www.ijarp.com ISSN 2456-9992 Page: 01-11

Review Article

International Journal Advanced Research Publications

Volume: 02
Issue: 10

THE MANAS–HRIDAYA AXIS IN STRESS-RELATED CARDIOVASCULAR PATHOPHYSIOLOGY: AN INTEGRATIVE REVIEW OF ATICHINTA AND RASAVAHA SROTAS DUSHTI

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Article Received: 07 September 2026, Article Revised: 27 September 2026, Published on: 17 October 2026

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Doi: https://doi-doi.org/101555/ijarp.3159

ABSTRACT

Background: Psychological stress is associated with cardiovascular risk through autonomic, neuroendocrine, inflammatory, vascular, myocardial, and behavioral pathways. Ayurveda describes *Atichinta* (excessive worry or mental preoccupation) among the factors associated with *Rasavaha Srotas Dushti* and identifies *Hridaya* as a principal root of *Rasavaha Srotas*.

Objective: To critically synthesize classical Ayurvedic descriptions of *Atichinta, Rasavaha Srotas*, and *Hridaya* and examine their possible conceptual relationships with contemporary mechanisms of stress-related cardiovascular disease, while clearly distinguishing textual evidence from modern mechanistic inference.

Methods: A narrative review was undertaken using classical Ayurvedic sources, including Charaka Samhita, Sushruta Samhita, and Sharangadhara Samhita, together with selected biomedical literature identified through PubMed and major cardiovascular literature. The modern evidence synthesis focused on psychological stress, autonomic and hypothalamic–pituitary–adrenal responses, endothelial dysfunction, inflammation, coronary microvascular dysfunction, mental stress-induced myocardial ischemia, and Takotsubo syndrome. The literature was updated through September 2026. No formal systematic-review risk-of-bias assessment or quantitative meta-analysis was undertaken.

Discussion: Classical Ayurveda places *Atichinta* within the etiological framework of *Rasavaha Srotas Dushti* and assigns *Hridaya* an important role in *Rasa*, circulation, and

Manas-related concepts. Contemporary evidence supports plausible associations between psychological stress and cardiovascular dysfunction through sympathetic and hypothalamic–pituitary–adrenal activation, altered vascular function, inflammation, and stress-related myocardial effects. These parallels may support hypothesis generation, but *Sanga, Rasa Dushti*, and *Vyana Vayu* should not be presented as direct anatomical or molecular equivalents of microvascular obstruction, inflammation, or autonomic physiology.

Conclusion: The proposed *Manas–Hridaya* Axis should be regarded as a hypothesis-generating integrative framework rather than an established pathophysiological model. Future research should operationalize Ayurvedic constructs independently and prospectively test their relationships with validated psychological, autonomic, vascular, and cardiovascular outcomes.

KEYWORDS: *Atichinta, Rasavaha Srotas*, *Hridaya*, *Manas*, Psychological stress, Cardiovascular disease.

1. INTRODUCTION

Psychological stress is increasingly recognized as one component of cardiovascular risk. Prospective evidence indicates that chronic work-related and private-life stress is associated with a modest increase in coronary heart disease risk, while acute emotional stress may trigger cardiovascular events in susceptible individuals. Contemporary research implicates autonomic activation, hypothalamic–pituitary–adrenal (HPA) axis responses, haemodynamic changes, vascular dysfunction, inflammation, and behavioral pathways. Stress is therefore better understood as part of a multifactorial cardiovascular risk network rather than as a single direct cause of cardiovascular disease [4–7].

Ayurveda approaches disease through a different conceptual vocabulary. *Rasavaha Srotas* is described in relation to the transport and nourishment of *Rasa Dhatu*, with *Hridaya* and *Dhamani* identified as principal roots or structural bases in classical descriptions. Charaka specifically lists *Atichinta* among the factors associated with *Rasavaha Srotas Dushti* [1]. *Hridaya* also occupies an important position in Ayurvedic descriptions of *Rasa, Ojas*, consciousness, and *Manas*-related concepts [1–3,9].

The present review does not assume that *Rasavaha Srotas* is anatomically identical to the modern cardiovascular system. Ayurvedic *Srotas* are functional and conceptual entities embedded within a different physiological taxonomy. Modern cardiovascular anatomy can

therefore be used as a comparative framework, but not as a definitive translation of *Rasavaha Srotas*.

