

# Mapping the Clinical Complaints of Sleep Insufficiency Due to Nidra Vegadharana:-: A Convergent Analysis of Classical Ayurvedic Symptoms and Modern Systemic Biomarkers

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**Abstract** - Sleep constitutes a fundamental biological imperative, recognized across medical traditions as essential for homeostatic maintenance, cognitive preservation, and systemic resilience. In Ayurveda, Nidra is enumerated among the Trayopastambha—the three pillars sustaining life—alongside Ahara (nutrition) and Brahmacharya (regulated conduct). Contemporary systems biology similarly categorizes sleep as a critical neurophysiological process subserving metabolic clearance, synaptic plasticity, neuroendocrine regulation, and immunological surveillance. This article presents a comprehensive convergent analysis of sleep insufficiency, delineating direct correlations between classical Ayurvedic clinical symptomatology of Anidra (insomnia/sleep deprivation states) and validated modern physiological biomarkers. Through systematic mapping of 27 classical symptoms against contemporary pathophysiological endpoints, we demonstrate that ancient clinical observations presaged modern understanding of sleep-related systemic decompensation. The analysis encompasses neurocognitive, metabolic, endocrine, immunological, and gastrointestinal dimensions, revealing that sleep insufficiency represents a multi-systemic stressor activating convergent pathological cascades. We propose an integrative framework wherein Ayurvedic diagnostic paradigms and lifestyle therapeutics complement evidence-based sleep medicine, offering enhanced clinical precision and therapeutic breadth for the management of sleep deficiency disorders.

**Keywords**--Nidra, Anidra, Nidra vegadharana , Sleep Deprivation, Vata Dosh, Circadian Rhythm, Cortisol, Leptin, Ojas

## I. INTRODUCTION

Contemporary global health surveillance indicates that approximately 33% of the adult population experiences insufficient sleep duration or quality, with prevalence rates exceeding 45% in urbanized, high-income nations (Liu et al., 2021). This epidemic of sleep deficiency carries profound public health implications, contributing to an estimated \$411 billion in annual economic losses across industrialized economies and serving as a modifiable risk factor for cardiovascular disease, metabolic syndrome, neurocognitive decline, and all-cause mortality (Hafner et al., 2017; Cappuccio et al., 2010).

While modern sleep medicine has elucidated the neurobiological substrates, genetic determinants, and molecular effectors of sleep regulation, classical Ayurvedic texts articulated comprehensive clinical frameworks for understanding sleep insufficiency approximately two millennia prior to the advent of polysomnography. The Charaka Samhita, Sushruta Samhita, and Ashtanga Hridaya contain detailed descriptions of Nidra physiology, Anidra (insomnia) pathogenesis, and the systemic consequences of sleep disruption observations that demonstrate remarkable concordance with contemporary pathophysiological models (Sharma & Dash, 2019; Vagbhata, 2019).

This convergence between ancient phenomenological observation and modern molecular medicine presents a unique opportunity for integrative scholarship. By mapping classical Ayurvedic symptomatology onto validated modern biomarkers, we can not only validate traditional knowledge systems but also generate novel hypotheses regarding the systemic integration of sleep physiology. The present article undertakes a systematic comparative analysis, examining the neurocognitive, metabolic, endocrine, immunological, and gastrointestinal manifestations of sleep insufficiency through dual theoretical lenses, culminating in an integrative framework for clinical assessment and therapeutic intervention.

## II. CONCEPTUAL FOUNDATIONS: SLEEP IN CLASSICAL AND CONTEMPORARY PERSPECTIVE

### 2.1 The Ayurvedic Ontology of Nidra

Within the Ayurvedic nosology, Nidra (sleep) is conceptualized as a fundamental state of consciousness governed by the interplay of the Triguna, the three cosmic qualities of Sattva (clarity), Rajas (activity), and Tamas (inertia). According to Acharya Charaka, sleep ensues when the Manas (mind) and Indriyas (sense and motor faculties) become fatigued and withdraw from their respective objects, a process termed Klama (exhaustion).



The predominant influence of Tamas and Kapha Dosha—characterized by qualities of heaviness, stability, unctuousness, and density—creates the physiological conditions conducive to deep, restorative sleep (Charaka Samhita, Sutrasthana 21.35-40).

Notably, Ayurveda distinguishes between physiologically appropriate sleep (Samayika Nidra) occurring at designated hours, pathological excess sleep (Atinidra), and insufficient sleep (Anidra). The latter is recognized as a Vata-Pitta predominant disorder, wherein the dry, mobile, and catabolic qualities of Vata disrupt the grounding, anabolic functions of Kapha. Chronically, this imbalance depletes Ojas—the subtle essence of immunity, tissue integrity, and psychological resilience—while promoting the accumulation of Ama (metabolic toxins) through impaired Agni (digestive-metabolic fire) (Sharma, 2020).

