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THE MANAS-HRIDAYA AXIS: A NARRATIVE REVIEW OF ATICHINTA (EXCESSIVE STRESS), RASAVAHASROTAS DUSHTI, AND THE PATHOGENESIS OF CARDIOVASCULAR DISEASE

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ABSTRACT

Background: Chronic psychological stress is an independent risk factor for cardiovascular disease (CVD), contributing to approximately 30% of myocardial infarctions. In Ayurveda, excessive stress (*Atichinta*) is identified as a primary etiological factor for vitiation (*Dushti*) of *Rasavaha Srotas* the channels carrying the body's primary nutritive fluid (*Rasa Dhatu*). While *Rasavaha Srotas* is conceptually synonymous with the cardiovascular system, the precise mechanistic pathways linking *Atichinta* to *Srotodushti* remain undefined. **Objective**: To propose a conceptual framework for the *Manas-Hridaya* axis, mapping the neuroendocrine, inflammatory, and vascular pathways of chronic stress onto the classical Ayurvedic stages of *Atichinta*, *Rasavaha Srotodushti*, and *Hridaya Roga*. **Methods**: A narrative review was conducted using Ayurvedic primary sources (*Charaka Samhita*, *Sushruta Samhita*) and PubMed (2000–2026) for chronic stress neuro-cardiology, endothelial dysfunction, Takotsubo cardiomyopathy, mental stress-induced myocardial ischemia, and HPA axis. **Discussion**: The *Manas* (mind) and *Hridaya* (heart) are anatomically connected in classical texts, with the heart as the common root for both *ManovahaSrotas* and *Rasavaha Srotas*. *Atichinta* causes *Sanga* (obstruction) in *RasavahaSrotas*. Modern research validates this: chronic stress hyperactivates the sympathetic nervous system and HPA axis, triggering vascular inflammation, endothelial dysfunction, and myocardial ischemia. *Agni Dushti* (deranged metabolic fire) correlates with stress-induced mitochondrial dysfunction and a pro-inflammatory state. **Conclusion**: *Atichinta* is a direct pathophysiological driver of *Rasavaha Srotodushti*. The *Manas-Hridaya* axis provides a model wherein clinical signs

of *Sanga* can be correlated with microvascular dysfunction and inflammation, enabling integrative therapeutic strategies.

KEYWORDS: Rasavaha Srotas, Atichinta, Stress, cardiomyopathy; Endothelial dysfunction, Manovaha Srotas, Hridaya, Integrative physiology.

1. INTRODUCTION

The ancient Ayurvedic physicians understood what modern cardiology is only now beginning to validate: the arrow of causality between the mind and the heart is a direct, two-way street [1-3]. Contemporary research has unequivocally established chronic psychological stress as a major, independent risk factor for cardiovascular disease (CVD), with stress contributing to up to 30% of myocardial infarctions and increasing the risk of coronary heart disease by approximately 40%–50% across meta-analyses of prospective cohort studies [4-6]. A large Norwegian population study found that individuals with health anxiety had a 2.12-fold higher risk of developing ischemic heart disease compared to those without such anxiety [7]. The foundational texts of Ayurveda, particularly the *Charaka Samhita* and *Sushruta Samhita*, provide a highly structured model for this very process [1-3]. In the face of a modern CVD landscape responsible for an estimated 17.9 million deaths annually worldwide this ancient model may hold the key to novel, person-centric prevention and treatment strategies [8].

The central player in this model is *Rasavaha Srotas* the body's primary channel system responsible for the formation and circulation of *Rasa Dhatu*, the nutritive plasma/lymph that is the first product of digestion and the essential substrate for all other tissues [9-11]. The root (*Mula Sthana*) of RasavahaSrotas is *Hridaya* (the heart) [3]. Simultaneously, the classical texts identify *Atichinta* (excessive, pathological worrying or mental stress) as a specific cause of *Srotodushti* (channel vitiation), with one classical treatise explicitly stating that*Atichinta* causes *Rasavaha Srotodushti* and impairs *Hridaya* function [12]. The term "Chinta" has been defined in Ayurveda not merely as ordinary worry but as a pathological cognitive processing that directly damages cellular faculties (*Dhatu*), thereby initiating disease [13]. Thus, the Ayurvedic framework precisely postulates that *Atichinta* → *RasavahaSrotodushti* → *Hridaya Rogas* (heart diseases).

