routine investigation for all patients with migraine²¹.

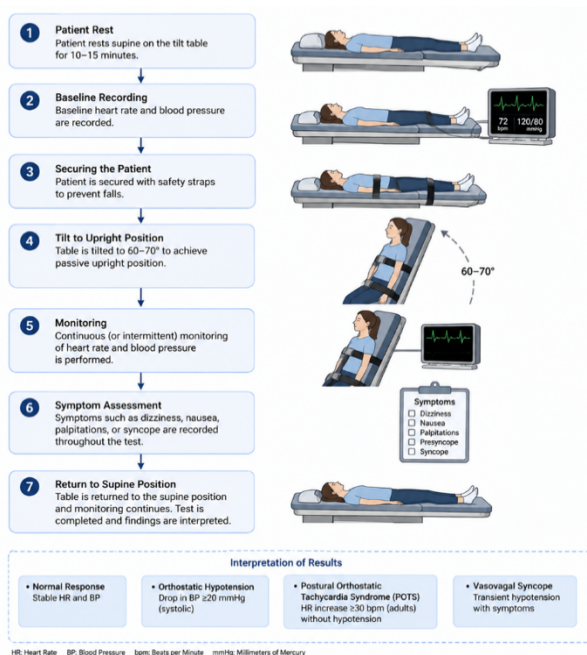


Figure 1: Tilt table testing procedure

6. Clinical Evidence and Implications of Tilt Table Testing in Migraine

Over the past four decades, numerous clinical studies have investigated cardiovascular autonomic responses during TTT in patients with migraine. Despite substantial variation in study design, participant characteristics, migraine phenotype, diagnostic criteria, and testing protocols, the overall evidence suggests an association between migraine and impaired orthostatic cardiovascular regulation²². Recent systematic reviews have further supported the presence of abnormalities in heart rate variability (HRV), cardiovascular reflex function, and responses to orthostatic stress, although methodological heterogeneity continues to limit direct comparison across studies. For example, one study identified abnormalities across several domains of autonomic function while emphasizing the influence of differences in assessment methods and study populations on the reported findings²³.

Our recent clinical study in female patients with migraine demonstrated a significantly reduced acceleration index, impaired systolic blood pressure (SBP) recovery, diminished compensatory

diastolic blood pressure (DBP) responses, and a higher frequency of OH among patients with impaired cardiovascular autonomic function compared with healthy controls²⁴. These findings suggest that impaired cardiovascular sympathetic responses may contribute to abnormal postural adaptation in migraine and support the potential value of TTT for identifying clinically relevant autonomic abnormalities that may not be apparent during conventional autonomic assessment. Similarly, another reported impaired cardiovascular autonomic reactivity during TTT in patients with both episodic and chronic migraine²⁵. Collectively, these findings indicate that cardiovascular autonomic dysfunction is a clinically relevant feature in at least a subset of patients with migraine and that TTT may provide useful complementary information when autonomic impairment is suspected (Table 3).

Table 3. Cardiovascular responses during tilt table testing in healthy individuals and patients with migraine

Parameter	Healthy Individuals	Migraine Patients	Clinical Utility of TTT
HRR	Moderate increase	Exaggerated, blunted or delayed response	Detects abnormal HRR
SBP	Maintained or slight transient decrease	Greater decline or delayed recovery	Detects OH
DBP	Slight increase	Blunted increase or abnormal response	Assesses sympathetic function
Sympathetic activation	Appropriate activation	Dysregulated or impaired	Evaluates sympathetic activity
Parasympathetic withdrawal	Rapid withdrawal	Incomplete or delayed withdrawal	Assesses autonomic balance
Baroreflex sensitivity	Preserved	Reduced	Detects baroreflex impairment
Peripheral vasoconstriction	Adequate	Impaired	Assesses vascular compensation
Orthostatic tolerance	Normal	Frequently reduced	Detects orthostatic intolerance
Recurrent dizziness	Uncommon	Common	Supports autonomic evaluation
Presyncope	Rare	More frequent	Identifies neurally mediated responses
Unexplained syncope	Rare	Increased prevalence	Helps diagnose VVS and OH
Suspected POTS	Rare	More common	Supports POTS diagnosis
Autonomic dysfunction	Generally absent	Likely despite normal routine autonomic tests	Detects overt/subclinical disease

HRR: heart rate response; SBP: systolic blood pressure; DBP: diastolic blood pressure; OH: orthostatic hypotension; POTS: postural orthostatic tachycardia syndrome; VVS: vasovagal syncope

The clinical application of TTT should, however, be guided by the patient's symptoms and clinical context rather than by migraine diagnosis alone. TTT is not warranted as a routine screening investigation for all patients with migraine but may be particularly useful in those with recurrent orthostatic dizziness, presyncope, unexplained syncope, palpitations, exercise intolerance, or persistent orthostatic

symptoms despite an otherwise unremarkable neurological and cardiovascular evaluation. It may also be considered in patients with suspected OH, POTS, neurally mediated syncope, or other unexplained autonomic symptoms²⁶. In selected patients, TTT may provide additional diagnostic information even when CAFT results are inconclusive or normal²⁷.

