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NARRATIVE REVIEW ARTICLE

Stellate ganglion block for sleep disorders: a narrative review of clinical evidence and mechanistic pathways

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ABSTRACT

Background: Sleep disorders—including insomnia, obstructive sleep apnoea (OSA), circadian rhythm disturbances, and hypersomnia—are prevalent conditions associated with impaired quality of life and increased cardiometabolic risk. Chronic insomnia is increasingly conceptualized as a disorder of persistent autonomic hyperarousal characterized by sympathetic predominance, systemic inflammation, and hypothalamic–pituitary–adrenal (HPA) axis dysregulation. Stellate ganglion block (SGB), a cervical sympathetic intervention, may restore autonomic balance and modulate downstream neuroendocrine and immune pathways involved in sleep regulation.

Methods: A narrative review was conducted to synthesize clinical and mechanistic evidence regarding SGB in sleep dysfunction. PubMed and Google Scholar were searched using combinations of the terms sleep disorders, insomnia, autonomic nervous system, sympathetic activity, HPA axis, melatonin, circadian rhythm, inflammation, and stellate ganglion block. Clinical case series, controlled studies, and translational investigations were included. Due to heterogeneity in design and outcomes, findings were narratively synthesized.

Results: Clinical reports describe improvements in sleep latency, nocturnal awakenings, and subjective sleep quality following SGB. Mechanistic evidence suggests reductions in sympathetic outflow, attenuation of HPA axis hyperactivity, modulation of melatonin secretion, and suppression of pro-inflammatory cytokine signaling, consistent with integrative neurobiological models of sleep regulation.

Conclusions: SGB is a mechanistically plausible adjunctive intervention for sleep disorders associated with sympathetic overactivation. Robust randomized controlled trials with objective sleep metrics and long-term follow-up are required to confirm efficacy and durability.

Introduction

Sleep dysfunction is a pervasive public health concern, encompassing disorders such as insomnia, obstructive sleep apnea (OSA), restless leg syndrome, circadian rhythm disturbances, hypersomnia, and parasomnias. Insomnia alone affects up to one-third of adults, and roughly half of older adults experience sleep-related breathing disorders, most commonly OSA, yet most remain undiagnosed[1]. Chronic sleep disruption impairs restorative sleep, cognitive function, and quality of life, while also contributing to obesity, metabolic dysfunction, cardiovascular disease, hypertension, mood disorders, and substance use [2]. At a societal level, sleep deprivation has been implicated in major industrial and technological disasters, including the Challenger explosion, the Exxon Valdez oil spill, and the Chornobyl nuclear accident (3). In OSA, recurrent airway obstruction triggers sympathetic surges that further exacerbate cardiovascular risk, highlighting the interplay between autonomic dysregulation and sleep pathology. Current management strategies primarily emphasize behavioral interventions, including sleep hygiene optimization and cognitive behavioral therapy for insomnia (CBT-I). Pharmacologic therapies such as melatonin receptor agonists, orexin receptor antagonists, non-benzodiazepine hypnotics, and benzodiazepines can provide symptomatic benefit but are often limited by adverse effects, risk of dependence, and challenges with long-term adherence[4,5]. These limitations underscore the need for adjunctive approaches that target underlying neurobiological contributors to sleep dysfunction, rather than merely addressing symptoms. Stellate ganglion block (SGB) has emerged as a promising intervention in this context, supported by accumulating clinical observations. Concurrently, understanding of its mechanistic effects has expanded. Beyond modulation of sympathetic nervous system activity, SGB has been associated with downstream immunologic effects including reductions in pro-inflammatory cytokines such as interleukin-6[6] as well as normalization of melatonin secretion[7] and attenuation of hypothalamic pituitary adrenal (HPA) axis hyperactivity[8]. Through these integrated autonomic, neuroendocrine, and neuroimmune pathways, SGB may influence circadian regulation, stress responsivity, and persistent autonomic hyperarousal processes central to the pathogenesis of many sleep disorders. This review synthesizes the available clinical and experimental evidence supporting SGB as an adjunctive therapy for sleep disorders and integrates current understanding of the neurobiological mechanisms through which SGB may modulate sleep regulation.

Clinical and Experimental Evidence

Collectively, clinical investigations ranging from case reports and case series to randomized controlled trials provide convergent evidence that stellate ganglion block (SGB) improves sleep parameters across diverse sleep disorders (see findings in the Summary Table).

