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Broken Heart Syndrome (BHS): Investigating Role of Cardiology, Psychology, and Neurology, Mechanism Neuro-Cardiac Axis and Acute Myocardial Infarction

Yash Srivastav[1*], Shivani Singh[1], Kamini Prajapati[1], Stuti Verma[1], Amita Singh[1], Saroj Kumar[1], Brijesh Kumar Pal[1]

[1]D.K.R.R Pharmacy College, Amberpur, Sitapur (Uttar Pradesh), India. 261303

***Corresponding Author E-mail: yashsrv.108@gmail.com**

Abstract:

Broken Heart Syndrome (BHS), which is alternatively referred to as Takotsubo Syndrome (TTS) and stress-induced cardiomyopathy, is a cardiac condition with a temporary impairment of the left ventricle resulting from emotional or physical stress. Despite the absence of any coronary occlusion, BHS manifests similar symptoms as myocardial infarction and acute coronary syndrome. This review focuses on the cardiological, psychological, and neurological aspects of BHS with specific emphasis on the interplay between the two systems through the neuro-cardiac axis. In this regard, the paper considers several important issues regarding the historical background, pathogenesis, mechanism, underlying physiological phenomena, and other causes of this cardiovascular syndrome. These include the autonomic dysregulation, catecholamine toxicity, inflammatory processes, oxidative stress, as well as psychological and neurological factors that contribute to myocardial dysfunction and stress-induced cardiomyopathy. Various advanced cardiovascular imaging modalities and neuroimaging procedures employed in the diagnosis of BHS are examined in detail. From the study, it can be established that excessive sympathetic activity, autonomic imbalance, and brain-heart interaction are critical components in the development of BHS.

Keywords: Broken Heart Syndrome (BHS); Takotsubo Syndrome; Stress Cardiomyopathy; Neuro-Cardiac Axis; Cardiovascular Disease; Catecholamine Toxicity; Autonomic Dysfunction; Myocardial Stunning; Acute Myocardial Infarction; Neurocardiology; Psychological Stress; Brain-Heart Interaction.

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1. INTRODUCTION

Classical approaches to heart diseases were founded on the idea that these ailments have their roots in structural deviations, metabolism, and vessels disorders; however, contemporary research tends to indicate psychological and neurological factors that might contribute to such pathologies[1]. Emotional distress, anxiety, depressive mood, trauma, and other neurological disorders influence heart morbidity through neuro-endocrine and neuro-autonomic mechanisms. Among the list of stress-induced heart diseases, the BHS or TTS becomes an exemplifying case that demonstrates the connection between neurological and heart disorders. It shows how emotional or physical stress may lead to heart disease[2].

With the emergence of neuro-cardiology area of study, it became possible to comprehend the complex interrelations between central nervous and cardiovascular systems, which are often mentioned as a neuro-cardiac axis[3]. Neurological, hormonal, inflammatory, and autonomic mechanisms control the work of the heart muscle; therefore, the dysfunction of these factors causes the heart malfunctions like sympathetic overactivity, catecholamines liberation, myocardium stunning, arrhythmia, or endothelial dysfunction. Thus, BHS can be considered one of the most important interdisciplinary questions for medical professionals such as cardiologists, neurologists, and psychologists.

1.1 Background and Context

BHS, also referred to as TTS, or stress cardiomyopathy, is a reversible heart condition involving temporary impairment of the left ventricle function without obstruction in coronary arteries. It was first reported in Japan in the late 1990s; BHS got its name from the “Takotsubo” trap for capturing octopuses because the heart's left ventricle resembles a balloon shape[4]. BHS mainly occurs among postmenopausal women who have experienced significant emotional or physical stress like sadness, extreme nervousness, trauma, or suffering from a neurological condition. Clinically, it resembles both acute coronary syndrome and myocardial infarction with symptoms like chest pain, difficulty breathing, ECG changes, and increased cardiac markers without coronary artery disease. The current research in neuro-cardiology has focused on the importance of the interaction between the neuro and cardiac systems with autonomic imbalance and excess of catecholamines causing myocardial dysfunction in BHS[5].

