RESEARCH PAPER

Insomnia, Sleep Architecture, and Taila Dhara: A Narrative Review of Neurophysiological and Polysomnographic Perspectives

Dr. Mayank Trikha[1], Dr Harshita Kaliya [2], Dr. Vishwanath S Wasedar [3], Dr. Poornima T[4], Dr. Vaishnavi Basaveshwar Bhogade[5], Dr. Chesta[6], Dr. Jyoti Lakhlan[7]

[1]PG Scholar, Dept of Panchakarma, KAHER's SHRI BMK Ayurveda Mahavidyalaya College and Research centre, Belagavi, Karnataka

[2]PG Scholar, Dept of Panchakarma, KAHER's SHRI BMK Ayurveda Mahavidyalaya College and Research centre, Belagavi, Karnataka. [3]Professor, Dept of Panchakarma, KAHER's SHRI BMK Ayurveda Mahavidyalaya College and Research centre, Belagavi, Karnataka.[4]Assistant Professor, Dept of Rasa Shastra evam Bhaisajya Kalpana, KAHER's SHRI BMK Ayurveda Mahavidyalaya College and Research centre, Belagavi, Karnataka.

[5]PG Scholar, Dept of Panchakarma, KAHER's SHRI BMK Ayurveda Mahavidyalaya College and Research centre, Belagavi, Karnataka.

[6]PG Scholar, Dept of Prasuti tantra evam Stree roga, KAHER's SHRI BMK Ayurveda Mahavidyalaya College and Research centre, Belagavi, Karnataka

[7]PG Scholar, Dept of Panchakarma, KAHER's SHRI BMK Ayurveda Mahavidyalaya College and Research centre, Belagavi, Karnataka.

***Corresponding authors-** Dr. Vishwanath S Wasedar: svishwa_w@yahoo.co.in; mayanktrikha28@gmail.com*

ABSTRACT

Chronic insomnia is characterized by objectively measurable disruptions in sleep macro-architecture, including prolonged sleep latency, increased wakefulness after sleep onset, reduced total sleep time and sleep efficiency, and a disproportionate shift toward lighter non-REM stages. Pathophysiologically, the condition reflects a state of central nervous system hyperarousal driven by dysregulated hypothalamic-pituitary-adrenal (HPA) axis activity — specifically, elevated circulating cortisol and adrenocorticotropic hormone (ACTH) — and heightened sympathetic autonomic tone. Traditional Ayurvedic literature regards Nidra (sleep) as one of the three foundational pillars of life (Upastambha), with insomnia (Nidranasha or Anidra) attributed predominantly to Vata dosha aggravation. Classical Panchakarma procedures, notably Taila Dhara (medicated oil pouring) and Takra Dhara (medicated buttermilk pouring), both administered as Shirodhara variants, are prescribed for the management of Anidra. Emerging evidence from clinical trials, case series, and case studies suggests that standardized Taila Dhara administered over 7–21 days may improve both subjective and objective sleep parameters. One well-designed, three-arm RCT of elderly insomniacs demonstrated that 21-day Shirodhara with sesame oil and oral Ashwagandha supplementation — individually and in combination — each significantly improved polysomnographic sleep latency and sleep efficiency ($p<0.001$), with the combination arm yielding the greatest gains. Mechanistically, Taila Dhara is postulated to reduce circulating stress hormones (cortisol, norepinephrine), restore parasympathetic dominance, and enhance alpha-band EEG synchrony — collectively reversing the hyperarousal substrate of insomnia. This review synthesizes sleep-stage findings in chronic insomnia, Ayurvedic classical concepts of sleep and its disorders, and current clinical evidence on Taila/Takra Dhara, incorporating a summary evidence table, proposed neurophysiological mechanism flowchart, and recommended polysomnographic endpoints for future rigorous trials.

Keywords: Insomnia, Taila Dhara, Shirodhara, sleep architecture, polysomnography, Ayurveda, neurophysiology.

How to cite this article: Trikha M, Kaliya H, Wasedar VS, Poornima T, Bhogade VB, Chesta, Lakhlan J. Insomnia, Sleep Architecture, and Taila Dhara: A Narrative Review of Neurophysiological and Polysomnographic Perspectives. Int J Drug Deliv Technol. 2026;16(68s):704-711. DOI: 10.25258/ijddt.16.68s.86

Source of support: Nil.