In a case series of patients with long-standing, treatment-refractory insomnia, Dong reported sustained clinical improvement following repeated alternating right- and left-sided SGB using 10 mL of 1% lidocaine. Over three to four treatment courses administered across 55–75 days, Insomnia Severity Index (ISI) scores declined from the moderate-to-severe range[21–23] to subthreshold or absent insomnia, with benefits maintained for up to 12 months. Transient Horner's syndrome confirmed procedural success in all patients, and adverse effects were limited to temporary hoarseness in two cases. Dong and colleagues proposed that sympathetic inhibition and restoration of autonomic balance, along with downstream effects on melatonin regulation, cerebral blood flow, and limbic–cortical hyperarousal circuits, underlie these sustained improvements, suggesting potential cumulative benefits with repeated blocks[9].

Expanding upon these findings in a randomized controlled trial, Liu evaluated ultrasound-guided

SGB using 0.375% ropivacaine at volumes of 4 mL, 6 mL, and 8 mL, compared with saline, in 80 patients with insomnia. After seven alternating bilateral blocks over 18 days, all active-treatment groups demonstrated significant improvements in Pittsburgh Sleep Quality Index (PSQI) scores relative to controls, without differences among active volumes. Horner syndrome occurred consistently in active groups, and carotid artery dilation with increased blood flow was observed, confirming physiological effect. Although higher volumes produced faster onset and longer block duration, they were associated with greater adverse effects and did not enhance sleep outcomes. Liu concluded that low-volume (4 mL) ultrasound-guided SGB offers optimal efficacy with improved safety[10].

Addressing procedural variables, Liu M and Qiu L conducted a randomized trial comparing sitting versus supine positioning for ultrasound-guided SGB in 181 patients with refractory insomnia. After seven sessions over four weeks, both groups demonstrated significant improvements in PSQI, AIS, and HAMA scores, with overall therapeutic response exceeding 80% and no between-group differences in efficacy. The sitting position, however, was associated with greater patient comfort and reduced psychological stress, without increased complications, supporting its use as a patient-centered alternative[11].

Postoperative Sleep Disturbance (PSD)

The role of SGB in perioperative sleep regulation has been examined in several randomized trials (see Table 1). In patients undergoing radical gastrointestinal surgery, Yan demonstrated that preoperative SGB significantly improved postoperative sleep efficiency, total sleep time, and sleep maintenance during the first two postoperative nights compared with standard care. Actigraphy and PSQI assessments confirmed reduced postoperative sleep disturbance. Biochemically, SGB was associated with lower postoperative IL-6, IL-1, and IL-10 levels, increased melatonin on the first postoperative night, and greater hemodynamic stability, supporting a mechanism involving anti-inflammatory modulation, circadian stabilization, and autonomic regulation[12].

In a lumbar spine surgery cohort, Luo further reported that preoperative right-sided SGB reduced the incidence and severity of postoperative sore throat (POST) and improved deep sleep quality across postoperative days 1–5, reinforcing the role of perioperative sympathetic modulation in sleep restoration[13]. Similarly, Luo confirmed in a randomized trial that preoperative ultrasound-guided SGB reduced early POST and significantly improved postoperative sleep quality compared with controls, aligning with prior evidence supporting autonomic stabilization as a contributor to improved recovery trajectories[13].

Excessive Daytime Sleepiness (EDS)

Beyond insomnia and perioperative sleep disturbance, SGB has also been explored in disorders of excessive sleepiness (see Table 1). In a case report, Xu described marked improvement in excessive daytime sleepiness (EDS) following cervical sympathetic blockade. The patient demonstrated enhanced wakefulness and daytime functioning after SGB, suggesting that modulation of the cervical sympathetic chain may influence broader sleep–wake regulatory circuits. Xu proposed that reduction of sympathetic hyperactivity and stabilization of hypothalamic and brainstem arousal pathways, along with normalization of neuroendocrine rhythms, may underlie these effects. Although limited to a single case, this observation extends the potential therapeutic scope of SGB beyond nocturnal insomnia to dysregulated daytime arousal states[14].