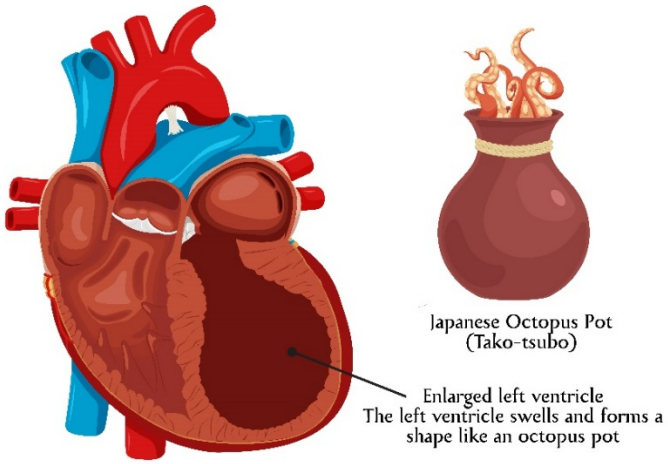

Figure 1: Morphological Appearance of the Left Ventricle in Takotsubo Syndrome Resembling a Japanese Octopus Trap (Takotsubo)

1.2 Objectives of the Review

The primary objectives of this review are:

1. To investigate the role of cardiology, psychology, and neurology in BHS.
2. To analyze how the neuro-cardiac axis works when it comes to stress-induced cardiomyopathy.
3. To establish a connection between BHS and acute myocardial infarction.
4. To assess current research on diagnosis, imaging, biomarkers, and treatment options.
5. To discuss current gaps in research and suggest areas for future study.

1.3 Importance of the Topic

Stress-associated heart disease is on the rise, which makes it vital to have an in-depth understanding of the relationship between stress, neurological mechanisms, and the health of the heart[6]. BHS serves as a clinical example of how a combination of psychological issues and autonomic imbalance can affect myocardium. There is more and more evidence connecting stress, depression, brain deterioration, stroke, and heart diseases, indicating the importance of interdisciplinary approach in these fields, which makes a critical review of BHS necessary[7].

2. BROKEN HEART SYNDROME: CARDIOLOGICAL, PSYCHOLOGICAL, AND NEURO-CARDIAC PERSPECTIVES

BHS is a multi-faceted disease that combines the complicated interplay among the heart, brain, and mind. With the recent breakthroughs in cardiology and neurocardiology, there is now a better understanding of the pathophysiology of stress-induced cardiomyopathy, autonomic dysregulation, and brain-heart interactions. The ensuing sections will discuss the history, cardiological, psychological, diagnostic, and neuro-cardiac facets of BHS[8].

2.1 Historical Evolution and Clinical Concept of Broken Heart Syndrome

BHS has progressed from being categorized as a rare cardiomyopathy to a significant neuro-cardiac syndrome with considerable medical importance. The first literature on BHS described the transient apical ballooning of the left ventricle after emotional stress, whereas subsequent studies have documented other variants, including basal, mid-ventricular, and focal types. The syndrome is often characterized by pseudo-STEMI electrocardiographic features, thereby posing diagnostic difficulties as compared to myocardial infarction.

The syndrome has exhibited diverse presentations using various nomenclature, including Takotsubo cardiomyopathy and stress-induced left ventricular dysfunction. Recent scientific studies have also provided insights into the molecular mechanisms involved in myocardial injury and autonomic dysregulation in stress cardiomyopathy[9].

Though previously thought of as reversible, contemporary reports have shown that BHS may be associated with life-threatening consequences like arrhythmias, cardiogenic shock, thromboembolic complications, ventricular rupture, and sudden cardiac death. Cases of cardiac rupture secondary to stress cardiomyopathy illustrate the potential for lethal outcomes due to BHS.

2.2 Cardiological Perspective of Broken Heart Syndrome

Cardiologic view on BHS centers on heart problems that result from psychological or physical stress. While BHS is usually temporary, it mimics both acute coronary syndrome and myocardial infarction, making the condition difficult to diagnose and treat[10]. The latest studies in cardiology have shed light on stress-related myocardial dysfunction and autonomic disorders linked to this syndrome.