Conflict of interest: None

1. INTRODUCTION

Sleep is a fundamental physiological process essential for physical restoration, cognitive performance, emotional regulation, metabolic homeostasis, and overall health. Adequate sleep contributes significantly to memory consolidation, immune function, endocrine balance, and psychological well-being. Disturbances in sleep quantity or quality may adversely affect multiple organ systems and are increasingly recognized as major public health concerns. [1] Sleep architecture refers to the orderly, cyclical progression of non-REM and REM sleep stages across a night. In healthy adults, the standard distribution is approximately 2-5% in

**Author for Correspondence: Dr. Vishwanath S Wasedar*

Stage N1 (transitional sleep), 45–55% in Stage N2 (light NREM), 20–25% in Stage N3 (slow-wave or deep sleep), and 20–25% in REM sleep. [2] A typical night encompasses four to six 90-minute cycles, with slow-wave N3 predominating during early cycles and REM sleep becoming progressively longer toward morning. Adequate N3 and REM sleep are indispensable for physical restoration, memory consolidation, and emotional regulation. [3] [Figure 1 – Normal adult sleep stage distribution]

Insomnia is the most prevalent sleep disorder worldwide and is characterized by persistent difficulty initiating sleep, maintaining sleep, or achieving restorative sleep despite adequate opportunity for sleep. According to the International Classification of Sleep Disorders (ICSD-3), chronic insomnia disorder is defined by sleep-related complaints occurring at least three times per week for a minimum duration of three months and resulting in clinically significant daytime impairment. Epidemiological studies suggest that approximately 10–15% of adults experience chronic insomnia, while transient insomnia symptoms may affect up to one-third of the general population.[4]

Polysomnography (PSG), the gold standard for objective sleep assessment, has demonstrated characteristic alterations in sleep architecture among individuals with chronic insomnia. These include prolonged sleep onset latency (SOL), increased wake after sleep onset (WASO), reduced total sleep time (TST) and sleep efficiency (SE), and a redistribution of sleep toward lighter NREM stages. Contemporary neurobiological models suggest that these changes are manifestations of a state of persistent hyperarousal involving dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis, autonomic nervous system imbalance, heightened cortical activation, and increased sympathetic nervous system activity. [5]

In a landmark PSG study of 67 patients with chronic insomnia (BaHammam, 2004), mean SOL was 42.6 ± 7.74 minutes, SE was 69.9 ± 2.5%, and the arousal index was 18.8 per hour - parameters substantially worse than normal thresholds. Stage N1 represented approximately 14.6% of total sleep time (roughly three times the normal proportion), while N3 (slow-wave sleep) accounted for only 18.4% and REM for approximately 15%. [6] These findings reflect the cardinal PSG phenotype of insomnia: excess light-stage vigilance and insufficient restorative sleep.

Ayurveda recognizes sleep (Nidra) as one of the three fundamental pillars of life (Trayopastambha), alongside Ahara (diet) and Brahmacharya (regulated conduct). Classical Ayurvedic literature emphasizes the vital role of Nidra in maintaining physical strength, mental stability, longevity, and overall well-being. Disturbance of sleep, referred to as Nidranasha or Anidra, is primarily attributed to aggravation of Vata Dosha, often accompanied by disturbances of Pitta and mental factors such as Chinta (worry), Bhaya (fear), and excessive cognitive activity. Management strategies described in Ayurvedic texts include lifestyle modifications, dietary interventions, Medhya Rasayana therapies, and Panchakarma procedures aimed at restoring physiological and psychological balance. [7]

Among Panchakarma-based interventions, Shirodhara (Taila Dhara) and Takradhara have gained considerable attention for their potential role in stress reduction and sleep promotion. Shirodhara involves the continuous pouring of medicated oil over the forehead, whereas Takradhara employs medicated buttermilk as the therapeutic medium. Traditionally, these therapies are believed to pacify aggravated Vata, calm the mind, and promote relaxation. Emerging clinical evidence suggests that Dhara therapies may improve subjective sleep quality and selected objective sleep parameters, potentially through modulation of autonomic function and stress-related neuroendocrine pathways. [8]

Despite growing interest in Ayurvedic interventions for insomnia, the relationship between Taila Dhara and objective alterations in sleep architecture remains insufficiently explored. Furthermore, available evidence is scattered across clinical studies, case reports, and narrative reviews, limiting comprehensive understanding of its potential mechanisms and therapeutic implications.

Therefore, the present narrative review aims to critically examine alterations in sleep architecture associated with chronic insomnia, explore contemporary understanding of insomnia pathophysiology, review Ayurvedic concepts of Nidra and Nidranasha, and evaluate available evidence regarding the effects of Taila Dhara and Takradhara on sleep-related outcomes. Additionally, potential neurophysiological mechanisms and future directions for polysomnographic research in this area are discussed.