The Sushruta Samhita further elaborates that sleep quality reflects systemic homeostasis, asserting that Sukha (well-being) and Duhkha (suffering) are contingent upon proper sleep, and that conditions of pain, disease, and debility are frequently rooted in sleep disturbances (Sushruta Samhita, Sharirasthana 4.34). This systemic conceptualization anticipates modern understandings of sleep as an integrative homeostatic process affecting all physiological systems.

### *2.2 Neurobiological Foundations of Sleep in Contemporary Medicine*

Modern sleep science has established that sleep is not a passive state of quiescence but an active, dynamically regulated neurophysiological process. The two-process model of sleep regulation, proposed by Borbély (1982) and subsequently refined, provides a foundational framework:

Process (Homeostatic Sleep Drive): Accumulation of sleep pressure during wakefulness, mediated by neurochemicals including adenosine, prostaglandin D<sub>2</sub>, and cytokines. Adenosine progressively accumulates in the basal forebrain and cortex during waking hours, promoting sleep onset by inhibiting cholinergic and orexinergic arousal systems (Porkka-Heiskanen et al., 1997). The clearance of adenosine during sleep resets this homeostatic pressure, establishing a cyclical regulatory pattern.

Process C (Circadian Rhythm): The endogenous, approximately 24 hour biological rhythm generated by the suprachiasmatic nucleus (SCN) of the anterior hypothalamus. This "master clock" entrains peripheral circadian oscillators through neuroendocrine outputs, primarily via the pineal secretion of melatonin.

The SCN also regulates the sleep-wake cycle through projections to the ventrolateral preoptic nucleus (VLPO), which promotes sleep, and the locus coeruleus, tuberomammillary nucleus, and orexinergic neurons, which promote wakefulness (Saper et al., 2005).

Recent advances have further elucidated the structural architecture of sleep, comprising non-rapid eye movement (NREM) stages (N1, N2, N3) and rapid eye movement (REM) sleep. Deep NREM sleep (slow-wave sleep) appears particularly crucial for metabolic clearance via the glymphatic system, synaptic downscaling, and growth hormone secretion, while REM sleep subserves emotional processing and memory consolidation (Xie et al., 2013; Walker & Stickgold, 2010).

### *2.3 Integrating Ontologies: Points of Convergence*

Despite differing epistemological foundations, Ayurvedic and modern sleep frameworks converge on several fundamental principles:

1. Sleep as systemic homeostasis: Both traditions recognize sleep as essential for the maintenance of all physiological systems, not merely neurological function.
2. Circadian rhythm recognition: Ayurveda's emphasis on Dinacharya (daily regimen) reflects an implicit understanding of circadian biology, prescribing specific activities and sleep-wake timing aligned with solar cycles.
3. Neuroendocrine feedback: Ayurvedic conceptualization of Vata as the governing principle of movement, nerve transmission, and catabolism parallels modern recognition of sympathetic-adrenal activation and cortisol dysregulation in sleep deprivation.
4. Metabolic integration: The centrality of Agni (digestive fire) in Ayurveda finds modern correlatives in the profound metabolic disruptions, insulin resistance, leptin/ghrelin dysregulation—observed in sleep-deficient states.

## III. PATHOPHYSIOLOGY OF SLEEP INSUFFICIENCY: CONVERGENT MECHANISMS

### *3.1 Ayurvedic Pathogenesis (Samprapti) of Anidra*

The Ayurvedic model posits that Anidra arises through a sequence of pathological events culminating in systemic decompensation:



*Stage 1: Dosha Provocation.* Chronic sleep loss, excessive mental activity (Ati Chinta), or irregular lifestyle (Vishama Vihara) primarily aggravates Vata Dosha, particularly its Prana (neuro-cognitive) and Vyana (circulatory/cardiovascular) subtypes. Secondary Pitta aggravation occurs through the Vata-Pitta pathological synergism, generating thermal and catabolic stress.

*Stage 2: Srotas Obstruction.* The aggravated Vata and Pitta disrupt the normal flow of nutrients and metabolic products through the bodily channels (Srotas), particularly affecting Manovaha Srotas (psychological channels), Pranavaha Srotas (respiratory-neural channels), and Annavaha Srotas (digestive channels). This obstruction impairs the Vata-mediated transport of Ojas from its site of formation to peripheral tissues.

*Stage 3: Dhatu Depletion.* Progressive Vata aggravation promotes catabolic processes, leading to Dhatu Kshaya (tissue depletion). Initially, Rasa Dhatu (plasma/nutrient fluid) and Rakta Dhatu (blood) are compromised, followed by sequential depletion of deeper tissues (Mamsa, Meda, Asthi, Majja, and ultimately Shukra Dhatu).

*Stage 4: Ojas Kshaya.* The cumulative effect of tissue depletion and metabolic toxin accumulation manifests as diminished Ojas, the subtle essence of immunity and vitality. Clinically, this presents as reduced resistance to disease (Vyadhikshamathva), premature aging, psychological fragility, and systemic inflammatory susceptibility (Sharma & Dash, 2019; Vagbhata, 2019).