Summary Table : Clinical and Experimental Evidence of SGB on Sleep

Study / Author	Study Design	Population	SGB Protocol	Primary Sleep Outcomes	Mechanistic Findings	Safety / Notes
Dong	Case series (n=3)	Long-standing, treatment-refractory insomnia	10 mL 1% lidocaine; alternating R/L; 3–4 courses over 55–75 days	↓ ISI from 21–23 (moderate–severe) to subthreshold/no insomnia; sustained 3–12 months	Sympathetic inhibition; melatonin modulation; ↑ cerebral blood flow; limbic–cortical stabilization	Transient Horner's syndrome in all; temporary hoarseness in 2; no serious complications
Liu	Randomized controlled trial (n=80)	Insomnia	0.375% ropivacaine (4, 6, 8 mL) vs saline; 7 alternating bilateral blocks over 18 days	Significant PSQI improvement in all active groups vs control; no dose-response difference	Internal carotid dilation; ↑ blood flow	Higher volumes → ↑ adverse effects; 4 mL optimal safety-efficacy balance
Liu M, Qiu L	Randomized controlled trial (n=181)	Refractory insomnia	Ultrasound-guided SGB; sitting vs supine; 7 sessions over 4 weeks	Significant improvements in PSQI, AIS, HAMA; >80% therapeutic response; no positional difference	Autonomic stabilization	Sitting position improved comfort without increased complications
Yan (GI surgery)	Randomized controlled trial (n=40)	Radical GI malignancy surgery	Preoperative SGB	↑ sleep efficiency, total sleep time, ↓ awakenings; ↓ PSQI and PSD incidence	↓ IL-6, IL-1, IL-10; ↑ melatonin; improved hemodynamics	Demonstrates anti-inflammatory and neuroendocrine modulation
Luo (Lumbar spine surgery)	Randomized controlled trial (n=60)	Lumbar spine surgery under GA	Preoperative right-sided SGB 15 min before induction	↓ POST; ↑ deep sleep quality days 1–5 post-op	Perioperative autonomic modulation	↓ early POST; ↑ recovery profile
Xu	Case report (n=1)	Excessive Daytime Sleepiness (EDS)	SGB (details not specified in summary)	Marked improvement in wakefulness and daytime function	Hypothalamic & brainstem regulation; sympathetic downregulation; neuroendocrine stabilization	Single case; hypothesis-generating

Discussion

Overview

Accumulating evidence supports stellate ganglion block (SGB) as a promising adjunctive intervention for sleep disorders. Unlike conventional hypnotic or psychotropic therapies, which primarily modulate cortical neurotransmission, SGB targets upstream regulatory systems fundamental to sleep physiology. By acting across these interconnected autonomic, endocrine, and immune pathways, SGB likely addresses the core pathophysiologic mechanisms underlying hyperarousal-driven sleep disruption rather than merely suppressing downstream symptoms.

Sleep regulation is governed by an integrated network involving the suprachiasmatic nucleus (SCN), ventrolateral preoptic nucleus (VLPO), median preoptic nucleus (MnPO), locus coeruleus (LC), orexin neurons, pineal melatonin signaling, and the HPA axis[15-17]. Genetic factors account for approximately 31–58% of insomnia susceptibility, underscoring its strong biological foundation[18]. Rather than reflecting dysfunction of a single neurotransmitter system, insomnia is increasingly conceptualized as dysregulation of a coordinated sleep–wake control network[19].

Sleep is therefore best understood as the emergent product of multiple interacting neural, autonomic, endocrine, and immune systems. The relative contributions of these systems and the mechanisms through which sympathetic modulation may restore network stability are summarized below.

Circadian rhythm disruption destabilizes the reciprocal coupling between the sleep–wake cycle and autonomic output, particularly augmenting sympathetic nervous system activity. Circadian alignment may be perturbed by nocturnal light exposure (e.g., shift work, jet lag, sleep deprivation)[20], autoimmune disorders[21], persistent autonomic hyperarousal[20], and other environmental or physiological stressors.

Under physiologic nocturnal conditions, sleep particularly slow-wave sleep is characterized by reduced sympathetic outflow and a relative shift toward parasympathetic dominance. When circadian synchrony is disturbed, this nocturnal sympathetic withdrawal becomes attenuated or absent. The result is sustained noradrenergic activity during periods that should be biologically quiescent.

Over time, impaired ventrolateral preoptic nucleus (VLPO) inhibition combined with persistent sympathetic predominance establishes a bidirectional feedback loop linking sleep fragmentation, hypothalamic pituitary adrenal (HPA) axis activation, and inflammatory signaling. From a systems-neurobiology perspective, circadian misalignment is not merely a disorder of timing; it reflects a failure of VLPO-mediated inhibitory control over ascending arousal networks, permitting pathologically elevated sympathetic tone across the 24-hour cycle[20].