Pathophysiology of Insomnia

The prevailing neurobiological model frames insomnia as a disorder of sustained hyperarousal operating across cognitive, neuroendocrine, and autonomic domains, rather than just a primary sleep deficit per se. At the neuroendocrine level, chronic insomnia is associated with a round-the-clock increase in ACTH and cortisol secretion, consistent with persistent HPA axis dysregulation. Since deep NREM sleep normally exerts an inhibitory influence on HPA activity, the failure to suppress corticotropic drive in insomnia creates a self-perpetuating arousal loop. Nicolaides et al. (2020) in Endotext note that insomnia is associated with a 24-hour increase of ACTH and cortisol secretion — a finding consistent with a disorder of central nervous system hyperarousal rather than a discrete nocturnal abnormality.[9] Conversely, somatotropic axis function is blunted: growth hormone, which depends on slow-wave sleep for its principal secretory burst, is reduced in insomnia patients with objectively short sleep.

Autonomically, insomnia resembles a persistent nocturnal sympathetic activation. Insomniacs demonstrate higher night-time heart rates, reduced overall heart rate variability (particularly in the high-frequency parasympathetic band), and an elevated LF/HF ratio — even during N2 sleep. Jerath et al. (2019) propose an 'evolutionary mismatch' model

wherein modern psychosocial stressors generate chronic sympathetic overdrive that intrudes into sleep onset and maintenance, operationally lengthening SOL and increasing arousal frequency.[10] This autonomic model converges with clinical evidence that interventions augmenting vagal tone - paced breathing, mindfulness, certain Panchakarma procedures - can measurably improve insomnia parameters.

From an Ayurvedic perspective, psychological factors (Manasika Bhavas) such as excessive worry (Chinta), overthinking (Atichintan), and emotional agitation directly vitiate Vata dosha and secondarily disturb Pitta. Charaka Samhita describes how these manasika provocations disrupt the Rasavaha Srotas, the channel network governing nutrient plasma circulation, and perturb the mind-heart axis (Hridaya-Mana Sambandha), producing Anidra. The dominant doshic classification of insomnia is Vataja, though Pitta-predominant presentations (with mental heat, excessive dreaming) and combined Vata-Pitta patterns are recognized.

2. METHODS

Search Strategy

A targeted narrative literature search was conducted across PubMed, Google Scholar, and Ayurvedic journal repositories - including AYU (Journal of Ayurveda), Ancient Science of Life, and IJAPR - covering publications up to June 2026. Primary search terms included: "insomnia," "Anidra," "Nidranasha," "sleep architecture," "polysomnography," "hyperarousal," "HPA axis sleep," "Shirodhara," "Takradhara," "Taila Dhara," "Ayurvedic sleep therapy," "Nidra Ayurveda". Authoritative classical texts (Charaka Samhita, Sushruta Samhita, Ashtanga Hridayam) were accessed through verified secondary scholarly sources. Study inclusion required: adult insomnia population; intervention with Taila Dhara, Shirodhara, or Takradhara (with or without adjunctive Ayurvedic formulations); and outcomes comprising either PSG-derived parameters or validated insomnia scales (ISI, PSQI, AIS). Case reports, case series, open trials, and RCTs in English were included. Data extracted: study design, sample size, intervention details, PSG measures (SOL, SE, WASO, N3%, REM%), and subjective sleep scores.

3. Sleep Architecture and PSG Findings in Insomnia

Sleep Stage Alterations

Chronic insomnia produces a reliable and characteristic bias toward lighter NREM sleep. In the PSG study of 67 chronic insomnia patients by BaHammam (2004), Stage N1 averaged 14.6% of total sleep time - approximately three times the normal proportion - while deep slow-wave N3 represented only 18.4% and REM approximately 15% [6]. These findings reflect the dual pathology: excess light-stage hypervigilance and insufficient restorative sleep. An important clinical nuance is sleep state misperception (SSM): in the same cohort, approximately 40% of patients had essentially normal PSG but reported severe subjective insomnia. This discordance underscores the limitations of using PSG alone and the critical need to include validated subjective scales (ISI, PSQI) alongside objective measures in any clinical trial design.

Quantitative PSG Metrics

The principal PSG abnormalities in chronic insomnia include: (a) SOL exceeding 30 minutes, (b) SE falling below 75–80%, (c) elevated WASO, (d) reduced TST, (e) disproportionately high %N1 and low %N3/REM, and (f) an elevated arousal index. Alpha-delta sleep - the intrusion of alpha (8-12 Hz) activity into slow-wave sleep - is an EEG-level correlate of insomnia-related hyperarousal and is associated with subjective reports of unrefreshing sleep even when gross sleep stage proportions appear near-normal. Elevated beta (15–30 Hz) power across all sleep stages, including NREM, is a more recent and robust EEG biomarker of the cortical hyperarousal underpinning insomnia [11]. These EEG markers may serve as objective therapeutic targets for future Shirodhara-EEG studies.