### 3.2 Modern Pathophysiological Mechanisms

Contemporary biomedical research has identified multiple interlocking pathophysiological mechanisms activated by insufficient sleep:

*Neuroendocrine Dysregulation:* Sleep curtailment triggers hypothalamic-pituitary-adrenal (HPA) axis hyperactivity, with meta-analytic evidence demonstrating robust cortisol elevation following acute and chronic sleep restriction (Boonstra et al., 2020). This hypercortisolemia exerts catabolic effects on musculoskeletal tissue, impairs glucose metabolism, suppresses immune function, and promotes central adiposity. Simultaneously, growth hormone secretion—normally peaking during slow-wave sleep—is attenuated, compromising tissue repair and metabolic regulation (Takahashi et al., 2017).

*Autonomic Imbalance:* Sleep loss shifts autonomic balance toward sympathetic predominance, evidenced by elevated heart rate, reduced heart rate variability, and increased blood pressure. This contributes to cardiovascular morbidity through endothelial dysfunction, arterial stiffness, and pro-inflammatory vascular changes (Somers et al., 2022).

*Glymphatic System Impairment:* Deep sleep facilitates convective flow of cerebrospinal fluid through perivascular spaces, clearing metabolic waste products including amyloid-beta, tau protein, and lactate (Xie et al., 2013). Sleep restriction impairs this clearance mechanism, contributing to the accumulation of neurotoxic proteins—a proposed mechanism linking chronic sleep deficiency with neurodegenerative disorders (Nedergaard & Goldman, 2020).

*Inflammatory Activation:* Sleep loss activates systemic inflammatory cascades, elevating circulating levels of C-reactive protein (CRP), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF- $\alpha$ ). This low-grade "inflammaging" state contributes to metabolic dysfunction, cognitive decline, and increased susceptibility to infection (Irwin et al., 2016).

*Metabolic Dyshomeostasis:* Sleep restriction dysregulates appetite-regulating hormones, decreasing leptin (satiety signal) and increasing ghrelin (hunger signal), while impairing glucose tolerance and insulin sensitivity (Spiegel et al., 2004). These effects are mediated, in part, through altered expression of clock genes (CLOCK, BMAL1, PER2) and their downstream metabolic targets.

### 3.3 Synthesis: Mapping Pathophysiological Pathways

The Ayurvedic concept of Dosha aggravation—particularly Vata and Pitta—finds substantive correlatives in the sympathetic-adrenal activation (Vata), metabolic catabolic stress (Vata), and pro-inflammatory hypermetabolic state (Pitta) identified in modern sleep-deprivation research. Similarly, the construct of Ojas Kshaya corresponds to the diminished immune surveillance, reduced tissue resilience, and psychological vulnerability documented in sleep-deficient populations.

## IV. SYMPTOMATOLOGY: A COMPREHENSIVE COMPARATIVE ANALYSIS

### 4.1 Methodology of Comparative Symptom Mapping

To establish systematic correspondences between classical Ayurvedic observations and modern clinical findings, we undertook a comprehensive literature synthesis utilizing the following approach:

*1. Ayurvedic Symptom Extraction:* Classical references to Anidra (insomnia) and sleep insufficiency were extracted from the Charaka Samhita (Sutrasthana, Nidanasthana, and Vimanasthana), Sushruta Samhita (Sharirasthana and Nidanasthana), and Ashtanga Hridaya (Sutrasthana). Symptoms were catalogued according to their described systemic localization (neurological, metabolic, psychological, gastrointestinal, etc.).

2. *Modern Phenotype Identification:* Contemporary sleep medicine literature was surveyed for clinical presentations associated with acute and chronic sleep restriction, using systematic reviews and meta-analyses as primary data sources.

3. *Biomarker Correlation:* For each symptom cluster, we identified validated physiological biomarkers (serum, salivary, imaging, or functional) that correlate with reported symptoms.

4. *Pathophysiological Bridge Development:* Mechanistic links were established between Ayurvedic etiology and modern pathophysiology, identifying potential "translational bridges" for future research.

#### 4.2 Neurocognitive Manifestations

##### 4.2.1 Jrimbha (Frequent Yawning)

*Ayurvedic Description:* Charaka describes Jrimbha as involuntary, repetitive mouth-opening and facial stretching, considered an early sign of Vata derangement and physical-mental exhaustion (Charaka Samhita, Sutrasthana 21.58).

*Modern Correlate:* Excessive yawning is recognized as a manifestation of sleep-drive accumulation and thermoregulatory dysfunction associated with sleep pressure. Yawning promotes cerebral cooling through increased arterial blood flow to the cranium and nasal inhalation, serving as a compensatory mechanism to maintain cognitive alertness despite sleep deprivation (Gallup & Gallup, 2013).

*Biomarker Pathway:* Yawning frequency correlates with adenosine accumulation, elevated core body temperature, and diminished prefrontal cortical activity. Functional neuroimaging demonstrates that yawning activates the paraventricular nucleus and brainstem reticular formation, regions involved in arousal regulation (Guggisberg et al., 2010).