This review is a narrative synthesis, not a systematic meta-analysis. Our primary objective is to bridge this taxonomic gap by first deconstructing the classical concepts of Rasavaha Srotas, its*Dushti*, and the role of *Chinta/Atichinta*, then mapping them onto modern pathophysiological mechanisms of stress-induced cardiovascular disease including nervous system dysregulation

[14-16], microvascular dysfunction [17-18], and systemic inflammation [19-20] and finally synthesizing an integrative model of the "Manas-Hridaya Axis." By identifying critical, testable research gaps, we aim to move this field from descriptive analogy to predictive, mechanistic science [21-22].

2. METHODS

A qualitative iterative search was conducted to synthesize Ayurvedic and modern concepts of stress-induced CVD.

Ayurvedic sources: *Charaka Samhita* (Cikitsasthana 15) and *Sushruta Samhita* (Sutrasthana 14) were searched for: *Rasavaha Srotas, Hridaya, Chinta, Atichinta, Srotodushti, Sanga, Dasha Dhamani, Vyana Vayu,* and *Rasa Dhatu*. Modern Ayurvedic literature (2015–2026) was also searched.

Modern literature: PubMed (2000–April 2026) used MeSH terms including: "chronic stress" + CVD; endothelial dysfunction + stress; Takotsubo cardiomyopathy; mental stress-induced myocardial ischemia; autonomic nervous system + heart disease; HPA axis + CVD; inflammation + atherosclerosis + stress; heart rate variability + stress; cortisol + CVD; microvascular dysfunction + stress; Prakriti + stress; meditation + CVD + RCT; and yoga + CVD.

Inclusion: Original research, reviews, and clinical studies linking psychological stress to cardiovascular pathology (neuroendocrine, inflammatory, vascular). Primary Ayurvedic texts and interpretative articles included.

Exclusion: Non-psychological stressors; behavioral risk factors alone; single case reports (unless unique mechanistic insight).

Data extraction: Qualitative synthesis into three domains: (1) Ayurvedic model of *Atichinta* → *Rasavaha Srotodushti*; (2) modern stress–CVD model; (3) integrative model and research gaps.

3. Mapping the Manas-Hridaya Axis

3.1 The Ayurvedic Model: Atichinta and RasavahaSrotas Dushti

3.1.1 RasavahaSrotas: Anatomy and Function

Both Charaka and Sushruta identify the *Mula Sthana* (root) of RasavahaSrotas as *Hridaya* (the heart) and the *Dasha Dhamanis* (the ten great vessels) [9-10]. This is a striking anatomical correlate to the modern cardiovascular systemthe heart and its major arteries [10]. The function of RasavahaSrotas is *Rasa Samvahana*, the circulation of the first-formed *Dhatu* (tissue) derived from digested food [1-3]. This subtle, nutrient-dense *Rasa* is the precursor for all other

tissues, including *Rakta* (blood), *Mamsa* (muscle), and *Meda* (fat) [23]. Therefore, the health of the entire body depends on the unimpeded flow of Rasa through its channels. Importantly, Hridaya is described not just as a pump but as a *Marma* (a vital point) and a multi-srotas portal, also acting as the seat of *Manas* (the mind) and the root of *ManovahaSrotas* [23-24]. This unique position positions the heart as the central hub where psychological and nutritional signals converge [25].

3.1.2 The Pathogenic Role of Atichinta (Excessive Stress)

The *Charaka Samhita* explicitly lists ***Atichinta*** (excessive worrying or mental stress) as a primary, causative factor (*Hetu*) for vitiating (*Dushti*) the *RasavahaSrotas* [1-3]. It is considered a direct cause of *Rasavaha Srotodushti* and a *Manasika Nidana* (mental cause), which can impair *Hridaya* function [12]. According to classical teachings, excessive *Chinta* (worry) destroys *Ojas* (the body's vital essence and immunological strength), leading to loss of strength, complexion, and resistance to disease [13].

The signs and symptoms of *RasavahaSrotodushti* include *Aruchi* (anorexia), *Anna Dvesha* (aversion to food), *Gourava* (heaviness), *Tandra* (drowsiness), and crucially, *Hridaya Upalepa* (a feeling of congestion in the heart) and *Hridgraha* (tightness in the chest) [1]. The pathology of *Atichinta* likely operates through interconnected *Doshic* mechanisms: (1) *Atichinta* aggravates Vata dosha, particularly *Vyana Vayu*, which governs circulation, leading to irregular and turbulent flow[26]; (2) it simultaneously suppresses *Agni* (digestive and metabolic fire), imparting *Sheeta* (cold) and *Guru* (heavy) qualities to the *Rasa*, making it prone to obstruction [1]. The *Sharangadhara Samhita* further notes that *Chinta*and*Shoka*(grief) are predominant causes of *Rasakshaya* (depletion of nutritive fluid), which directly parallels modern observations of stress-induced metabolic dysregulation [23].