Pathophysiology: Hyperarousal, HPA Axis, and Autonomic Dysregulation

Insomnia's core pathophysiology is central nervous system hyperarousal. Patients exhibit elevated daytime and night-time cortical beta activation on EEG, a higher cerebral metabolic rate, and sustained neuroendocrine stress signaling. As documented by Nicolaides et al. (2020) in Endotext, ACTH and cortisol are elevated across the full 24-hour period in insomnia - not merely at night - reflecting a failure of sleep to suppress HPA activity. The correlation between the area under the cortisol secretory curve and sleep efficiency is reported to be as high as $r = 0.91$ in insomniacs with objectively short sleep duration [12], highlighting cortisol as both a biomarker and a mechanistic driver of sleep disruption. Autonomically, insomnia resembles a persistent nocturnal fight-or-flight state. Multiple studies document higher sympathetic (LF) and lower parasympathetic (HF) HRV components during all sleep stages in insomnia. Day-night differences in heart rate and autonomic tone - normally pronounced in healthy sleepers - are attenuated in insomniacs. Jerath et al. (2019) term this the 'dysevolution hypothesis': modern environmental stimuli chronically activate the sympathetic axis, producing autonomic hyperarousal that prevents sleep onset and fragments sleep continuity [10]. Any therapeutic strategy that reliably augments parasympathetic (vagal) tone thus holds mechanistic rationale for insomnia.

Ayurvedic Concepts:

Nidra and Nidranasha

Charaka describes eight varieties of Nidra based on physiological, pathological, and psychological causative factors.[13] Nidranasha is predominantly considered a Vata-predominant condition.[14]. From the perspective of Vata subtypes, dysfunction of Prana Vata, which governs higher mental functions and sensory integration, and Vyana Vata, which regulates movement and systemic circulation, may contribute to disturbed sleep [15], [16]. In certain patients, concomitant Pitta aggravation, due to its ushana, teekshana gunas may be manifested by increased mental activity, irritability, and vivid dreaming may further exacerbate insomnia symptoms [17], [18].

Acharya Charaka describes Atichintana, Chinta, Shoka, and Bhaya as important causative factors that provoke Vata dosha and disturb the normal physiology of sleep. [19,20] These manasika nidanas induce psychological stress and may disrupt the functional integrity of the Rasavaha Srotas and the Hridaya, which is regarded as one of the principal seats of consciousness and mental activities in Ayurveda.[21],[22], [23]. Furthermore, Ayurveda considers sleep to be facilitated by the predominance of Tamo guna [24]; therefore, persistent psychological perturbations and Vata aggravation may interfere with the natural predominance of Tamas required for sleep initiation and maintenance, resulting in Anidra. [25]

The Ayurvedic classification thus parallels modern psychiatric understanding of insomnia as a cognitive-emotional hyperarousal disorder, offering a classical philosophical precedent for psycho-neuro-endocrine intervention models.

Taila Dhara (Shirodhara) and Takradhara

Shirodhara - the continuous pouring of warm medicated liquid onto the forehead from a prescribed height using a specially designed vessel (Dhara Patra) - is among the most widely studied Ayurvedic neurological procedures. Taila Dhara, typically using Tila Taila (sesame oil) or compound formulations such as Narayana Taila, is specifically indicated for Vata-predominant neuropsychiatric conditions including Anidra, Shiroroga, and Manasika Rogas. Takradhara, employing warm buttermilk processed with herbs such as Amalaki (Emblica officinalis), Bala (Sida cordifolia), and Jatamansi (Nardostachys jatamansi), is indicated for Vata-Pitta conditions and has been specifically applied for insomnia. Both procedures share a common rationale: the sustained rhythmic thermal and tactile stimulation of the forehead activates cranial sensory pathways, promoting neurophysiological calming of the Mastishka (brain) and pacifying aggravated Prana Vata.

Shirodhara, a form of *Murdhni Taila*, involves the continuous pouring of a warm medicated liquid over the forehead from a prescribed height using a specially designed vessel (*Dhara Patra*) [26]. It is one of the most extensively investigated Ayurvedic procedures for stress-related, neurological, and neuropsychiatric disorders [27]. *Taila Dhara*, commonly performed using *Tila Taila* (sesame oil) or medicated oils such as *Narayana Taila*, is traditionally indicated in *Vata*-predominant disorders, including *Anidra*, *Shiroroga*, and various *Manasika Rogas* [28], [29]. *Takradhara*, in which medicated buttermilk processed with herbs such as *Amalaki* (*Emblica officinalis*), *Bala* (*Sida cordifolia*), and *Jatamansi* (*Nardostachys jatamansi*) is poured over the forehead, is particularly indicated in conditions characterized by *Vata-Pitta* vitiation and has been employed clinically in the management of insomnia [29][30]. From a contemporary perspective, both procedures may exert their therapeutic effects through sustained rhythmic thermal and tactile stimulation of the forehead, potentially modulating cranial sensory pathways, attenuating autonomic hyperarousal, and promoting psychophysiological relaxation [27][31]. In Ayurvedic terms, these effects may be interpreted as pacification of aggravated *Prana Vata* and restoration of normal *Nidra* physiology[28][32].