By objectively assessing integrated cardiovascular responses to controlled orthostatic stress, TTT can complement CAFT, HRV analysis, and clinical assessment. Its findings may help characterize the nature and severity of autonomic impairment and guide individualized management. Management should remain symptom-oriented and may include patient education, optimization of hydration and lifestyle measures, review of potentially contributory medications, and referral for further autonomic or cardiovascular evaluation when appropriate²⁸. Importantly, a normal TTT does not exclude autonomic dysfunction, particularly when abnormalities are intermittent, mild, or not reproduced during a single testing session. Test results should therefore always be interpreted in the context of the clinical history and other relevant investigations.

Although TTT is generally considered safe, appropriate patient selection and clinical supervision are essential. Testing should be avoided or deferred in patients with acute cardiovascular instability, including acute myocardial infarction, unstable angina, severe symptomatic aortic stenosis, or uncontrolled severe hypertension, and in individuals who are unable to tolerate prolonged upright positioning²⁹. Thus, the principal clinical value of TTT in migraine lies in its targeted application to patients with symptoms suggestive of cardiovascular autonomic dysfunction, rather than its routine use as a screening test.

7. Limitations of Tilt Table Testing

Like other autonomic investigations, TTT has inherent diagnostic limitations. False-positive results may occur because prolonged orthostatic stress can provoke vasovagal responses even in otherwise healthy individuals, while false-negative results may arise when autonomic abnormalities are intermittent or are not reproduced during a single testing session. Consequently, TTT findings should not be interpreted in isolation but considered alongside the clinical history, physical examination, and findings from other relevant autonomic investigations. These limitations underscore the importance of appropriate patient selection and standardized testing protocols when using TTT to evaluate cardiovascular autonomic dysfunction in patients with migraine.

8. Future Perspectives

The future role of TTT in migraine will depend substantially on greater methodological standardization and technological refinement. Harmonizing key aspects of TTT protocols, including tilt angle, test duration, patient preparation, and hemodynamic monitoring, would improve the comparability and reproducibility of findings across studies and facilitate the development of clinically meaningful reference standards. Advances in continuous beat-to-beat BP and hemodynamic monitoring may further enhance the detection of transient cardiovascular abnormalities and provide a more comprehensive characterization of autonomic responses to orthostatic stress.

Beyond the laboratory setting, emerging wearable technologies offer an opportunity to extend autonomic assessment into patients' everyday environments. Continuous monitoring of HR, HRV, BP, and related physiological parameters could capture intermittent autonomic abnormalities and symptom fluctuations that may not be evident during a single TTT session. Integrating ambulatory wearable-based monitoring with conventional autonomic testing could

therefore provide a more dynamic and individualized assessment of cardiovascular autonomic function in migraine.

The development of digital biomarkers from wearable devices and mobile health platforms is another promising direction. Such biomarkers could facilitate objective monitoring of autonomic function, identify changes associated with symptom exacerbation, and potentially support earlier recognition of clinically relevant autonomic dysfunction. In parallel, artificial intelligence (AI)-based models integrating clinical, physiological, and autonomic data may enable more refined risk stratification and help identify patients most likely to benefit from formal autonomic evaluation. With appropriate validation, these approaches could ultimately contribute to more personalized diagnostic and management strategies.

Realizing this potential will require large, prospective, multicenter studies incorporating standardized autonomic testing, advanced hemodynamic monitoring, wearable technologies, and longitudinal follow-up. Such studies could help clarify the mechanisms linking autonomic dysfunction and migraine, determine the prognostic and therapeutic relevance of abnormalities detected by TTT, and establish where TTT fits within the broader landscape of autonomic assessment. Ultimately, the future of TTT may lie not in its use as an isolated diagnostic test, but in its integration with continuous physiological monitoring, digital biomarkers, and data-driven approaches to provide a more comprehensive and individualized understanding of autonomic dysfunction in migraine.