3.1.3 RasavahaSrotodushti: The Pathway to Vyadhi (Disease)

The vitiation of a *Srotas* follows a specific sequence. The primary form of *Dushti* that results from *Atichinta* is *Sanga* (obstruction) [1]. The "*Sheeta*,""*Guru*" quality of the vitiated *Rasa*, coupled with Vata's deranged flow, leads to blockage, particularly in the microcirculatory channels [24]. This pattern provides a direct Ayurvedic correlate to modern concepts of microvascular dysfunction [17,27]. Chronic obstruction can lead to a state of "*Atipravritti*" (excessive flow through alternate routes) and, ultimately, the formation of "*Siragranthi*" (vascular nodules), which can be correlated to atheromatous plaques [24]. The classical text *Hridaya Chintamani* explicitly links stress-induced *Vata* aggravation to cardiac pathology, describing *Hridgraha* (angina-like chest tightness) as a direct manifestation of this process [28]. Importantly, this framework suggests that psychological pathology precedes organic

structural changes, a crucial insight that modern medicine has only recently begun to appreciate in conditions like mental stress-induced myocardial ischemia [29-31].

3.2 The Modern Model: Chronic Stress and Cardiovascular Disease

3.2.1 Nervous System Dysregulation

The primary modern correlates for *Atichinta* and *Vyana Vayu* are the hyperactivation of the sympathetic nervous system (SNS) and the hypothalamic-pituitary-adrenal (HPA) axis [32,33]. Chronic stress leads to a state of autonomic dysfunction, characterized by sustained tachycardia, hypertension, and a pro-arrhythmic state [14,15]. This stress-induced shift in autonomic balance is directly measurable as a reduction in heart rate variability (HRV) , a powerful predictor of adverse cardiovascular events [34-36]. The relationship is dose-dependent: a meta-analysis of HRV studies confirmed that lower HRV is consistently associated with reduced stress resilience and increased cardiovascular risk [34]. Central stress pathways connecting the brain to the cardiovascular system have been identified, with pre-autonomic brain-body networks directly influencing baroreflex functioning and sympathetic outflow to the heart and vasculature [32,37]. Animal models have demonstrated that chronic stress causes dysautonomia and structural remodeling of autonomic nuclei, further perpetuating cardiac risk [32].

3.2.2 Vascular Dysfunction and Inflammation

The hemodynamic changes from SNS activation directly traumatize the delicate endothelial lining, initiating an inflammatory cascade and endothelial dysfunction [38-41]. Chronic stress fosters a systemic pro-inflammatory state, marked by elevated cytokines like IL-6 and TNF-α, which promote leukocyte adhesion and accelerate atherosclerosis[42]. Elevated TNF-α in particular is strongly associated with endothelial dysfunction and angiogenesis-mediated atherosclerotic plaque progression, with secretion increasing as the duration of stress is prolonged [20]. Stress hormones – norepinephrine, epinephrine, and cortisol – act in concert to orchestrate this vascular inflammation [43,44]. Multiple prospective cohort studies have confirmed that elevated cortisol independently predicts increased cardiovascular and all-cause mortality [44]. This creates a direct cellular correlate to the *Sheeta* and *Guru* quality of vitiated *Rasa* becoming obstructed in small vessels and forming *Siragranthi*. Table 1 summarizes the key physiological pathways linking chronic stress to cardiovascular pathology.

3.2.3 Myocardial Consequences: From Ischemia to Stunning

Beyond chronic vascular changes, stress can acutely and dramatically affect the myocardium. Mental stress-induced myocardial ischemia (MSIMI) is a well-established

phenomenon where psychological stress, not exercise, triggers a reduction in blood flow, often silently [45]. Importantly, MSIMI can occur in patients with normal cardiac stress testing and is only weakly related to the severity of coronary artery disease, suggesting a microvascular mechanism [29]. A meta-analysis of MSIMI studies found that patients with MSIMI have a doubled risk of subsequent cardiovascular events, independent of traditional risk factors [31]. The condition is more prevalent in women and younger patients, underscoring the importance of sex-specific mechanisms in stress-induced cardiac pathology [30]. The most dramatic example of the *Atichinta-Hridaya* connection is Takotsubo syndrome (TTS) or stress cardiomyopathy, a transient, acute heart failure syndrome triggered by intense emotional or physical stress, mediated by a catecholamine surge and microcirculatory dysfunction [46-48]. TTS is increasingly recognized as a model disease for studying the brain-heart axis, providing direct clinical evidence that psychological events can induce profound, though reversible, structural changes in the left ventricle [46,49].