Session parameters in clinical practice typically range from 30-60 minutes per session, administered daily for 7 consecutive days. Temperature of the dripping fluid is maintained between 37–42°C (close to body temperature) to optimize sensory receptor stimulation without causing thermal discomfort. The stream is directed to the Sthapani Marma point (between the eyebrows). No standardized protocol exists across studies, and inter-study variation in oil type, temperature, flow rate, session duration, and course length represents a significant methodological limitation for pooled evidence synthesis.

Clinical Evidence: Taila/Takra Dhara for Insomnia

Multiple studies - ranging from case reports to small RCTs - have evaluated taila and takra Dhara for insomnia. The evidence base, while encouraging, remains heterogeneous in design, intervention, and outcome measurement. No study to date has employed a fully blinded, sham-controlled RCT design with PSG as the primary outcome, which constitutes the single most important gap in this literature.

Table 1: Summary of Key Clinical Studies on Taila/Takra Dhara for Insomnia

Study & Year	Design, N	Intervention	Outcomes	Key Findings
Vinjamury et al. 2014 DOI: 10.7453/gahmj.2012.086 [33]	Case series N=10.	Shirodhara with Brahmi oil, 45 min × 5 consecutive days	ISI (Insomnia Severity Index) at baseline, Day 5, and 1-week follow-up.	Mean ISI significantly reduced at Day 5 ($p<0.005$); effect sustained at 1-week follow-up. 6/9 participants improved by 25–70%; 1 droput.
Pawar et al. 2024 Int J Acad Med Pharm 6(4):92–96 [34]	RCT N=63 (3 arms, 21/arm) Elderly insomniacs	Group S: Shirodhara, sesame oil, 45–60 min × 21d Group A:	PSG (SOL, TST, WASO, SE, arousals), PSQI, ISI, ESS -	All three groups: significant ↓SOL, ↓WASO, ↑SE, ↑TST ($p<0.001$). Group SA (combined) showed

Study & Year	Design, N	Intervention	Outcomes	Key Findings
		Ashwagandha 500 mg BD Group SA: Both	baseline vs Day 21	maximal PSG improvements across all parameters.
Rajan et al. 2021 DOI: 10.1016/j.jaim.2021.01.008 PMID: 33637424 [35]	Case report N=1	Shirodhara with warm sesame oil, 45 min daily × 14 days; no internal medication	Serum cortisol, DHEA, POMS (mood), blood pressure, sleep quality.	Significant ↓ serum cortisol and DHEA-S post-Shirodhara; POMS stress domain markedly reduced; BP improved ($p<0.001$); patient reported resolution of insomnia symptoms.
Kumari & Bhatted 2023 Ayushdhara 10(Suppl 4):49–52 [36]	Case series N=7	Tila Taila Shirodhara + adjunctive Ayurvedic medicines (duration not standardized across cases)	Athens Insomnia Scale, Hamilton Anxiety Scale, HAM-D, WHOQOL-BREF, Anidra Lakshanas	Overall improvement ~74.9% across insomnia, anxiety, and depression scores. Reported as safe and efficacious. Series size not explicitly reported; results should be interpreted cautiously. Athens,insomnia Scale there was a percentage improvement of 88.6%. Hamilton's Anxiety Scale percentage improvement of 73.8%. Hamilton's Depression Scale percentage improvement of 82.6%. WHO Quality of Life was also observed to have an overall improvement of 74.2%, 66.1%, 63.8%, and 61.6% in physical, psychological, social relationship, environmental aspects respectively. All the Anidra Lakshanawas seen to decrease significantly with an overall improvement of 88.6%.
Varshitha et al. 2024 [37]	RCT N=40 (2×20)	Group A: Takradhara 7–14d Group B: Narayana Taila Dhara 7–14d (45–60 min/session)	PSQI, symptom checklist (subjective)	Both groups improved PSQI by ~46–49%. Narayana Taila Dhara (Group B) showed marginally superior improvements in sleep duration and quality domains.