The purpose of this narrative review is threefold: (1) to clarify the classical description of *Rasavaha Srotas* and *Atichinta* (2) to summarize established biomedical mechanisms linking psychological stress with cardiovascular dysfunction and (3) to propose a cautious *Manas–Hridaya* framework that generates testable research questions without presenting conceptual analogy as scientific validation.

2. METHODS

This article is a narrative review and was not designed as a systematic review or meta-analysis. Classical Ayurvedic concepts were examined primarily from Charaka Samhita, Sushruta Samhita, and Sharangadhara Samhita, with attention to *Rasavaha Srotas, Hridaya, Atichinta/Chinta, Srotodushti, Rasa Dhatu, Manas,* and *Vyana Vayu.*

For the contemporary evidence synthesis, targeted PubMed searches were conducted using combinations of terms related to psychological stress and cardiovascular disease, autonomic nervous system dysfunction, HPA-axis activation, endothelial dysfunction, inflammation, coronary microvascular dysfunction, mental stress-induced myocardial ischemia, heart rate variability, and Takotsubo syndrome. Selected cardiovascular reviews, consensus statements, and recent primary studies were also considered. The modern literature was updated through September 2026.

Eligible literature included original human studies, systematic reviews, meta-analyses, consensus statements, and relevant mechanistic studies. Classical texts and interpretive Ayurvedic literature were used for the Ayurvedic framework. Literature unrelated to psychological stress or cardiovascular mechanisms was not prioritized.

Because this was a narrative review, no protocol registration, formal risk-of-bias assessment, or quantitative meta-analysis was undertaken. The integrative mapping presented in this article is interpretive. Statements placing an Ayurvedic construct alongside a modern physiological process are presented as conceptual hypotheses unless direct empirical evidence exists.

3. Classical Ayurvedic Framework

3.1 Rasavaha Srotas: Classical Description and Functional Scope

Charaka identifies *Hridaya* and the *Dasha Dhamani* as the *Mula* of *Rasavaha Srotas*, while Sushruta provides a related description of its roots [1,2]. *Rasa Dhatu* is associated with

nourishment and distribution throughout the body. These descriptions support discussion of transport, nourishment, and systemic tissue maintenance within the Ayurvedic framework.

It is tempting to equate *Rasavaha Srotas* directly with the modern cardiovascular system. Such a one-to-one identification, however, exceeds the evidence. Ayurvedic *Srotas* are functional and conceptual entities, and their boundaries do not necessarily correspond to modern anatomical compartments. Cardiovascular anatomy can therefore serve as a comparative framework, but not as a definitive translation of *Rasavaha Srotas.*

Hridaya occupies a particularly important position in classical literature because it is associated with *Rasa, Ojas*, consciousness, and *Manas*-related concepts, while *Dhamani* are discussed in relation to circulation [1–3,9]. These overlapping functions provide a legitimate basis for exploring a mind–heart relationship within Ayurvedic theory.

3.2 Atichinta as a Nidana of Rasavaha Srotas Dushti

Atichinta may be translated as excessive worry, excessive mental preoccupation, or persistent overthinking depending on context. In Charaka's discussion of *Rasavaha Srotas*, *Atichinta* is explicitly listed among the factors that can vitiate the Srotas [1]. The classical verse is located in *Vimanasthana* chapter 5 *shloka* 13 and is an important textual basis for examining the relationship between a mental factor and a bodily channel.

This classical statement should be interpreted precisely. It supports the proposition that *Atichinta* is a *Nidana* associated with *Rasavaha Srotas Dushti*; it does not establish that psychological stress alone causes a modern cardiovascular disease, nor does it provide a molecular mechanism equivalent to sympathetic activation, endothelial inflammation, or coronary microvascular dysfunction.

Classical descriptions of *Rasavaha Srotas Dushti* include symptoms such as *Aruchi, Gaurava*, and *Tandra*, among other disturbances [1]. These descriptions should be preserved in their Ayurvedic context rather than retroactively relabeled as specific modern cardiovascular diagnoses.