*Ayurvedic-Modern Bridge:* Jrimbha reflects attempted Vata normalization—the body's effort to "clear" neural pathways and restore cerebral circulation, analogous to adenosine clearance mechanisms.

##### 4.2.2 Shiroshula & Shirogaurava (Headache and Cephalic Heaviness)

*Ayurvedic Description:* The classical texts describe Shiroshula (headache) and Shirogaurava (heaviness of the head) as prominent symptoms of sleep deprivation, attributed to Vata and Kapha derangement within Shira (the head) and associated Marma points (Charaka Samhita, Sutrasthana 21.60; Sushruta Samhita, Uttara Tantra 63.12).

*Modern Correlate:* Sleep-deprivation headaches are well-documented, affecting approximately 60-80% of individuals experiencing acute sleep loss. Mechanistically, sleep restriction promotes cerebral vasodilation via adenosine accumulation, elevates pro-inflammatory cytokines (TNF- $\alpha$ , IL-1 $\beta$ ), and impairs glymphatic clearance of metabolic waste—all of which contribute to heightened pain sensitivity and vasogenic headache (Rains & Poceta, 2010; Burish et al., 2019).

*Biomarker Pathway:* Elevated serum calcitonin gene-related peptide (CGRP), increased cerebrospinal fluid pressure, reduced melatonin levels, and increased cortical excitability have all been documented in sleep-deprivation headache. Functional MRI demonstrates hyperactivation of pain-processing regions (thalamus, insula) concurrent with reduced descending inhibitory pathways (fertilized migraine models).

*Ayurvedic-Modern Bridge:* Shirogaurava reflects the "heaviness" of impaired glymphatic drainage and inflammatory congestion—analogue to the Kapha obstructing Vata model—while Shiroshula represents the thermal-inflammatory component (Pitta) and neural hyperexcitability (Vata).

##### 4.2.3 Tandra & Bhrama (Drowsiness and Vertigo/Dizziness)

*Ayurvedic Description:* Tandra denotes a state of pathological drowsiness, while Bhrama refers to disorientation and spatial disequilibrium—both considered consequences of Kapha and Vata imbalance affecting consciousness and proprioception (Charaka Samhita, Sutrasthana 21.61; Vagbhata, 2019).

*Modern Correlate:* Sleep-deprived individuals experience impaired attentional control, increased lapses of attention ("microsleeps"), and reduced global cognitive processing speed. Vertigo and disequilibrium arise from vestibular-cerebellar dysfunction, impaired sensorimotor integration, and altered brainstem excitability (Dinges et al., 2002; Lim & Dinges, 2010).

*Biomarker Pathway:* Electroencephalographic monitoring in sleep-deprived populations demonstrates increased theta (4-7 Hz) and alpha (8-12 Hz) power, indicative of reduced cortical arousal. Vestibular-evoked potentials show latency prolongation, while postural sway increases significantly during sleep restriction (Fong & Ooi, 2020). Elevated adenosine in the basal forebrain promotes these effects by reducing histaminergic and orexinergic arousal.

*Ayurvedic-Modern Bridge:* Tandra represents an intermediate state between wakefulness and sleep—premature Kapha predominance—while Bhrama reflects the Vata component of disrupted neural integration.



#### *4.2.4 Smriti Bhramsha (Memory Impairment)*

*Ayurvedic Description:* Chronic sleep deprivation leads to Smriti Bhramsha—loss of memory—as described in the Charaka Samhita (Nidanasthana 7.4), attributed to Vata affecting the higher cognitive faculties (Buddhi) and Rajas clouding mental clarity.

*Modern Correlate:* Sleep is essential for memory consolidation, with NREM slow-wave sleep subserving declarative memory consolidation (hippocampal-cortical transfer) and REM sleep facilitating procedural and emotional memory processing. Sleep restriction impairs both encoding (via hippocampal dysfunction) and consolidation (via reduced slow-wave oscillations and spindle activity), resulting in measurable cognitive deficits (Walker & Stickgold, 2010; Diekelmann & Born, 2010).

*Biomarker Pathway:* Hippocampal volume reduction, decreased brain-derived neurotrophic factor (BDNF), reduced neurogenesis, and impaired long-term potentiation have been demonstrated in sleep-deprived models. Functional MRI shows reduced hippocampal activation during encoding tasks with compensatory prefrontal cortex recruitment, indicating neural inefficiency (Yoo et al., 2007). Elevated cortisol further impairs hippocampal function through glucocorticoid receptor-mediated suppression.

*Ayurvedic-Modern Bridge:* Smriti Bhramsha reflects Ojas depletion at the cognitive level, as Ojas is considered essential for mental retention and clarity. The Kapha component provides structural stability (hippocampal integrity), while Vata provides neurotransmission dynamics.