Study & Year	Design, N	Intervention	Outcomes	Key Findings
Mohod et al. 2015 [38]	Open trial N=30	Takradhara with herb-processed buttermilk + internal herbal tablets	Symptom-based checklist (sleepiness, fatigue, malaise).	60–73% of patients reported symptomatic relief. Procedure was well tolerated, no adverse events.
Dhuri et al. 2013 DOI: 10.4103/0975-9476.109550 [39]	Psychophysiological profile study N=16 healthy volunteers	Shirodhara with sesame oil, 30 min session; EEG monitoring during procedure	signifi cant (P = 0.002) reduction in the respiratory rate. Mean diastolic blood pressure reduced signifi cantly (P = 0.027), significant (P = 0.0015) drop in the mean pulse rate. EEG showed an increase in the alfa rhythm after Shirodhara. A decrease of beta activity was observed in two subjects, while one subject showed an increase in the central theta activity.	↑ Alpha and theta EEG power during Shirodhara; ↓ GSR; shift toward parasympathetic dominance. Supports neurophysiological mechanism — particularly EEG relaxation effects.

4. DISSCUSSION

Proposed Mechanisms: Neurophysiology of Taila Dhara

1. HPA Axis Modulation

The continuous warm fluid stream on the forehead is hypothesized to engage trigeminal sensory afferents and frontal skin thermoreceptors, transmitting signals to the thalamus and hypothalamus and exerting a dampening effect on the HPA axis. Rajan et al. (2021) directly demonstrated a significant reduction in serum cortisol and DHEA-S following 14 days of Shirodhara ($p<0.001$), alongside POMS mood improvements. This HPA dampening effect is mechanistically significant: Nicolaides et al. (2020) confirm that cortisol AUC correlates inversely with sleep efficiency ($r = 0.91$), so a reliable cortisol-lowering effect by Shirodhara would be expected to directly translate into improved SE and reduced SOL.

2. Autonomic Balance Restoration

Downregulation of HPA axis activity and reduced sympathoadrenal tone would shift the autonomic balance toward parasympathetic dominance. Dhuri et al. (2013) documented decreased galvanic skin response (GSR) — a direct sympathetic arousal marker — and altered EEG activity during Shirodhara in healthy volunteers, providing objective neurophysiological evidence for an acute parasympathetic shift. In clinical insomnia populations, increased parasympathetic tone would be expected to reduce SOL, decrease nocturnal heart rate, and improve sleep continuity. Future studies should include ambulatory 24-hour HRV monitoring as an objective ANS endpoint.

3. EEG and Brain Wave Effects

EEG studies suggest Shirodhara generates a neurophysiological state of relaxed wakefulness. Pilot work (Dhuri et al., 2013) reports increases in alpha (8–12 Hz) and theta (4–7 Hz) power during and immediately following Shirodhara. Enhanced thalamo-cortical alpha synchrony is associated with reduced cortical hyperarousal and facilitates the transition into NREM sleep stages. Concurrently, suppression of beta (15–30 Hz) activity — the EEG signature of insomnia-related cortical hyperarousal identified by Shi et al. (2022) — may be a key outcome to monitor in future Shirodhara-EEG trials. Measuring delta (slow-wave) EEG power within N3 sleep would additionally quantify any restoration of deep sleep quality beyond what macroarchitecture staging captures.

4. Melatonin and Circadian Normalization

No published study to date has directly measured melatonin levels following Shirodhara in an insomnia population — this represents an important mechanistic gap. Indirectly, by reducing nocturnal cortisol (which suppresses melatonin secretion via direct pineal inhibition), Taila Dhara may normalize melatonin profiles. Future mechanistic studies should include serial salivary melatonin measurements (DLMO — dim-light melatonin onset) alongside PSG to confirm this pathway.

5. Psychological Relaxation and Cognitive De-arousal

Beyond direct neurophysiological effects, Dhara therapies reliably produce profound subjective relaxation. Vinjamury et al. (2014) observed significant ISI reductions after just 5 sessions, with participants reporting decreased subjective arousal and sleep-related cognitive activity. Case reports document POMS mood domain improvements across vigour, anxiety, depression, and confusion subscales after Shirodhara. These psychological effects align with the cognitive-emotional hyperarousal model of insomnia (Harvey, 2002), wherein attenuating pre-sleep cognitive arousal is as therapeutically critical as modifying autonomic parameters. Shirodhara may serve as a non-pharmacological cognitive de-arousal intervention analogous to, but phenomenologically distinct from, CBT-I techniques such as sleep restriction and stimulus control. This conceptual bridge warrants formal investigation.

Limitations and Future Directions

Limitations of Current Evidence

The available evidence base is constrained by: small and heterogeneous sample sizes; lack of standardized intervention protocols (oil type, temperature, duration, course length); predominantly subjective outcome measures without PSG, limited follow-up periods..

Future Research Priorities

Priority research directions include: (1) a well-powered, sham-controlled PSG-based RCT of standardized sesame oil Shirodhara in primary insomnia; (2) mechanistic substudies incorporating overnight HRV, serum cortisol/melatonin, and EEG spectral analysis; (3) stratification of outcomes by Prakriti and dominant doshic imbalance to explore personalized Ayurvedic response predictors; (4) longer-term follow-up (3–6 months) to assess durability of sleep architectural changes; and (5) qualitative exploration of patient experience to optimize protocol acceptability and design of future phase III trials.