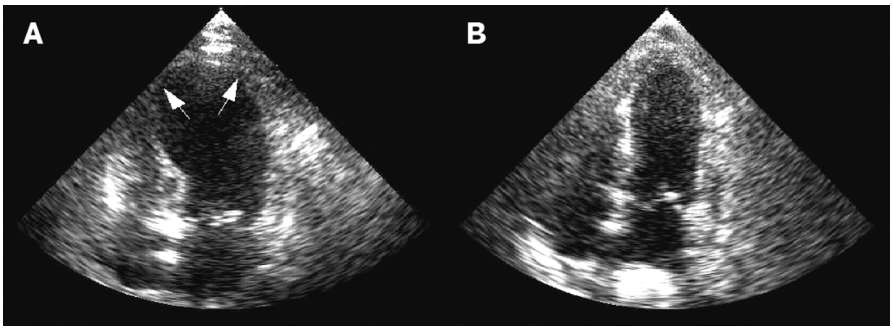

Figure 2: Echocardiographic Appearance of Apical Ballooning in BHS

- **Pathophysiology and Cardiac Dysfunction:** Myocardial stunning is the primary mechanism underlying BHS, which results from the excess secretion of catecholamines due to stress. Sympathetic activation causes an increase in norepinephrine and epinephrine, causing cardiomyocyte toxicity, oxidative stress, and dysfunction of the coronary microvasculature[11]. In contrast to myocardial infarction, BHS does not cause permanent injury but rather temporary cardiac damage, despite the similarity between their clinical presentations.
- **Cardiovascular Imaging and Diagnostic Approaches:** Imaging is critical in distinguishing BHS from acute coronary syndrome. Echocardiography typically shows apical ballooning and wall-motion abnormalities. Cardiac MRI and nuclear imaging techniques can be utilized to assess myocardial anatomy and physiology. Elevated levels of troponin, BNP, cytokines, and catecholamines are frequently seen in laboratory investigations[12].
- **Relationship with Acute Myocardial Infarction:** BHS manifests symptoms similar to those seen in acute myocardial infarction, such as chest pain, ECG changes, dyspnea, and increased cardiac enzymes; yet, significant coronary obstruction is generally not observed. While there are similarities between the two conditions in terms of clinical presentation, the treatment modalities for each condition differ vastly, with revascularization being necessary for AMI but not BHS.

2.3 Psychological Dimension of Broken Heart Syndrome

The psychological component of BHS is vital in the examination of the connection between psychological stress and heart problems. The negative impacts of psychological factors, including anxiety, depression, trauma, bereavement, and prolonged stress, have a major impact on the body's physiological responses, resulting in dysfunction of the heart muscle and increasing the risk of cardiovascular diseases[13]. Psychological aspects not only cause BHS but also play a key role in healing from the disorder.

- BHS has been recognized as being among the strongest inducers of psychological stress. Psychological stress involves situations that include emotional shock, grief, anxiety, disputes, monetary issues, and fear.
- Psychological stress causes heart diseases by causing irregularities in the cardiovascular system due to the influence on the nervous system and inflammation.
- Individuals with BHS frequently suffer from psychological stress, lack of emotional resilience, fear, uncertainty, and post-traumatic symptoms during the course of the disease.
- The presence of depression and anxiety is considered an important factor contributing to heart diseases and is correlated with increased morbidity from cardiovascular conditions.
- Emotional stress plays a part in causing chronic sympathetic nervous activity, impairment of endothelial function, and inflammation[14].
- Cognitive Behavioral Therapy (CBT) is considered a promising intervention strategy that can help mitigate psychological stress in patients.
- Activation of the hypothalamus-pituitary-adrenal axis due to stress causes the secretion of cortisol, sympathetic hyperactivity, and increased production of catecholamines, which could adversely affect blood vessels and the heart muscle.
- Amygdala and limbic structures contribute significantly to emotion regulation and cardiac control. Overstimulation of these structures can lead to an autonomic imbalance that makes people prone to stress cardiomyopathy[15].

3. NEUROLOGICAL PERSPECTIVE AND THE NEURO-CARDIAC AXIS

The neurologic view on BHS emphasizes the intricate relationship between the brain and the cardiovascular system via the neuro-cardiac axis. Emerging discoveries in the field of neurocardiology have proven that psychological stress, autonomic disturbances, and nervous conditions play an important role in regulating heart physiology using neural, hormonal, and inflammatory mechanisms. Disturbances of such mechanisms result in the development of myocardial dysfunction and stress cardiomyopathy[16].