Heightened intracerebral sympathetic tone promotes excessive noradrenergic output from the locus coeruleus (LC). The LC is anatomically and functionally interconnected with orexin (hypocretin)–containing neurons in the lateral hypothalamus, as demonstrated by viral transneuronal labeling studies[22]. Increased LC activity amplifies orexin-mediated wake stabilization, thereby reinforcing ascending arousal circuitry. This interaction is bidirectional: orexin neurons activate the locus coeruleus, while LC-derived norepinephrine further stimulates orexinergic neurons[23]. The net effect is a self-reinforcing arousal loop that sustains hypervigilance and impairs both sleep initiation and maintenance.

In parallel, increased intracerebral sympathetic tone activates corticotropin-releasing hormone (CRH) neurons within the hypothalamus, triggering downstream activation of the hypothalamic–pituitary–adrenal (HPA) axis and subsequent cortisol release[24]. Elevated sympathetic activity also disrupts circadian melatonin secretion via

sympathetic projections to the pineal gland[7] and promotes pro-inflammatory cytokine production[6]. Collectively, these mechanisms converge to integrate autonomic, endocrine, and immune pathways into a unified hyperarousal phenotype, as summarized in Figure 1.

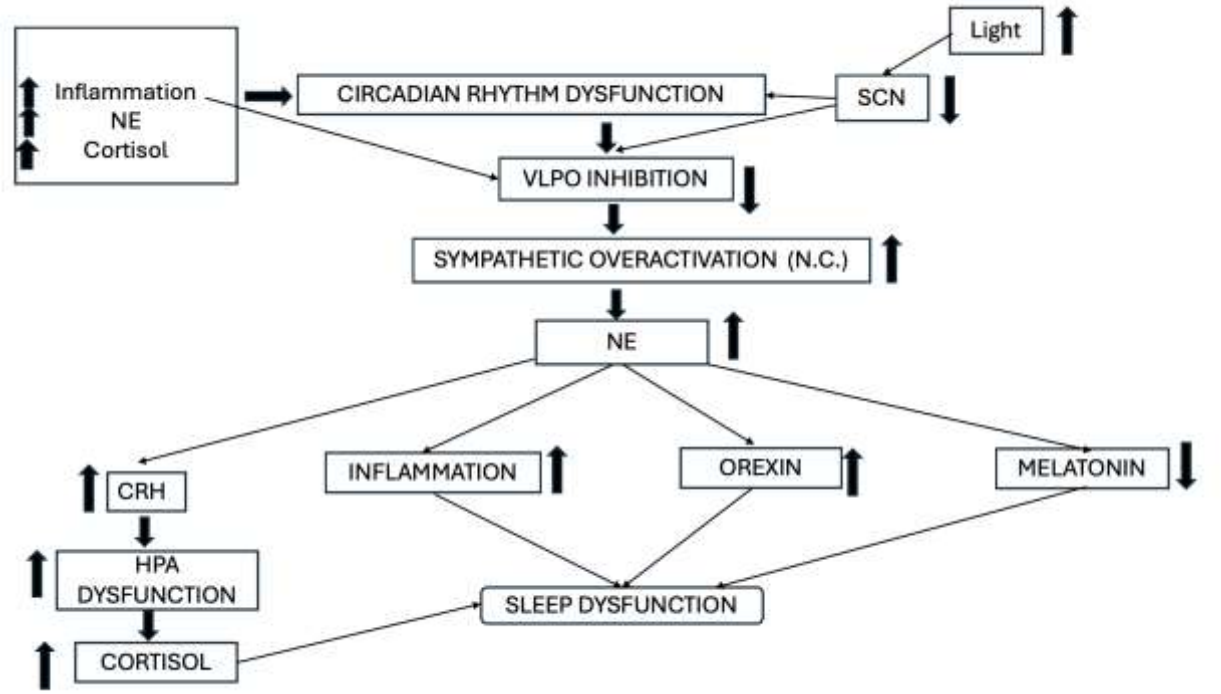

Figure 1: Integrated model of sleep dysfunction

Circadian rhythm (CR) disruption from light impairs SCN entrainment, inhibits VLPO activity, and promotes NC–mediated sympathetic activation with increased norepinephrine (NE). Systemic inflammation and sustained NE further destabilize CR. Elevated NE activates CRH and the HPA axis, increases cortisol and inflammatory signaling, enhances orexin activity, and suppresses melatonin, driving sleep dysfunction.