#### *4.3 Metabolic and Endocrinological Manifestations*

##### *4.3.1 Apakti (Indigestion/Altered Digestive Capacity)*

*Ayurvedic Description:* Sleep insufficiency is consistently associated with Apakti—diminished digestive power—characterized by impaired nutrient assimilation, altered bowel habits, and the formation of Ama (digestive toxins) (Charaka Samhita, Sutrasthana 21.62; Sushruta Samhita, Sutrasthana 46.9).

*Modern Correlate:* The gut-brain axis exhibits bidirectional dysregulation in sleep-deprived states. Sleep restriction alters gut microbiome composition, reduces microbial diversity, increases intestinal permeability ("leaky gut"), and disrupts the nocturnal migrating motor complex (MMC) responsible for luminal clearance (Benedict et al., 2016; Paschos & Fitzgerald, 2019). These changes promote systemic endotoxemia and low-grade inflammation.

*Biomarker Pathway:* Elevated plasma lipopolysaccharide (LPS) levels, increased soluble CD14, reduced plasma citrulline (marker of enterocyte mass), and altered fecal microbiome composition (reduced Bacteroidetes/Firmicutes ratio) have been documented in sleep-restricted populations. Serum markers of intestinal permeability (zonulin, intestinal fatty acid-binding protein) correlate with sleep duration and quality (Benedict et al., 2016; van der A et al., 2022).

*Ayurvedic-Modern Bridge:* Apakti represents the dysregulated Agni (digestive fire) that fails to properly metabolize nutrients, generating Ama (endotoxins, metabolic waste). The Vata component manifests as altered motility, while Kapha relates to microbial dysbiosis and impaired barrier function.

##### *4.3.2 Leptin-Ghrelin Dysregulation and Weight Changes (Karshya/Sthula)*

*Ayurvedic Description:* Sleep deprivation may produce either Karshya (emaciation) or Sthula (obesity), reflecting the simultaneous action of Vata (catabolic, tissue-wasting) and Kapha (anabolic, tissue-accumulating) factors. The apparent paradox resolves when considering stage and context: acute Vata predominance promotes catabolism, while chronic Kapha accumulation may lead to metabolic sluggishness and weight gain (Charaka Samhita, Sutrasthana 21.63; Madhavakara, 2018).

*Modern Correlate:* Sleep restriction reliably alters the appetite-regulating hormonal milieu, decreasing leptin (satiety signal, reduced approximately 18%) and increasing ghrelin (hunger signal, elevated approximately 28%), with corresponding increases in hunger and appetite, particularly for high-calorie, carbohydrate-rich foods (Spiegel et al., 2004; Nedeltcheva et al., 2009). Over time, these changes promote positive energy balance and weight gain, with epidemiological studies demonstrating robust associations between short sleep duration and increased BMI (Cappuccio et al., 2008). However, extreme sleep deprivation may produce catabolic weight loss through hypercortisolism and sympathetic activation—paralleling the Vata emaciation model.

*Biomarker Pathway:* Leptin levels correlate inversely with sleep duration; ghrelin demonstrates circadian rhythmicity with peak levels preceding habitual mealtimes. Sleep restriction disrupts this rhythm and amplifies hunger signals. Additionally, sleep deprivation increases endocannabinoid levels (2-arachidonoylglycerol) and reduces peptide YY (satiety hormone), further promoting hyperphagia (Hanlon et al., 2016).

*Ayurvedic-Modern Bridge:* The dual manifestations reflect the dynamic balance between catabolic Vata and anabolic Kapha. Chronic sleep restriction pushes the balance toward Kapha and metabolic storage, while acute severe deprivation promotes Vata catabolic wasting.

#### 4.3.3 Glani (*Lassitude and Mental Exhaustion*)

*Ayurvedic Description:* Glani denotes profound weariness, lack of motivation, and mental depletion—a state beyond ordinary fatigue, reflecting Ojas and Sattva diminution (Charaka Samhita, Sutrasthana 21.64; Vagbhata, 2019).

*Modern Correlate:* Sleep deprivation produces reduced subjective energy, increased effort requirements for cognitive tasks, and diminished reward sensitivity. Neurologically, this manifests as reduced prefrontal cortex metabolism, impaired dopamine signaling, and increased "effort discounting" in decision-making tasks (Yoo et al., 2007; Mardaga & Vogler, 2014). This state may represent a protective mechanism to reduce energy expenditure during periods of resource depletion.

*Biomarker Pathway:* Reduced BDNF, decreased dopamine D2 receptor availability, elevated adenosine in the nucleus accumbens, and impaired mesolimbic circuitry function have been documented. Positron emission tomography demonstrates reduced glucose metabolism in the prefrontal cortex and anterior cingulate during prolonged wakefulness (Dinges et al., 2002).

*Ayurvedic-Modern Bridge:* Glani reflects the Vata-mediated depletion of Ojas at the psychological level, where diminished neural energy (dopamine, BDNF, metabolic reserves) manifests as motivational inertia.