3.3 The Integrative Framework: Introducing the Manas-Hridaya Axis

The convergence of Ayurvedic and modern perspectives allows us to propose a unified Manas-Hridaya Axis: a bidirectional functional unit where the heart serves as the central hub for the channels of mind (*ManovahaSrotas*) and nutrition (*RasavahaSrotas*) [1,23,24]. The axis operates as follows:

1. Psychological input (*Atichinta*) → Sympathetic overdrive (*Vyana Vayu* aggravation) → Hemodynamic stress → Endothelial trauma.
2. Simultaneously, *Atichinta* → *Agni Dushti* → Production of *Sheeta/Guru*quality*Rasa* →Set point for pro-inflammatory, hypercoagulable state.
3. Combined effects → *Sanga* (microvascular obstruction) → *Hridaya Roga* (MSIMI, TTS, CAD).

The clinical and translational promise of this integrative model is substantial. It suggests that the Ayurvedic physician, when diagnosing *RasavahaSrotodushti*, may be observing the clinical stigmata of early autonomic dysfunction and microvascular disease stages at which modern pathology has not yet manifested as obstructive coronary disease. Conversely, the modern cardiologist might benefit from an Ayurvedic lens: the presence of a *Pitta Prakriti* (a constitutional type characterized by heightened metabolic "fire") could help identify individuals with exaggerated sympathetic responses, predicting a higher risk of MSIMI [50,51]. **Table 2** summarizes the key correlates of this integrated axis.

4. DISCUSSION

The ancient wisdom of Ayurveda provides a master framework for the phenomenon of stress-induced heart disease. The journey from the subtle psychological event (*Atichinta*) to the gross, organic disease (*Hridaya Roga*) is meticulously mapped through a sequence of functional breakdowns that aligns precisely with modern psychoneuroimmunological models of CVD [1,2,3,13]. We have synthesized this alignment into a unified *Manas-Hridaya Axis*, a bidirectional functional unit where the heart serves as the central hub for the channels of mind (*ManovahaSrotas*) and nutrition (*RasavahaSrotas*) [24].

The model aligns with contemporary understanding of the brain-heart axis, a critical physiological axis that is increasingly recognized as central to health and disease[52,53]. This axis is not merely conceptual but has identifiable anatomical (insular cortex, autonomic ganglia, cardiac nerves), cellular (macrophages, endothelial cells, cardiomyocytes), and molecular (catecholamines, cytokines, NOX enzymes) substrates [54,55]. The discovery that resident cardiac macrophages (CCR2-MHCII+) become activated during sympathetic overstimulation, leading to fibrosis, impaired diastolic function, and NADPH oxidase 4 (NOX4)-dependent oxidative stress, provides a direct cellular correlate to the Ayurvedic concept of *Vata-induced Srotodushti* leading to structural remodeling [56]. Similarly, the link between chronic stress, glucocorticoid release, endothelial glucocorticoid resistance, and accelerated atherosclerosis [39,42] maps precisely onto the sequence of *Agni Dushti* → *Rasa Dushti* → *Sanga*.

The clinical and translational promise of this integrative model is substantial. If validated, it would suggest that Ayurvedic assessments of *RasavahaSrotodushti* – based on simple clinical parameters like chest tightness, heaviness, anorexia, and drowsiness – could serve as cost-effective screening tools for early microvascular dysfunction and stress-induced cardiac vulnerability. This could enable earlier intervention, potentially through lifestyle and mind-body practices that have already shown efficacy in reducing stress and improving cardiovascular outcomes [57-63].

This review has several limitations. The primary sources of Ayurveda were composed over centuries, and later commentaries are not uniform. The term *Srotas* itself carries connotations of channel dynamics that may not map perfectly onto the fixed cellular architecture of the microvasculature. Moreover, the relationship between psychological stress and CVD is bidirectional and confounded by behavioral factors (diet, exercise, sleep) that we have not extensively discussed [6,4]. Finally, the absence of rigorous interventional studies in the

Ayurvedic tradition means that causality cannot be inferred; all homologies remain correlative until tested.