9. Conclusion

Tilt table testing provides a standardized, non-invasive assessment of integrated cardiovascular responses to orthostatic stress and may be particularly useful in selected patients with migraine who experience orthostatic intolerance, recurrent dizziness, presyncope, or unexplained syncope. Although available evidence supports its potential clinical value, heterogeneity in study populations, testing protocols, and outcome measures currently limits definitive conclusions regarding its broader application. At present, TTT should therefore be considered a targeted diagnostic investigation in carefully selected patients with migraine and suspected cardiovascular autonomic dysfunction rather than a routine screening tool. Future multicenter studies using standardized protocols, continuous hemodynamic monitoring, and comprehensive autonomic assessment are needed to establish its diagnostic and prognostic utility and to clarify its implications for patient management. Integration of autonomic biomarkers with advanced analytical approaches may further facilitate individualized assessment and improve the identification and characterization of autonomic dysfunction in migraine.

Acknowledgments

The author gratefully acknowledges the researchers whose published work contributed to the development of this review.

Disclosure of AI Use

ChatGPT (OpenAI, GPT-5.5) was used for improving the language of the manuscript and for creating Figure 1.

References

1. Headache Classification Committee of the International Headache Society (IHS). The international classification of headache disorders, 3rd edition. *Cephalalgia* 2018;38(1):1-211. DOI: 10.1177/0333102417738202.
2. Safiri S, Pourfathi H, Eagan A, Mansournia MA, Khodayari MT, Sullman MJM, et al. Global, regional, and national burden of migraine in 204 countries and territories, 1990 to 2019. *Pain*. 2022;163(2):e293-e309. DOI: 10.1097/j.pain.0000000000002275.