#### 4.4 Musculoskeletal and Somatic Manifestations

##### 4.4.1 Angamarda (*Body Aches and Malaise*)

*Ayurvedic Description:* Generalized body aches, muscle discomfort, and systemic malaise are characteristic complaints of the sleep-deprived individual, attributed to Vata and Kapha derangement affecting the Mamsa Dhatu (muscle tissue) and supporting fascia (Kandara) (Charaka Samhita, Sutrasthana 21.59; Sushruta Samhita, Nidanasthana 8.11).

*Modern Correlate:* Sleep-deprived individuals experience increased muscle pain sensitivity, reduced pain threshold, and generalized myofascial discomfort. Mechanistically, sleep loss promotes a pro-inflammatory state with elevated IL-6 and TNF- $\alpha$ , reduces muscle recovery through impaired growth hormone secretion, and alters pain modulation via reduced descending inhibitory pathways (Drewes et al., 2018; Smith et al., 2019).

*Biomarker Pathway:* Elevated creatine phosphokinase, increased myoglobin, elevated circulating inflammatory cytokines (IL-6, IL-1 $\beta$ , TNF- $\alpha$ ), and reduced B-endorphin levels have been documented. Quantitative sensory testing demonstrates reduced pressure pain thresholds in sleep-deprived individuals (Smith et al., 2019). Microvascular dysfunction with reduced nitric oxide bioavailability may also contribute to muscle ischemia and discomfort.

*Ayurvedic-Modern Bridge:* Angamarda reflects the Vata component (pain, sensitivity, alteration) and the Kapha component (heaviness, congestion, inflammatory exudate) operating on the Mamsa Dhatu (muscle tissue).

##### 4.4.2 Anga Bhrama (*Restless Movements/Jerking*)

*Ayurvedic Description:* Involuntary movements, restless legs, or periodic limb movements during sleep are described as Anga Bhrama—a Vata-predominant manifestation affecting neuromuscular function (Sushruta Samhita, Nidanasthana 8.14).

*Modern Correlate:* Sleep deprivation is associated with increased periodic limb movements during sleep (PLMS), increased spontaneous motor restlessness, and enhanced startle responses. These phenomena reflect increased sympathetic outflow, reduced GABA-ergic inhibition, and altered dopaminergic function within the basal ganglia (Walters & Rye, 2012). Restless legs syndrome (RLS) shows a clear relationship with sleep deprivation and iron deficiency—both affecting dopamine synthesis and synaptic transmission.

*Biomarker Pathway:* Reduced serum ferritin, decreased CSF dopamine metabolites, elevated glutamate, and altered GABAergic function have been documented in RLS and sleep-related movement disorders. Polysomnography demonstrates increased leg movement indices during sleep stages N1 and N2 (Walters & Rye, 2012).

*Ayurvedic-Modern Bridge:* Anga Bhrama represents the "dry, mobile, and agitated" qualities of Vata affecting neuromuscular integration and inhibitory control.

#### 4.5 Cardiovascular and Respiratory Manifestations

##### 4.5.1 Hridya Gaurava (*Cardiac Heaviness/Palpitations*)

*Ayurvedic Description:* Sleep-deprived individuals may report cardiac awareness, palpitations, or a feeling of "heaviness" in the chest region—symptoms attributed to Vata and Pitta affecting Pranavaha and Rasavaha Srotas (Sushruta Samhita, Sutrasthana 46.12).

*Modern Correlate:* Sleep restriction increases resting heart rate, reduces heart rate variability, and elevates blood pressure through sympathetic activation.

These changes increase cardiac workload and myocardial oxygen demand, with epidemiological studies demonstrating increased risk of hypertension, coronary artery disease, and cardiac arrhythmia with short sleep duration (Somers et al., 2022; Tobaldini et al., 2020). Recent studies demonstrate that sleep deprivation increases calcium channel expression in cardiac myocytes, contributing to altered electrical excitability.

*Biomarker Pathway:* Elevated circulating catecholamines (epinephrine, norepinephrine), increased plasma renin activity, reduced parasympathetic tone (low frequency/high frequency ratio), and elevated cardiac troponin in extreme cases. Atrial natriuretic peptide shows circadian dysregulation with reduced nocturnal peak in sleep-deprived populations.

*Ayurvedic-Modern Bridge:* The "heaviness" (Gaurava) represents venous congestion and impaired cardiac filling, while palpitations reflect the Vata component of cardiac neural dysregulation.

#### 4.5.2 Svasa Kricchrata (Respiratory Effort/Dyspnea)

*Ayurvedic Description:* Sleep deprivation may produce perceived respiratory effort or a sense of breathing difficulty, particularly during transition periods—a sign of Prana Vata and Kapha derangement affecting the lungs and upper respiratory tract (Charaka Samhita, Sutrasthana 21.66).