Despite these caveats, the research gaps identified herefrom the absence of clinical correlations between *Sanga* and coronary flow reserve to the lack of *Prakriti*-stratified studies on MSIMI susceptibilityrepresent a powerful roadmap, urging researchers to move beyond simple validation and toward the creation of a unified, predictive model of mind-body health in cardiovascular medicine.

5. Tables

Table 1: Major Modern Pathophysiological Pathways Linking Chronic Stress to Cardiovascular Disease.

Pathway	Key Mediators	Primary Cardiovascular Effects	Clinical Outcomes
Sympathetic Nervous System (SNS) Activation	Norepinephrine, epinephrine	Tachycardia, increased contractility, vasoconstriction, reduced HRV	Hypertension, myocardial ischemia, arrhythmias [14, 15, 32, 36]
HPA Axis Dysregulation	Cortisol, CRH, ACTH	Sodium retention, impaired glucose metabolism, endothelial dysfunction	Hypertension, insulin resistance, metabolic syndrome[33,43,44, 64, 65]
Endothelial Dysfunction	Reduced NO bioavailability, increased endothelin-1, oxidative stress	Impaired vasodilation, increased vascular permeability, leukocyte adhesion	Atherosclerosis, vasospasm, thrombosis [38-41]
Chronic Inflammation	IL-6, TNF-α, CRP, NF-κB activation	Leukocyte recruitment, plaque destabilization, systemic inflammatory state	Atherosclerotic plaque progression, MI, stroke [19,20,42,6]
Hypercoagulability	Platelet aggregation, fibrinogen, PAI-1	Prothrombotic state, increased thrombus formation	Venous thromboembolism, acute coronary syndromes [66,67]
Microvascular Dysfunction	Endothelial, smooth muscle, pericyte dysfunction	Reduced coronary flow reserve, impaired vasodilation, microvascular spasm	Angina (INOCA), heart failure, MSIMI, Takotsubo [17,18,27,49]

Table 2: The Manas-Hridaya Axis – An Integrated Pathway from Atichinta to Hridaya Roga.

Stage of	Ayurvedic	Ayurvedic Functional	Modern	Key	Cross-Vali

Pathogenesis	Correlate	Description	Correlate	Mechanistic Link	dation
1. Psychological Trigger	*Atichinta* [1, 2,3,12, 13]	Excessive pathological worrying or mental stress. Indicated as the most potent mental cause (*Manasika Nidana*) of *Rasakshaya* (depletion of nutritive fluid). [23]	Chronic Psychosocial Stress [4-7]	HPA axis & SNS hyperactivation [14,15,33]; stress measured as HRV reduction [34]	Stress questionnaires & heart rate variability (HRV) studies in high-stress occupations [34]
2. Neural Transduction	*Prana* & *Vyana Vayu* [1] [2] [26]	The sub-doshas of Vata that govern the mind and circulation. *Vyana Vayu* is synthesized in myocytes and responsible for action potential genesis and vascular tone. [26]	Autonomic Nervous System [14,32,37]; Brain–Heart Axis [25,52,53]	*Vyana Vayu* carries the "emotional signal" from the mind to the heart. Known anatomical pathways: insula → autonomic ganglia → cardiac nerves [25,49]	PET/fMRI brain imaging during mental stress: insula, anterior cingulate activation [37]
3. Deranged Nutrition	*Rasa Dhatu Dushti* [1] [23]	Nutritive *Rasa* becomes *Sheeta* (cold) and *Guru* (heavy). *Agni Dushti* leads to production of *Ama* (toxins). [1]	Pro-inflammatory, hypercoagulable state [20, 42,66]	Stress-induced *Agni Dushti* → elevated cortisol (promotes pro-inflammatory phenotype) [43,44]; increased fibrinogen, PAI-1 [66]	Metabolic syndrome parameters: CRP, IL-6, fibrinogen, insulin resistance [65]
4. Channel Dysfunction	*Sanga* (Obstruction) [1] [24]	Functional obstruction of fine channels of *RasavahaSrotas*. Described as *Hridaya Upalepa* (congestion in the heart). [1]	Endothelial & Microvascular Dysfunction [17 ,18,39,40]	Inflammation, oxidative stress, vasoconstriction, reduced coronary	Coronary flow reserve (CFR) by PET/MRI; microvascular angina diagnosis