3.3 Srotodushti and the Concept of Sanga

Sanga is one of the classical patterns used to describe abnormal channel function. In an integrative discussion, *Sanga* may be compared conceptually with impaired flow or functional disturbance. However, *Sanga* should not be equated automatically with endothelial dysfunction, microvascular obstruction, thrombosis, or atherosclerotic plaque. These modern entities have specific anatomical, cellular, and diagnostic definitions.

Similarly, terms such as *Siragranthi* should not be presented as proven Ayurvedic descriptions of atheromatous plaques unless the textual interpretation and empirical evidence

explicitly support that correspondence. Modern atherosclerosis involves lipid accumulation, inflammation, plaque formation, remodeling, and variable thrombogenicity; the analogy with Ayurvedic vascular pathology remains interpretive.

4. Contemporary Evidence: Stress and Cardiovascular Disease

4.1 Autonomic and Neuroendocrine Pathways

Psychological stress activates the sympathetic nervous system and the HPA axis. Repeated or sustained activation may contribute to altered heart rate, blood pressure regulation, vascular tone, metabolic function, and autonomic balance [4–7]. Reduced heart rate variability is commonly studied as a marker of altered autonomic regulation, although HRV is not a specific diagnostic marker of psychological stress or cardiovascular disease [15,16].

These mechanisms provide a plausible modern context for discussing the Ayurvedic importance of *Vata* and *Vyana Vayu*. However, *Vyana Vayu* is an Ayurvedic functional construct and should not be described as identical to the autonomic nervous system. Contemporary Ayurvedic interpretations have proposed relationships between *Vyana Vayu* and cardiovascular physiology, but these remain interpretive models rather than established anatomical equivalences [9,10].

4.2 Endothelial Dysfunction and Inflammation

Chronic psychological stress is associated with vascular and inflammatory changes that may adversely affect cardiovascular health. Proposed mechanisms include altered sympathetic signaling, oxidative stress, impaired nitric-oxide-mediated vasodilation, inflammatory signaling, and changes in leukocyte and platelet activity [7,14]. The magnitude and clinical relevance of these pathways vary among individuals and depend on the type, duration, and context of stress.

The Ayurvedic concepts of *Agni, Rasa Dushti*, and *Srotodushti* may be discussed alongside these mechanisms as a conceptual analogy. However, *Agni* cannot currently be measured as a direct equivalent of mitochondrial function, metabolic rate, cortisol activity, or inflammatory signaling. Likewise, *Ama* should not be described as synonymous with cytokines, reactive oxygen species, or metabolic waste without direct evidence.

4.3 Coronary Microvascular Dysfunction and Mental Stress-Induced Ischemia

Mental stress can provoke myocardial ischemia in some patients with coronary artery disease and is increasingly recognized as a clinically relevant phenotype. Mechanisms may include altered coronary vasomotor responses, microvascular dysfunction, haemodynamic demand, and neurohumoral activation [17–19]. Importantly, mental stress-induced ischemia is a

specific cardiovascular phenomenon requiring appropriate clinical testing; it cannot be inferred solely from symptoms such as chest heaviness.

Coronary microvascular dysfunction is recognized in patients with angina or ischemia without obstructive epicardial coronary disease. Assessment may involve coronary flow reserve, invasive or non-invasive vasomotor testing, and appropriate clinical evaluation [11–13]. These measurements offer potential endpoints for future Ayurvedic research on *Sanga*, but no current evidence establishes that Sanga itself is a biomarker of microvascular disease.

4.4 Takotsubo Syndrome

Takotsubo syndrome provides a clinically recognized example of a relationship between acute emotional or physical stress and transient cardiac dysfunction. Catecholamine-related mechanisms, coronary microvascular abnormalities, and myocardial effects are implicated in its pathophysiology [20–22]. Nevertheless, Takotsubo syndrome is a specific clinical diagnosis and should not be presented as a direct modern equivalent of an Ayurvedic *Hridaya Roga* or *Atichinta*-induced *Srotodushti*.

5. Proposed Manas–Hridaya Axis

On the basis of the classical and modern literature, a cautious conceptual model can be proposed. The model is intended to generate testable hypotheses rather than to claim that Ayurvedic and biomedical categories are identical.