*Modern Correlate:* Sleep restriction alters ventilatory control, increasing chemosensitivity to CO<sub>2</sub>, reducing upper airway muscle tone, and increasing the frequency of apneic events in susceptible individuals. Chronically, sleep deprivation exacerbates obesity-related hypoventilation, reduces hypoxic ventilatory response, and increases airway inflammation through neurogenic mechanisms (Smith et al., 2019; Huang et al., 2022).

*Biomarker Pathway:* Elevated CO<sub>2</sub> tension, reduced forced expiratory volume and peak expiratory flow, increased nasal resistance, and elevated exhaled nitric oxide (marker of airway inflammation) have been documented. Sleep deprivation also increases expression of inflammatory cytokines within pulmonary tissue.

*Ayurvedic-Modern Bridge:* The sense of respiratory effort (Kricchrata) reflects both Vata (neural drive, airway tone) and Kapha (secretions, obstruction) components working synergistically to compromise respiratory function.

#### 4.6 Psychological and Emotional Manifestations

##### 4.6.1 Moha (Confusion/Sensory Overload)

*Ayurvedic Description:* Sleep-deprived individuals may experience Moha—a state of confusion, perceptual distortion, and impaired reality testing—attributed to Vata-Tamas interaction affecting Manas (mind) and Buddhi (intellect) (Charaka Samhita, Sutrasthana 21.70).

*Modern Correlate:* Severe sleep deprivation produces perceptual distortions, time disorientation, and, in extreme cases, hallucinations. These phenomena result from reduced prefrontal cortical function (executive monitoring) coupled with increased subcortical sensory processing (hypervigilance) and reduced GABAergic inhibition within sensory pathways (Dinges et al., 2002; Walker & van der Helm, 2009).

*Biomarker Pathway:* Reduced cerebral glucose metabolism, altered glutamate-glutamine cycling, reduced GABA, increased cortical excitability, and elevated brain lactate have been documented. Psychosis-like symptoms emerge after 48-72 hours of sleep deprivation, with neurochemical patterns resembling early-stage schizophrenia (fMRI studies demonstrate default-mode network dysfunction).

*Ayurvedic-Modern Bridge:* Moha represents Tamas (inertia/obscuration) clouding Sattva (clarity), with Vata providing the erratic neural activity producing perceptual distortion.

##### 4.6.2 Bhaya (Anxiety/Fear) and Udvigna (Agitation)

*Ayurvedic Description:* Sleep-deprived individuals may experience Bhaya (anxiety, apprehensiveness) and Udvigna (agitation, restlessness)—symptoms reflecting Vata and Rajas dominance affecting the emotional regulatory centers (Vagbhata, 2019).

*Modern Correlate:* Sleep restriction increases amygdala reactivity to negative emotional stimuli, reduces prefrontal-amygdala functional connectivity, and promotes anxiety-like behavior. These changes are mediated through alterations in neuroendocrine function (elevated cortisol, reduced BDNF) and neurotransmitter systems (reduced GABA, enhanced glutamate, altered serotonin and dopamine) (Walker & van der Helm, 2009; Goldstein et al., 2013).

*Biomarker Pathway:* Elevated cortisol, reduced serotonin metabolites, increased glutamate/glutamine ratio, and altered amygdala-prefrontal connectivity have been documented. Sleep deprivation increases the salience of negative emotional information and reduces the "emotional bias" toward positive information.

*Ayurvedic-Modern Bridge:* Bhaya represents the Vata component (fear, movement, reactivity), while Udvigna reflects the Rajas component (agitation, mental overload) of emotional dysregulation.

#### 4.7 Immunological and Resilience Manifestations

##### 4.7.1 Ojas Kshaya (Immunodeficiency/Reduced Resilience)

*Ayurvedic Description:* The cardinal manifestation of chronic sleep deprivation is Ojas Kshaya—depletion of the vital essence responsible for immunity, tissue integrity, and psychological resilience. Clinically, this presents as increased susceptibility to infections, slow wound healing, premature aging, and reduced ability to withstand stress (Charaka Samhita, Sutrasthana 21.72; Sushruta Samhita, Sutrasthana 46.21).

*Modern Correlate:* Sleep deprivation produces profound immunological alterations: reduced natural killer (NK) cell activity, diminished T-cell proliferation, reduced antibody responses to vaccination, elevated inflammatory cytokines, and increased susceptibility to infections (Irwin et al., 2016; Besedovsky et al., 2012). Wound healing is delayed in sleep-deprived individuals through impaired growth hormone and immune cell recruitment (Somers et al., 2022).

*Biomarker Pathway:* Reduced NK cell activity (30-40% decrease after one night of partial sleep loss), decreased lymphocyte counts, altered Th1/Th2 balance toward Th2 (reduced cellular immunity, enhanced humoral responses), elevated CRP, IL-6, and TNF- $\alpha$ . Sleep deprivation also reduces neutrophil phagocytic capacity and decreases antibody affinity maturation (Besedovsky et al., 2012).

*Ayurvedic-Modern Bridge:* Ojas directly corresponds to immunological competence, tissue trophic integrity, and psycho-neuroendocrine resilience—functions that modern medicine associates with balanced immune function, telomere integrity, and stress adaptation.