3.1 Neuro-Cardiac Axis and Autonomic Dysfunction

The neuro-cardiac axis involves the bi-directional interconnection between the brain and heart through autonomic, hormonal, and inflammatory processes. The amygdala, hypothalamus, insular cortex, and the brainstem autonomic centers are among several structures that mediate this connection in terms of cardiovascular control. Sympathetic hyperactivity and lack of parasympathetic influence are considered critical components of BHS as they facilitate myocardial stunning, cardiac arrhythmias, and blood vessel problems[17].

The resulting autonomic imbalance associated with emotional and physiological stress causes an increase in catecholamine levels and the impairment of cardiovascular control. Research into neurocardiology also suggests that exercise therapy and autonomic balance improvement can lead to the betterment of cardiovascular conditions caused by psychological stress[18].

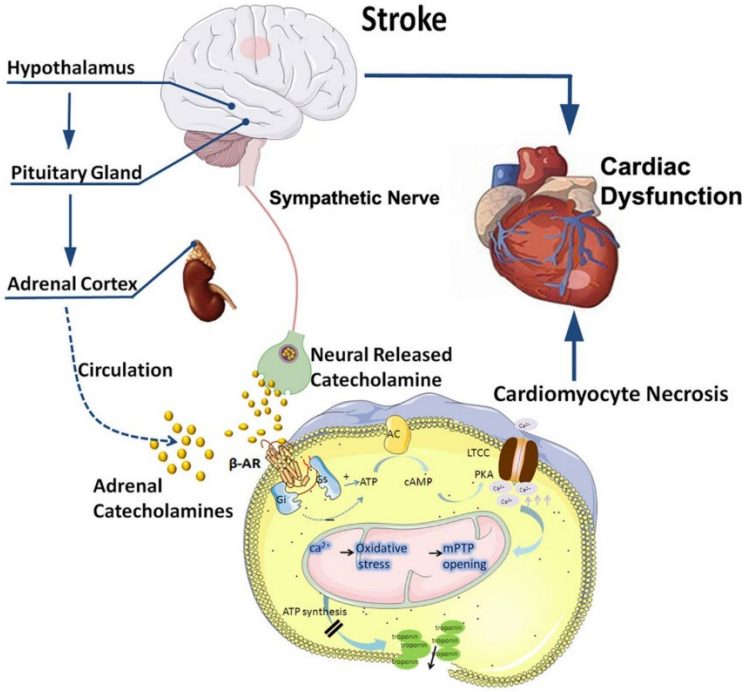

Figure 3: Mechanism of the Neuro-Cardiac Axis and Catecholamine-Mediated Cardiac Dysfunction in Broken Heart Syndrome

The latest research on the subject highlights the crucial role of brain-heart interaction in the emergence of stress cardiomyopathy syndrome. Abnormal functioning of emotional centers can exacerbate the effects of stress and raise the risk of cardiovascular complications. In addition, chronic autonomic imbalance may lead to vascular damage and inflammation, thereby interfering with myocardial recovery[19].

3.2 Neurological Disorders, Neuro-Imaging, and Cardiac Injury

Neurological disorders like stroke, traumatic brain injury, epilepsy, and neurodegenerative conditions are commonly linked to neurogenic heart injury and autonomic dysfunction. Cardiovascular complications after strokes usually arise due to sympathetic overactivity and toxicity with catecholamines, but traumatic brain injury can also lead to cardiovascular disturbance due to the brain-heart connection.

Neurological disorders could affect autonomic control, inflammation, and cardiovascular stability, resulting in myocardial dysfunction and arrhythmias[20]. Additionally, chronic stress arising from neurological disorders could further compromise the functions of the endothelium and cardiac output, as well as impair autonomic flexibility in vulnerable people.

Brain heart syndrome patients exhibit functional problems with the brain structures responsible for emotions and autonomic function according to neuro-imaging. Neuro-imaging is now increasingly applied in the assessment of autonomic functions, stress reactions, and neuro-cardiac interaction. These techniques could be useful in the identification of therapeutic targets for the treatment of stress cardiomyopathy[21].