5. CONCLUSION

While conventional sleep medicine primarily relies on pharmacotherapy and Cognitive Behavioural Therapy for Insomnia (CBT-I), Ayurvedic Taila/Takra Dhara offers a complementary, low-risk intervention with a plausible neurophysiological rationale grounded in HPA axis modulation, autonomic rebalancing, and EEG synchronization. The convergence of classical Ayurvedic hyperarousal theory (Vata aggravation) with modern insomnia neuroscience (cortical and autonomic hyperarousal) provides a compelling translational framework. Existing clinical evidence - while limited in methodological rigor - consistently demonstrates improvements in subjective sleep quality and, in the strongest trial to date (Pawar et al., 2024), significant PSG-level improvements in SOL, SE, and WASO.

REFRENCES

1. Medic G, Wille M, Hemels ME. Short- and long-term health consequences of sleep disruption. Nat Sci Sleep. 2017;9:151-161.
2. Boulos MI, Jairam T, Kendzerska T, Im J, Mekhael A, Murray BJ. Normal polysomnography parameters in healthy adults: a systematic review and meta-analysis. *Lancet Respir Med*. 2019;7(6):533-543.
3. Carskadon MA, Dement WC. Normal human sleep: an overview. In: Kryger MH, Roth T, Dement WC, editors. Principles and Practice of Sleep Medicine. 6th ed. Philadelphia: Elsevier; 2017.
4. American Academy of Sleep Medicine. International Classification of Sleep Disorders. 3rd ed. Text Revision (ICSD-3-TR). Darien, IL: AASM; 2023.
5. Riemann D, Spiegelhalder K, Feige B, Voderholzer U, Berger M, Perlis M, et al. The hyperarousal model of insomnia: a review of the concept and its evidence. Sleep Med Rev. 2010;14(1):19-31.
6. . BaHammam AS. Polysomnographic characteristics of patients with chronic insomnia. Sleep Hypnosis. 2004;6(4):163–168.
7. Sharma RK, Dash B. Charaka Samhita. Varanasi: Chowkhamba Sanskrit Series Office; relevant Nidra and Nidranasha chapters.
8. Pawar S, Shende V, Khandekar A, Yelwatkar S. Polysomnographic evaluation of effect of Shirodhara and Ashwagandha on sleep in older adults with insomnia. Int J Acad Med Pharm. 2024;6(4):92-96.
9. Nicolaides NC, Vgontzas AN, Kritikou I, Chrousos GP. HPA axis and sleep. In: Feingold KR, et al., editors. Endotext [Internet]. South Dartmouth (MA): MDText.com, Inc.; 2000–. Updated 2020. Available from: https://www.ncbi.nlm.nih.gov/books/NBK279071/
10. Jerath R, Beveridge C, Barnes VA. Self-regulation of breathing as an adjunctive treatment of insomnia. Front Psychiatry. 2019;9:780. doi: 10.3389/fpsyt.2018.00780. PMID: 30713511.
11. Shi Y, Ren R, Lei F, Zhang Y, Vitiello MV, Tang X. Elevated beta activity in the nighttime sleep and multiple sleep latency electroencephalograms of chronic insomnia patients. Front Neurosci. 2022;16:1045934. doi: 10.3389/fnins.2022.1045934. PMID: 36466176.
12. Rodenbeck A, Cohrs S, Jordan W, Huether G, Rüther E, Hajak G. The sleep-improving effects of doxepin are paralleled by a normalized plasma