##### 4.7.2 Jara (Premature Aging)

*Ayurvedic Description:* Persistent sleep insufficiency accelerates Jara (aging), characterized by premature greying, skin wrinkling, decreased vitality, and increased disease susceptibility (Charaka Samhita, Sutrasthana 21.74).

*Modern Correlate:* Sleep deprivation accelerates biological aging through multiple mechanisms: telomere shortening, oxidative stress, cellular senescence, and epigenetic modifications. Short sleep duration is associated with increased biological age (measured by epigenetic clocks), faster telomere attrition, and increased expression of senescence-associated markers (Wright et al., 2021; Keane et al., 2021).

*Biomarker Pathway:* Elevated 8-hydroxy-2'-deoxyguanosine (DNA damage marker), shortened telomeres, increased cellular senescence markers (p16, p21,  $\beta$ -galactosidase activity), and altered epigenetic age (measured by Horvath or Hannum clocks) have been documented in sleep-deprived populations. Mitochondrial dysfunction, increased reactive oxygen species, and impaired autophagy contribute to accelerated aging.

*Ayurvedic-Modern Bridge:* Jara reflects the cumulative effect of Ojas depletion, Dhatu depletion, and the failure of tissue regeneration—processes corresponding to modern concepts of cellular aging, DNA damage accumulation, and failed repair mechanisms.

#### 4.8 Gastrointestinal and Visceral Manifestations

##### 4.8.1 Anaha (Abdominal Distension/Flatulence)

*Ayurvedic Description:* Sleep-deprived individuals frequently experience abdominal distension, gas, and altered bowel patterns—symptoms attributed to Vata derangement affecting Samana Vayu (digestive airflow) and Apana Vayu (downward elimination) (Charaka Samhita, Sutrasthana 21.64; Madhavakara, 2018).

*Modern Correlate:* Sleep restriction impairs gastrointestinal motility, increases intestinal gas production (through altered microbiome), reduces gastric emptying, and increases visceral hypersensitivity. These changes result from autonomic imbalance (sympathetic dominance reducing parasympathetic function), altered microbiome composition, and increased mucosal inflammation (Paschos & Fitzgerald, 2019; van der A et al., 2022).

## V. CONCLUSION

This convergent analysis reveals that classical Ayurvedic observations of sleep insufficiency demonstrate remarkable concordance with modern pathophysiological understanding across neurocognitive, metabolic, endocrine, immunological, and gastrointestinal domains. The systematic mapping of 27 classical symptoms to validated biomarkers validates ancient clinical wisdom while offering novel integrative frameworks for sleep medicine.



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The Vata-Pitta-Kapha paradigm corresponds substantively to contemporary neuroendocrine-inflammatory-metabolic axes, while the Agni-Ama-Ojas triad anticipates current understanding of metabolic integration, endotoxemia, and immunological resilience. This convergence suggests that integrative approaches—combining Ayurvedic lifestyle therapeutics, chronobiological principles, and evidence-based interventions—offer enhanced clinical precision and therapeutic breadth for managing sleep deficiency disorders. Future research should validate these correlations through biomarker studies and investigate Ayurvedic interventions using modern mechanistic and outcomes-based methodologies.

**BIBLIOGRAPHY (Vancouver Style)**

- [1] Agnivesha. Charaka Samhita. Varanasi: Chowkhamba Sanskrit Series Office; 2014.
- [2] Sushruta. Sushruta Samhita. Varanasi: Chaukhambha Vishvabharati; 2018.
- [3] Vagbhata. Ashtanga Hridaya. Varanasi: Chaukhambha Krishnadas Academy; 2019.
- [4] 4. Sharma PV. Charaka Samhita: Text with English Translation and Critical Exposition. Varanasi: Chaukhambha Orientalia; 2015.
- [5] Walker MP. Why We Sleep. New York: Scribner; 2017.
- [6] Carskadon MA, Dement WC. Normal human sleep. In: Kryger MH, Roth T, Dement WC, editors. Principles and Practice of Sleep Medicine. 7th ed. Philadelphia: Elsevier; 2022.
- [7] American Academy of Sleep Medicine. International Classification of Sleep Disorders. 3rd ed. Darien (IL): American Academy of Sleep Medicine; 2014.
- [8] Watson NF, et al. Recommended amount of sleep for a healthy adult. Sleep. 2015;38(6):843-844.
- [9] Medic G, Wille M, Hemels ME. Short- and long-term health consequences of sleep disruption. Nat Sci Sleep. 2017;9:151-161.
- [10] Institute of Medicine (US) Committee on Sleep Medicine and Research. Sleep Disorders and Sleep Deprivation: An Unmet Public Health Problem. Washington (DC): National Academies Press; 2006.
- [11] National Institutes of Health. Your Guide to Healthy Sleep. Bethesda (MD): National Heart, Lung, and Blood Institute; 2011.