4. MOLECULAR AND CELLULAR MECHANISMS OF BROKEN HEART SYNDROME

The molecular and cellular mechanisms involved in BHS include an intricate interplay between neurohormonal activation, autonomic dysfunction, inflammation, oxidative stress, and

myocardial dysfunction[22]. Emotional or physiological stress leads to activation of the sympathetic nervous system and the hypothalamus-pituitary-adrenal axis, causing the overproduction of catecholamines that affect myocardial physiology. The biochemical changes are important contributors to ventricular dysfunction and stress cardiomyopathy[23].

Catecholamine toxicity has been identified as one of the major mechanisms behind myocardial injury in BHS[24]. High concentrations of norepinephrine and epinephrine adversely affect cardiac cells through calcium overload, mitochondrial dysfunction, oxidative stress, and cell death. Overactivation of the sympathetic nervous system might cause impaired coronary microcirculation and reduced contractility, contributing to myocardial stunning and temporary ventricular dysfunction[25].

Other mechanisms implicated in the pathophysiology of BHS include inflammation and oxidative stress. Stress-induced pro-inflammatory cytokines lead to impaired myocardial performance through their effect on endothelial cell function, cellular signaling, and myocardial metabolism[26]. Chronic oxidative stress can also impair the integrity of blood vessels and contribute to autonomic dysfunction. These mechanisms may account for the relationship between stress cardiomyopathy and other conditions including infection, COVID-19, and chronic inflammatory diseases[27].

Interaction between neurotransmitters, stress hormones, and the autonomic nervous system is critical for regulation of cardiac muscle contraction and vascular constriction. Imbalance of neurohormonal pathways leads to arrhythmia, endothelial dysfunction, vasospasm, and poor myocardial reperfusion[28]. New studies have stressed the significance of neural regulation of the cardiovascular system and heart innervation for proper cardiovascular response during periods of stress[29].

Progress in molecular cardiology and neurocardiology has provided important information on the biochemical processes that underlie BHS. Therapeutic modalities related to modulation of the autonomic nervous system, antioxidative treatment, and neurocardiological approach can facilitate myocardial reperfusion and alleviate cardiovascular effects of stress cardiomyopathy[30]. It will be helpful to identify the exact molecular markers for effective therapy of stress cardiomyopathy.

Table 1: Major Molecular and Cellular Mechanisms Involved in Broken Heart Syndrome

Molecular Mechanism	**Major Effects on the Heart**	**Clinical Significance in BHS**
Catecholamine Excess	Myocardial stunning, calcium overload, oxidative stress	Transient ventricular dysfunction
Inflammation	Endothelial injury and altered cellular signaling	Cardiovascular instability
Oxidative Stress	Mitochondrial dysfunction and tissue damage	Impaired myocardial recovery

Autonomic Dysregulation	Sympathetic overactivity and arrhythmias	Neuro-cardiac imbalance
Neurohormonal Activation	Altered vascular tone and myocardial function	Stress-induced cardiomyopathy

5. DISCUSSION

BHS is a complex disorder in which multiple organ systems including the heart, nervous system, and psyche interact to produce the condition. As noted in the review, the involvement of emotion, autonomic dysfunction, inflammation, and the brain-heart connection are all key factors contributing to stress-induced cardiomyopathy[31]. It is increasingly clear from the fields of cardiology, psychology, and neurocardiology that BHS should be viewed beyond a cardiovascular disease and recognized as a neuro-cardiac disorder[32].

5.1 Interpretation and Analysis of the Findings

From the reviewed studies, one can conclude that excessive stimulation of the sympathetic nervous system and the release of catecholamines are the main factors responsible for myocardial abnormalities in BHS[33]. Psychological and physiological stress causes a neuroendocrine response to occur, affecting the function of the heart through autonomic dysfunction, oxidative stress, and inflammation. The scientific literature proves the existence of a link between psychological conditions, brain damage, and heart failure. Therefore, the notion of the neuro-cardiac connection plays an important role in stress-induced heart disease. However, BHS is typically regarded as a reversible disorder.