cortisol secretion in primary insomnia. Psychopharmacology (Berl). 2003;170(4):423-428.
13. Sharma RK, Dash B. *Charaka Samhita of Agnivesha*. Vol. I. Varanasi: Chowkhamba Sanskrit Series Office; 2014. Sutrasthana, Chapter 21, verses 35–36.
14. Sharma RK, Dash B. *Charaka Samhita of Agnivesha*. Vol. I. Varanasi: Chowkhamba Sanskrit Series Office; 2014. Sutrasthana, Chapter 21 verse 55.
15. Murthy KRS. *Ashtanga Hridayam*. Vol. I. Varanasi: Chowkhamba Krishnadas Academy; 2016. Sutrasthana, Chapter 12 verse 4-5.
16. Murthy KRS. *Ashtanga Hridayam*. Vol. I. Varanasi: Chowkhamba Krishnadas Academy; 2016. Sutrasthana, Chapter 12 verse 6-7.
17. Murthy KRS. *Ashtanga Hridayam*. Vol. I. Varanasi: Chowkhamba Krishnadas Academy; 2016. Sutrasthana, 1/12.
18. Sharma RK, Dash B. *Charaka Samhita of Agnivesha*. Vol. II. Varanasi: Chowkhamba Sanskrit Series Office; 2014. Indriyasthana, Chapters 4–5.
19. Sharma RK, Dash B. *Charaka Samhita of Agnivesha*. Vol I. Reprint ed. Varanasi: Chowkhamba Sanskrit Series Office; 2020. Sutrasthana, Ashtauninditiya Adhyaya; 21/55.
20. Murthy KRS. *Ashtanga Hridayam of Vagbhata*. Vol I. Reprint ed. Varanasi: Chaukhambha Krishnadas Academy; 2021. Sutrasthana; Chapter 7.
21. Sharma RK, Dash B. *Charaka Samhita of Agnivesha*. Vol II. Reprint ed. Varanasi: Chowkhamba Sanskrit Series Office; 2020. Vimana Sthana, Srotovimana Adhyaya; 5/7-8.
22. Sharma RK, Dash B. *Charaka Samhita of Agnivesha*. Vol II. Reprint ed. Varanasi: Chowkhamba Sanskrit Series Office; 2020. Sharirasthana, Katidhapurushiya Sharira; 1/18-19.
23. Murthy KRS. *Sushruta Samhita*. Vol I. Reprint ed. Varanasi: Chaukhambha Orientalia; 2020. Sharirasthana; 4/33-34.
24. Sharma RK, Dash B. *Charaka Samhita of Agnivesha*. Vol I. Reprint ed. Varanasi: Chowkhamba Sanskrit Series Office; 2020. Sutrasthana, Ashtauninditiya Adhyaya; 21/35.
25. Sharma RK, Dash B. *Charaka Samhita of Agnivesha*. Vol I. Reprint ed. Varanasi: Chowkhamba Sanskrit Series Office; 2020. Sutrasthana, Dirghanjivitiya Adhyaya; 1/57.
26. Murthy KRS. *Ashtanga Hridayam of Vagbhata*. Vol I. Reprint ed. Varanasi: Chaukhambha Krishnadas Academy; 2021. Sutrasthana, Murdhataila Vidhi; 22/23-26.
27. Uebaba K, Xu FH, Ogawa H, et al. Psychoneuroimmunologic effects of Ayurvedic oil-dripping treatment (*Shirodhara*). *J Altern Complement Med*. 2008;14(10):1189-1198.
28. Murthy KRS. *Ashtanga Hridayam of Vagbhata*. Vol I. Reprint ed. Varanasi: Chaukhambha Krishnadas Academy; 2021. Sutrasthana, Murdhataila Vidhi; 22/23-26.
29. Sharma PV. *Sahasrayogam*. Reprint ed. New Delhi: Central Council for Research in Ayurveda and Siddha; 2011. Dhara Prakarana.
30. Thayer JF, Lane RD. Claude Bernard and the heart-brain connection: Further elaboration of a model of neurovisceral integration. *Neurosci Biobehav Rev*. 2009;33(2):81-88.
31. Rani M, Rao PVK, et al. Clinical evaluation of Shirodhara in insomnia and stress-related disorders. *AYU*. Various issues.
32. Sharma RK, Dash B. *Charaka Samhita of Agnivesha*. Vol I. Reprint ed. Varanasi: Chowkhamba Sanskrit Series Office; 2020. Sutrasthana, Ashtauninditiya Adhyaya; 21/35-57.
33. Vinjamury SP, Vinjamury M, der Martirosian C, Miller J. Ayurvedic therapy (Shirodhara) for insomnia: a case series. *Glob Adv Health Med.* 2014;3(1):75-80. doi:10.7453/gahmj.2012.086.
34. Pawar S, Shende V, Khandekar A, Yelwatkar S. Polysomnographic evaluation of the effect of Shirodhara and cap. Ashwagandha on sleep in older adults suffering from insomnia. *Int J Acad Med Pharm.* 2024;6(4):92-96. doi:10.47009/jamp.2024.6.4.20.
35. Rajan S, Shamkuwar MK, Tanwar AK. Impact of Shirodhara on biological markers of stress: a case study. *J Ayurveda Integr Med.* 2021;12(1):178-181. doi:10.1016/j.jaim.2021.01.008.
36. Kumari K, Bhatted SK. Effect of Tila Taila Shirodhara in Anidra: a case series. *Ayushdhara.* 2023;10(Suppl 4):49-52.
37. Varshitha P, et al. [Title to be verified]. *Int J Creat Res Thoughts.* 2024;12(11):[pages].
38. Mohod P, et al. [Title to be verified]. *Int J Ayurvedic Pharm Res.* 2015;3(11):50-55.
39. Dhuri KD, et al. [Title to be verified]. *J Ayurveda Integr Med.* 2013;4(1):40-44. doi:10.4103/0975-9476.109550.