3. Goadsby PJ, Holland PR, Martins-Oliveira M, Hoffmann J, Schankin C, Akerman S. Pathophysiology of migraine: a disorder of sensory processing. *Physiol Rev.* 2017;97(2):553-622. DOI: 10.1152/physrev.00034.2015.
4. Charles A. The pathophysiology of migraine: implications for clinical management. *Lancet Neurol.* 2018;17(2):174-82. DOI: 10.1016/S1474-4422(17)30435-0.
5. Shechter A, Stewart WF, Silberstein SD, Lipton RB. Migraine and autonomic nervous system function: a population-based, case-control study. *Neurology.* 2002; 58(3):422-7. DOI: 10.1212/wnl.58.3.422.
6. Miglis MG. Migraine and autonomic dysfunction: which is the horse and which is the jockey? *Curr Pain Headache Rep.* 2018;22(3):19. DOI: 10.1007/s11916-018-0671-y.
7. Benarroch EE. The central autonomic network: functional organization, dysfunction, and perspective. *Mayo Clin Proc.* 1993;68(10):988-1001. DOI: 10.1016/s0025-6196(12)62272-1.
8. 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. *Clin Auton Res.* 2011;21(2):69-72. DOI: 10.1007/s10286-011-0119-5.
9. Jacobsen TN, Morgan BJ, Scherrer U, Vissing SF, Lange RA, Johnson N, et al. Relative contributions of cardiopulmonary and sinoaortic baroreflexes in causing sympathetic activation in the human skeletal muscle circulation during orthostatic stress. *Circ Res.* 1993;73(2): 367-78. DOI: 10.1161/01.res.73.2.367.
10. Kamiya A, Kawada T, Sugimachi M. Systems physiology of the baroreflex during orthostatic stress: from animals to humans. *Front Physiol.* 2014;5:256. DOI: 10.3389/fphys.2014.00256.
11. Freeman R, Abuzinadah AR, Gibbons C, Jones P, Miglis MG, Sinn DI. Orthostatic hypotension: JACC state-of-the-art review. *J Am Coll Cardiol.* 2018;72(11):1294-309. DOI: 10.1016/j.jacc.2018.05.079.
12. Zadourian A, Doherty TA, Swiatkiewicz I, Taub PR. Postural orthostatic tachycardia syndrome: prevalence, pathophysiology, and management. *Drugs.* 2018;78(10):983-94. DOI: 10.1007/s40265-018-0931-5.
13. Edvinsson L. Role of CGRP in migraine. *Handb Exp Pharmacol.* 2019;255:121-30. DOI: 10.1007/164_2018_201.
14. Wattiez AS, Sowers LP, Russo AF. Calcitonin gene-related peptide (CGRP): role in migraine pathophysiology and therapeutic targeting. *Expert Opin Ther Targets.* 2020;24(2):91-100. DOI: 10.1080/14728222.2020.1724285.
15. Thijs RD, Brignole M, Falup-Pecurariu C, Fanciulli A, Freeman R, Guaraldi P, et al. Recommendations for tilt table testing and other provocative cardiovascular autonomic tests in conditions that may cause transient loss of consciousness: consensus statement of the European Federation of Autonomic Societies (EFAS) endorsed by the American Autonomic Society (AAS) and the European Academy of Neurology (EAN). *Clin Auton Res.* 2021;31(3):369-84. DOI: 10.1007/s10286-020-00738-6.
16. Cheshire Jr WP, Goldstein DS. Autonomic uprising: the tilt table test in autonomic medicine. *Clin Auton Res.* 2019;29(2):215-30. DOI: 10.1007/s10286-019-00598-9.
17. Jones PK, Gibbons CH. The role of autonomic testing in syncope. *Auton Neurosci.* 2014;184:40-5. DOI: 10.1016/j.autneu.2014.05.011.
18. Burstein R, Nosedà R, Borsook D. Migraine: Multiple processes, complex pathophysiology. *J Neurosci* 2015;35(17):6619-29. DOI: 10.1523/JNEUROSCI.0373-15.2015.
19. Pogacnik T, Sega S, Pecnik B, Kiauta T. Autonomic function testing in patients with migraine. *Headache.* 1993;33(10):545-50. DOI: 10.1111/j.1526-4610.1993.hed3310545.x.
20. Mueller BR, Campbell MC, Kaplan M, Grant J, Jean J, Vinokur M, et al. Beyond the headache: autonomic reflex dysfunction and heightened sensory sensitivity contribute to orthostatic intolerance in migraine. *Clin Auton Res.* 2026; 36(2):191-203. DOI: 10.1007/s10286-025-01176-y.
21. Thijs RD, Kruit MC, van Buchem MA, Ferrari MD, Launer LJ, van Dijk JG. Syncope in migraine: the population-based CAMERA study. *Neurology.* 2006;66(7):1034-7. DOI: 10.1212/01.wnl.0000204186.43597.66.
22. Fanciulli A, Jordan J, Biaggioni I, Calandra-Buonaura G, Cheshire WP, Cortelli P, et al. Consensus statement on the definition of neurogenic supine hypertension in cardiovascular autonomic failure by the American Autonomic Society (AAS) and the European Federation of Autonomic Societies (EFAS): Endorsed by the European Academy of Neurology (EAN) and the European Society of Hypertension (ESH). *Clin Auton Res.* 2018;28(4):355-62. DOI: 10.1007/s10286-018-0529-8.
23. Pavelić AR, Zeberholzer K, Wöber C. Reconceptualizing autonomic function testing in migraine: a systematic review and meta-analysis. *J Headache Pain.* 2024;25:54. DOI: 10.1186/s10194-024-01758-7.
24. Sen AR, Rezwana I, Sultana S, Alam SKM, Ferdousi S. Cardiovascular responses to tilting in female migraine patients with impaired autonomic reaction. *J Bangladesh Soc Physiol.* 2025;20(2):72-79.
25. Mamontov OV, Babayan L, Amelin AV, Giniatullin R, Kamshilin AA. Autonomously control of cardiovascular reactivity in patients with episodic and chronic forms of migraine. *J Headache Pain.* 2016;17:52. DOI: 10.1186/s10194-016-0645-6.
26. Cheshire WP, Freeman R, Gibbons CH, Cortelli P, Wenning GK, Hilz MJ, et al. Electrodiagnostic assessment of the autonomic nervous system: A consensus statement endorsed by the American Autonomic Society, American Academy of Neurology, and the International Federation of Clinical Neurophysiology. *Clin Neurophysiol.* 2021;132(2):666-82. DOI: 10.1016/j.clinph.2020.11.024.
27. Faraji F, Kinsella LJ, Rutledge JC, Mikulec AA. The comparative usefulness of orthostatic testing and tilt table testing in the evaluation of autonomic-associated dizziness. *Otol Neurotol.* 2011;32(4):654-9. DOI: 10.1097/MAO.0b013e3182117769.
28. Lahrmann H, Cortelli P, Hilz M, Mathias CJ, Struhal W, Tassinari M. EFNS guidelines on the diagnosis and management of orthostatic hypotension. *Eur J Neurol.* 2006;13(9):930-6. DOI: 10.1111/j.1468-1331.2006.01512.x.
29. van Zanten S, Sutton R, Hamrefors V, Fedorowski A, de Lange FJ. Tilt table testing, methodology and practical insights for the clinic. *Clin Physiol Funct Imaging.* 2024;44(2):119-30. DOI: 10.1111/cpf.12859.