Table 2: Comparative Analysis of Recent Studies on Broken Heart Syndrome, Neuro-Cardiac Axis, and Cardiovascular Mechanisms

Reference	**Study Focus**	**Major Findings**	**Relevance to Present Review**
Sukma & Balatif (2022)[34]	Broken Heart Syndrome and stress-induced heart failure	Reported the progression of emotional stress toward cardiac dysfunction and heart failure	Supports the psychological and cardiological basis of BHS
Talkington et al. (2025)[35]	Neurological sequelae and diagnostic imaging	Highlighted neuro-inflammatory and neuro-imaging mechanisms associated with cardiovascular complications	Supports neuro-cardiac interaction and imaging perspectives
Therkleson & Stronach (2015)[36]	Clinical case study of BHS	Demonstrated typical clinical presentation and	Reinforces clinical understanding of stress cardiomyopathy

		stress-related cardiac dysfunction	
Valenza et al. (2025)[37]	Brain–heart axis mechanisms	Explained neural, biochemical, and autonomic pathways linking brain and heart function	Strongly supports the neuro-cardiac axis discussion
Yang et al. (2024)[38]	Advanced cardiovascular research models	Discussed innovative 3D cardiovascular models for studying cardiac diseases	Supports future research and advanced cardiovascular investigations

5.2 Implications and Significance

The multidisciplinary approach associated with BHS has significant implications for contemporary clinical medicine and cardiovascular research. The syndrome brings to the forefront the issue of how cardiologists, psychologists, and neurologists must collaborate in the evaluation and management of patients. Increased awareness that psychological stress and dysautonomia play an essential role in the development of heart disease might facilitate earlier diagnosis and patient counseling, as well as enhance the effectiveness of treatment options available[39].

5.3 Research Gap and Limitations

Although considerable progress has been made regarding neurocardiology and stress cardiomyopathy in recent years, there are a few gaps in the existing scientific knowledge. There is no standardized biomarker that could reliably diagnose and help in the risk stratification of BHS patients[40]. The exact neurobiological link between the emotional state and the condition of the myocardium is unknown. Moreover, most studies currently performed address the immediate clinical symptoms; only a small number of publications discuss the long-term effects from the perspective of neurology and psychology.

5.4 Future Research Directions

Areas for further research would be the identification of neuro-cardiac biomarkers and understanding the molecular neurophysiology behind BHS. This can be achieved through advanced neuro-imaging, autonomic testing, and precision medicine techniques. The longitudinal effects of recurrence of BHS, psychological aspects associated with the disease, and long-term cardiovascular issues should also be investigated. In addition, the use of artificial intelligence and machine learning along with other multidisciplinary approaches may help in achieving better results in the prediction, prevention, and management of stress cardiomyopathy.

6. CONCLUSION

The BHS is an intricate case of a stress-induced cardiomyopathy that exhibits many interactions among the cardiovascular, nervous, and psychological systems via the neuro-cardiac pathway.

Scientific studies show the importance of toxic effects of catecholamines, autonomic dysregulation, inflammatory process, and brain-heart interaction during the pathogenesis of this condition. BHS bears resemblance to myocardial infarction which presents a diagnostic and therapeutic dilemma in the management of this syndrome. Thus, it necessitates an interdisciplinary intervention combining cardiology, neurology, psychology, and stress and autonomic management. Further research is necessary to increase knowledge in neuro-cardiac physiology and identification of biomarkers.