Proposed sequence:

1. Psychological stress or *Atichinta* may initiate mental and autonomic stress responses.
2. Autonomic and HPA-axis responses may alter heart rate, vascular tone, blood pressure, metabolic regulation, and inflammatory signaling.
3. Repeated exposure in susceptible individuals may contribute to endothelial and/or microvascular dysfunction and increased cardiovascular vulnerability.
4. Within the Ayurvedic framework, *Atichinta* is described as a *Nidana* for *Rasavaha Srotas Dushti*; *Sanga* and other *Srotodushti* patterns may be used to describe disturbed channel function.

The two frameworks can therefore be compared at the level of function and clinical observation, but their terms should not be treated as interchangeable biomedical diagnoses. The principal value of the Manas–Hridaya Axis is methodological: it provides a structured way to formulate questions that can be tested using both Ayurvedic assessments and validated biomedical endpoints.

6. Clinical and Translational Implications

The proposed framework may have value in integrative cardiovascular research if Ayurvedic constructs are operationalized prospectively. Researchers could examine whether systematically assessed *Atichinta* or related mental characteristics are associated with validated measures of perceived stress, anxiety/depression, sleep, autonomic function, blood pressure variability, endothelial function, or coronary microvascular function.

Assessment of *Hridaya*-related symptoms such as heaviness or chest discomfort should not replace standard cardiovascular evaluation. Any patient with new, persistent, exertional, or unexplained chest pain requires appropriate clinical assessment. Ayurvedic terminology may complement but should not substitute for electrocardiography, biomarkers, echocardiography, stress testing, coronary imaging, or other investigations when clinically indicated.

Mind–body interventions such as meditation and yoga have been studied for stress and cardiovascular risk reduction, but evidence for specific Ayurvedic interventions should be evaluated independently. It would be inappropriate to infer efficacy of Abhyanga, Panchakarma, or other Ayurvedic therapies for cardiovascular disease solely from the biological plausibility of the Manas–Hridaya model.

7. LIMITATIONS

This narrative review is subject to several limitations. The selection and interpretation of literature may be influenced by author judgment, and classical Ayurvedic concepts cannot be assumed to have exact anatomical or molecular equivalents within modern biomedicine. Although psychological stress is associated with cardiovascular disease, the relationship is bidirectional and influenced by multiple factors, and current evidence largely supports associations or mechanistic plausibility rather than causation. Proposed correlations between *Sanga* and microvascular dysfunction, *Rasa Dushti* and inflammatory/metabolic states, and *Vyana Vayu* and autonomic physiology remain hypothetical and require empirical validation. Moreover, symptoms such as chest heaviness, *Gaurava*, and *Hridgraha* are nonspecific and cannot independently establish coronary microvascular dysfunction or ischemia.

8. Research Priorities

Future studies should move from analogy to operational testing. Priority areas include:

- Development and validation of reproducible Ayurvedic assessment tools for *Atichinta* and *Rasavaha Srotas Dushti.*
- Prospective studies correlating Ayurvedic assessments with perceived-stress scales, anxiety/depression measures, HRV, ambulatory blood pressure, inflammatory biomarkers, endothelial function, and metabolic parameters.

- Studies of coronary microvascular function using validated endpoints such as coronary flow reserve in appropriately selected populations.
- Evaluation of whether Ayurvedic phenotypes add predictive value beyond established cardiovascular risk factors.
- Preregistered interventional studies of specific Ayurvedic or mind–body interventions with clinically meaningful cardiovascular outcomes.
- Mechanistic studies that explicitly test, rather than assume, proposed relationships among Ayurvedic constructs, autonomic regulation, HPA-axis activity, inflammation, and vascular function.