				flow reserve (CFR) [17] [27]	(INOCA) [17] [18]
5. Vascular Pathology	*Siragranthi* [24]	Nodular formations within blood channels (atheromatous plaques). "*Srotosanga* is analogous to atherosclerotic vascular disease". [24]	Atherosclerotic Plaque [19] [42] [6]	Chronic inflammation & endothelial injury → lipid deposition, foam cell formation [19] [42]	Imaging: coronary calcium score, CTA; histology from endarterectomy specimens
6. Cardiac Event	*Hridaya Roga* [1] [24] [28]	Manifestation of heart disease. Specific term *Hridgraha* (angina-like tightness). [28]	MSIMI, Takotsubo, MI, Arrhythmias [29] [30] [31] [46] [47] [48]	Catecholamine surge, coronary microvascular spasm, demand-supply mismatch, NOX4-dependent ROS [46] [48] [56]	ECG, troponin, echocardiography, coronary angiography, wall motion studies [29] [30] [46]

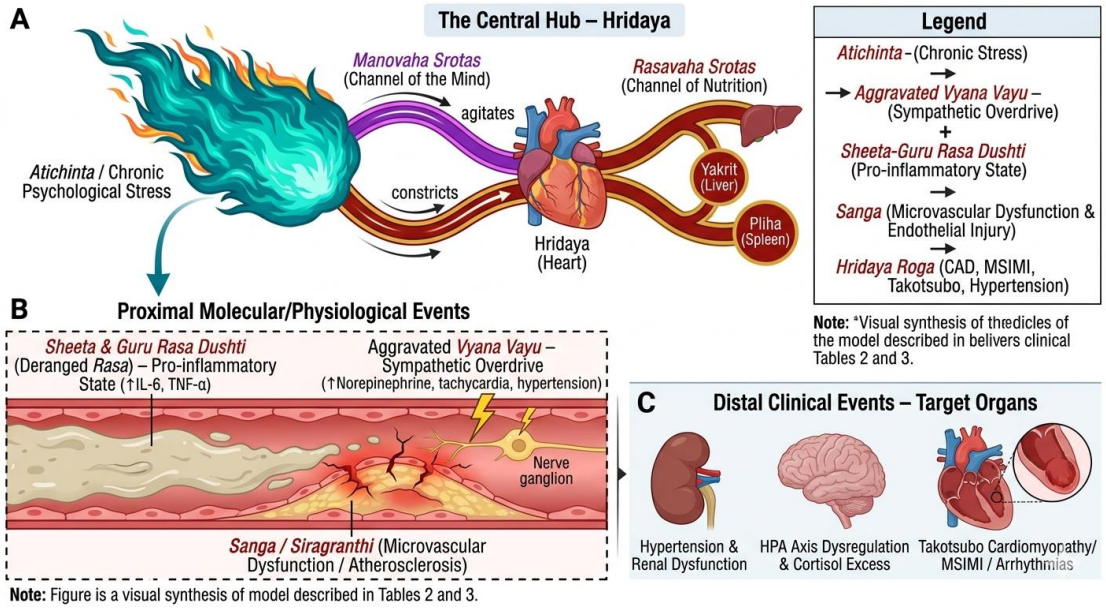

Figure 1: Multi-panel schematic depicting the integrated pathway of the Manas-Hridaya Axis.

6. CONCLUSION

The ancient concept of *Atichinta* leading to *Hridaya Roga* via *Rasavaha Srotodushti* is a functionalblueprint for modern psycho-neuro-cardiology. The proposed Manas-Hridaya Axis maps chronic psychological stress (sympathetic overdrive, endothelial inflammation, microvascular dysfunction, hypercoagulability) onto *Vyana Vayu* aggravation, *Rasa Dushti*, and*Sanga*. This yields falsifiable hypotheses: *Prakriti*-based susceptibility to stress-induced ischemia, coronary flow reserve as a measure of *Sanga*, testing *Vyana Vayu*-pacifying interventions(meditation, *Abhyanga*), and linking *Agni Dushti* to the HPA axis. Practical assessment includes *Hridaya Upalepa* as an early marker of microvascular dysfunction. For cardiologists, a stressed patient's "chest heaviness" without obstructive disease is not merely psychosomatic but genuine microvascular pathologythe *Sanga* described millennia ago. Bridging this gap transforms understanding and enables personalized, integrated cardiology.

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