REFERENCES

1. Alataş, Ö. D., & Biteker, M. (2020). Happy heart syndrome mimicking acute pulmonary embolism and acute coronary syndrome. Anatolian Journal of Cardiology, 23(4), 237.
2. Anđelić, S., & Šijački, A. (2015). From broken heart syndrome to pseudo-STEMI electrocardiographic findings. Halo 194, 21(1), 23-34.
3. Atmanto, Y. K. A. A., Wibawa, S. Y., & Rauf, D. E. (2023). Laboratory Aspect of Broken Heart Syndrome. INDONESIAN JOURNAL OF CLINICAL PATHOLOGY AND MEDICAL LABORATORY, 29(2), 194-199.
4. Bhalerao, S. K. (2025). Broken Heart Syndrome. A and V Pub International Journal of Nursing and Medical Research, 4(1), 58-61.
5. Bhatti, M. Q., Chapman, R., Kulik, T., Di Napoli, M., Bhatia, K., Elkind, M. S., ... & Divani, A. A. (2025). On neurocardiology updates: an interdisciplinary field at the intersection of neurology and cardiology. Current neurology and neuroscience reports, 25(1), 59.
6. Braun, J., Patel, M., Kameneva, T., Keatch, C., Lambert, G., & Lambert, E. (2023). Central stress pathways in the development of cardiovascular disease.
7. Case, A. T. (2015). Broken Heart Syndrome.
8. Chen, L., Luo, S., Feng, M., Chandra, A., Baizabal-Carvallo, J. F., Mofatteh, M., ... & Liao, X. (2025). Decoding the link between ischemic stroke and myocardial infarction: a pathophysiological and clinical perspective. European Journal of Medical Research, 30(1), 984.
9. Chin, S. H., & Ng, G. A. (2023). The neuro-cardiac axis in arrhythmogenesis: Role and impact of autonomic modulation. In Heart Rate and Rhythm: Molecular Basis, Pharmacological Modulation and Clinical Implications (pp. 187-224). Cham: Springer International Publishing.
10. Chin, S. H., & Ng, G. A. (2023). The Neuro-cardiac Axis in Arrhythmogenesis: Role and Impact. Heart Rate and Rhythm, 187.
11. Fahim, M. A., Yao, Y., Tipparaju, S. M., & Xuan, W. (2025). The heart-brain crosstalk in age related cardiovascular and neurodegenerative diseases. Fluids and Barriers of the CNS, 22(1), 89.
12. Fan, Z. X., Liang, N., Lu, J. F., & Liu, G. Z. (2025). Neurocardiology-from basics to clinic. World Journal of Cardiology, 17(11), 109941.
13. Gianaros, P. J., & Jennings, J. R. (2018). Host in the machine: A neurobiological perspective on psychological stress and cardiovascular disease. American Psychologist, 73(8), 1031.

14. Holmann, F. C. (2020). The Experiences of Women Who Endured the Broken Heart Syndrome (Doctoral dissertation, Alliant International University).

15. Hosny, N. K., Shousha, N. M., & Ali, A. M. (2025). Beyond the Physical: Meaning of Life and Stress Coping Strategies with Broken Heart Syndrome.

16. Ion, A., Stafie, C., Mitu, O., Ciobanu, C. E., Halitchi, D. I., Costache, A. D., ... & Costache, I. I. (2021). Biomarkers utility: at the borderline between cardiology and neurology. Journal of Cardiovascular Development and Disease, 8(11), 139.

17. Kerna, N. A., Holets, H. M., Hafid, A., Pruitt, K. D., Carsrud, N. D. V., Nwokorie, U., ... & Anderson, I. I. (2021). J, Rashid S."Broken-Heart Syndrome (BHS)—Real or Imagined? Also Known as Takotsubo Syndrome (TS), Tako-Tsubo-Like Left Ventricular Dysfunction, and Takotsubo Cardiomyopathy (TCM). Investigation and Review; the Diagnosis and Treatment of Its Varied Manifestations". EC Cardiology, 8, 60-74.

18. Kibria, F., Bragina, O. A., Trofimov, A. O., & Bragin, D. (2025). Bidirectional Interplay Between Traumatic Brain Injury and Cardiovascular Dysfunction in Athletes. Journal of Clinical Medicine, 14(21), 7712.

19. Li, J., Bai, J., Liu, G., Zhu, Z., & Cao, C. (2025). Exercise intervention in autonomic function, immunity, and cardiovascular health: a precision medicine approach. Journal of Cardiovascular Development and Disease, 12(7), 247.

20. Liu, Y., Yang, H., Xiong, J., Wei, Y., Yang, C., Zheng, Q., & Liang, F. (2025). Brain-heart axis: Neurostimulation techniques in ischemic heart disease. International Journal of Molecular Medicine, 56(4), 148.

21. Manolis, A. S., Manolis, A. A., Manolis, T. A., & Melita, H. (2021). COVID-19 and acute myocardial injury and infarction: related mechanisms and emerging challenges. Journal of Cardiovascular Pharmacology and Therapeutics, 26(5), 399-414.

22. Manolis, T. A., Manolis, A. A., & Manolis, A. S. (2024). Cognitive Behavioral Therapy in Cardiovascular Disease. Current Vascular Pharmacology.

23. Manolis, T. A., Manolis, A. A., & Manolis, A. S. (2024). Emotional stress in cardiac and vascular diseases. Current Vascular Pharmacology.