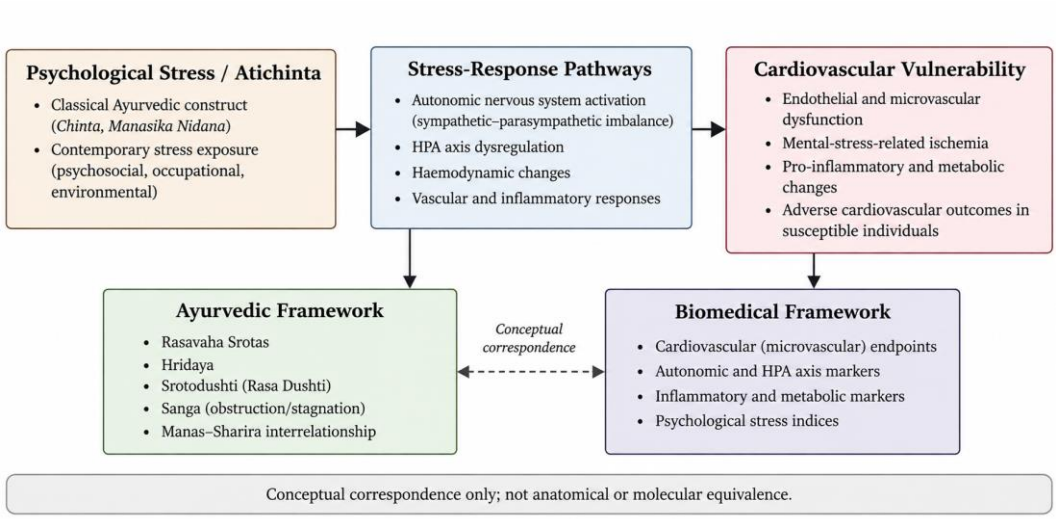


Figure 1: Proposed Manas–Hridaya Axis. Ayurvedic conceptual framework from contemporary biomedical stress–cardiovascular pathways.

Table 1. Contemporary pathways linking psychological stress with cardiovascular dysfunction

Pathway	Major mediators/markers	Potential cardiovascular effects
Sympathetic activation [4–7,15,16]	Catecholamines; HRV; heart rate; blood pressure	Tachycardia, vasoconstriction, altered autonomic balance, arrhythmogenic vulnerability
HPA-axis activation [4–7]	CRH, ACTH, cortisol	Metabolic and vascular effects; altered stress adaptation
Endothelial dysfunction [7,14]	Nitric oxide bioavailability, oxidative stress, vascular reactivity	Impaired vasodilation and increased vascular vulnerability
Inflammatory signaling [7,14]	CRP, IL-6, TNF-related pathways	Atherosclerotic progression and plaque vulnerability
Microvascular dysfunction [11–13,17–19]	Coronary flow reserve, vasomotor abnormalities	Angina/ischemia without obstructive coronary disease; stress-related ischemia
Stress cardiomyopathy [20–22]	Catecholamine surge; myocardial and microvascular mechanisms	Transient LV dysfunction and acute heart-failure phenotype

Table 2. Cautious conceptual mapping for the proposed Manas–Hridaya Axis.

Ayurvedic construct	Classical context	Possible modern research correlate	Interpretive status
Atichinta	Mental factor associated with Rasavaha Srotas Dushti [1]	Psychological stress / persistent worry	Conceptually comparable; not identical
Hridaya	Important site associated with Rasa and Manas-related concepts [1–3,9]	Heart and brain–heart regulatory network	Functional analogy only
Vyana Vayu	Ayurvedic functional principle related to movement and circulation	Autonomic and cardiovascular regulation	Hypothesis; not anatomical equivalence
Rasa Dushti	Disturbed Rasa-related physiology	Broad nutritional/metabolic/inflammatory states	Conceptual comparison
Sanga	One pattern of Srotodushti	Impaired flow or functional vascular disturbance	Hypothesis; not equivalent to microvascular obstruction
Hridaya-related symptoms	Classical clinical descriptors	Symptoms requiring standard cardiovascular assessment	Non-specific; not diagnostic biomarkers

9. CONCLUSION

Classical Ayurveda provides a coherent conceptual association between *Atichinta, Rasavaha Srotas Dushti*, and the central importance of *Hridaya*. Contemporary cardiovascular research independently demonstrates that psychological stress can influence cardiovascular risk through autonomic, neuroendocrine, inflammatory, vascular, and myocardial pathways [4–7]. These observations provide a reasonable basis for a *Manas–Hridaya* research framework, but they do not establish one-to-one equivalence between Ayurvedic concepts and modern anatomical or molecular entities. The *Manas–Hridaya* Axis should therefore be presented as a hypothesis-generating integrative model. Its scientific value will depend on whether Ayurvedic constructs can be operationalized and shown to predict or modify objectively measured cardiovascular outcomes in prospective, adequately controlled studies.

Financial Support and Sponsorship

Nil.

Conflicts of Interest

None declared.

Acknowledgement

None

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