24. Meadows, J. L., Shah, S., Burg, M. M., Pfau, S., & Soufer, R. (2020). The Foundational Role of Cardiovascular Imaging in the Characterization of Mental Stress–Induced Myocardial Ischemia in Patients with Coronary Artery Disease. Current cardiology reports, 22(12), 162.

25. Podolec, J., Kleczyński, P., Piechocki, M., Okarski, M., Lizończyk, K., Szkodoń, K., ... & Kabłak-Ziembicka, A. (2024). Depression in cardiac patients is a major cardiovascular event risk factor: a 12-month observational study. Journal of Clinical Medicine, 13(22), 6911.

26. Roberts, D. J., & Zygun, D. A. (2016). Non-neurological complications of acquired brain injury. Oxford Textbook of Neurocritical Care, 6, 354.

27. Romeo, F. J. (2024). When the heart truly breaks: Cardiac rupture in broken heart syndrome. International Journal of Cardiology, 414.

28. Ruiz, S. A. (2018). BUT DID YOU DIE? BROKEN HEART SYNDROME: A STUDY OF STRESS AND THE CARDIOVASCULAR SYSTEM.

29. Salem, J. B., Iacovoni, J. S., Calise, D., Arvanitis, D. N., & Beaudry, F. (2022). Proteomics reveals long-term alterations in signaling and metabolic pathways

following both myocardial infarction and chemically induced denervation. Neurochemical research, 47(8), 2416-2430.

30. Sethi, Y., Murli, H., Kaiwan, O., Vora, V., Agarwal, P., Chopra, H., ... & Cavalu, S. (2022). Broken heart syndrome: evolving molecular mechanisms and principles of management. Journal of clinical medicine, 12(1), 125.

31. Singh, R. B., Torshin, I., Albawareed, O., Verma, N. S., De Meester, F., Wichansawakun, S., & Mojto, V. (2019). Identification of new brain targets by neuro-imaging technology for treatment of neurocardiac disorders. Biomed J Sci & Tech Res, 14(1).

32. Singh, R. B., Torshin, V. I., Al-Bawareed, O. A., Narsingh, V., Mojto, V., De Meester, F., ... & Kartikey, K. (2018). Physiological imaging: A new approach for assessment of neuropsychiatric and cardiovascular diseases. World Heart Journal, 10(4), 321-336.

33. Sposato, L. A., Hilz, M. J., Aspberg, S., Murthy, S. B., Bahit, M. C., Hsieh, C. Y., ... & World Stroke Organisation Brain & Heart Task Force. (2020). Post-stroke cardiovascular complications and neurogenic cardiac injury: JACC state-of-the-art review. Journal of the american college of cardiology, 76(23), 2768-2785.

34. Sukma, A. A. M., & Balatif, R. (2022). Broken heart syndrome: Berawal dari stres menuju gagal jantung. SCRIPTA SCORE Scientific Medical Journal, 3(2), 132-137.

35. Talkington, G. M., Kolluru, P., Gressett, T. E., Ismael, S., Meenakshi, U., Acquarone, M., ... & Bix, G. J. (2025). Neurological sequelae of long COVID: a comprehensive review of diagnostic imaging, underlying mechanisms, and potential therapeutics. Frontiers in Neurology, 15, 1465787.

36. Therkleson, T., & Stronach, S. (2015). Broken heart syndrome: a typical case. Journal of Holistic Nursing, 33(4), 345-350.

37. Valenza, G., Matić, Z., & Catrambone, V. (2025). The brain–heart axis: integrative cooperation of neural, mechanical and biochemical pathways. Nature Reviews Cardiology, 22(8), 537-550.

38. Yang, Y., Yang, H., Kiskin, F. N., & Zhang, J. Z. (2024). The new era of cardiovascular research: Revolutionizing cardiovascular research with 3D models in a dish. Medical review, 4(1), 68-85.

39. Yin, X., Cai, D., Song, Z., & Song, C. (2025). Nourishment of nerves and innervation: A novel approach for the treatment of myocardial infarction. Cardiology, 150(6), 678-700.

40. Zghyer, F., Botheju, W. S. P., Kiss, J. E., Michos, E. D., Corretti, M. C., Mukherjee, M., & Hays, A. G. (2022). Cardiovascular imaging in stress cardiomyopathy (takotsubo syndrome). Frontiers in Cardiovascular Medicine, 8, 799031.