Abbreviations

- SCN – Suprachiasmatic nucleus
- VLPO – Ventrolateral preoptic nucleus
- NC – Nucleus coeruleus
- CRH – Corticotropin-releasing hormone
- HPA axis – Hypothalamic–pituitary–adrenal axis

SGB

Clinically, SGB is associated with rapid improvements in sleep latency, decreased nocturnal awakenings, and enhanced sleep depth, often emerging within hours to days following treatment[9-12]. These effects are likely mediated by multiple convergent mechanisms, with a central unifying feature being reduction of intracerebral norepinephrine (NE) tone.

Based on previously published data, stellate ganglion block (SGB) is hypothesized to decrease central NE activity as well as nerve growth factor (NGF) expression. Elevated NGF potentially secondary to PTSD and other chronic stress-related conditions promotes sympathetic axonal sprouting within the brain and increases noradrenergic fiber density, thereby amplifying central NE signaling. By attenuating NGF levels, SGB may facilitate pruning of aberrant sympathetic projections and produce a sustained, prolonged reduction in central norepinephrine tone[25].

Melatonin and SGB

Melatonin plays a central role in circadian entrainment and sleep initiation[4,17]. It secretion is

regulated by sympathetic projections from the superior cervical ganglion to the pineal gland, mediated by norepinephrine acting on adrenergic receptors[26,27]. Disruption of this pathway alters circadian phase and impairs sleep initiation. SGB modulates this sympathetic input and has been associated with restoration of physiologic melatonin secretion, thereby supporting circadian realignment and improved sleep depth and consolidation[7,12].

HPA and SGB

Cortisol exhibits a robust circadian rhythm tightly regulated by the hypothalamic–pituitary–adrenal (HPA) axis[28]. Disruption of this rhythm particularly sustained hypercortisolemia or phase misalignment impairs both sleep initiation and maintenance. Stress-induced activation of corticotropin-releasing hormone (CRH) neurons further suppresses sleep and promotes cortical and autonomic arousal[24]. Stellate ganglion block (SGB) has been demonstrated to stabilize autonomic tone and modulate neuroendocrine function, including downstream effects on the HPA axis[8].

Systemic Inflammation and SGB

Sleep insufficiency is strongly associated with systemic inflammation. Elevated circulating cytokines including IL-1β, IL-6, TNF-α, and IL-10 have been documented in states of chronic sleep restriction[29]. These inflammatory mediators alter neuronal excitability, impair ventrolateral preoptic nucleus (VLPO) function, increase nocturnal arousal, and diminish slow-wave sleep. Stellate ganglion block (SGB) has consistently been associated with reductions in pro-inflammatory cytokines, particularly IL-6 and TNF-α[6].

SGB may exert system-wide anti-inflammatory effects through modulation of sympathetic–immune interactions. Both primary (thymus, bone marrow) and secondary (spleen, lymph nodes, mucosa-associated lymphoid tissue) immune organs are densely innervated by sympathetic fibers, with norepinephrine (NE) serving as the principal neurotransmitter. Adaptive immune cells (T and B lymphocytes) express β_2-adrenergic receptors, whereas innate immune cells express both α- and β-adrenergic receptors, permitting broad autonomic regulation of immune activity. Activation of these receptors produces predominantly immunomodulatory effects: attenuation of pro-inflammatory cytokines (e.g., IL-1β, IL-6, TNF-α), upregulation of anti-inflammatory mediators (e.g., IL-10, TGF-β), expansion of regulatory T-cell populations, and a shift in Th1/Th2 balance toward a Th2-dominant profile. Collectively, these coordinated changes reflect systemic recalibration of immune tone rather than localized suppression[6].

Clinical evidence supports this framework. In patients undergoing radical gastrointestinal surgery, Yan demonstrated that preoperative SGB significantly improved postoperative sleep efficiency, total sleep time, and sleep maintenance during the first two nights compared with standard care, with actigraphy and PSQI confirming reduced sleep disturbance. Biochemically, SGB was associated with lower postoperative IL-6, IL-1, and IL-10 levels, increased melatonin on the first night, and greater hemodynamic stability supporting mechanisms of anti-inflammatory modulation, circadian stabilization, and autonomic regulation[12].

Orexin and SGB

Orexin neurons stabilize wakefulness and project to the locus coeruleus and other sympathetic centers, thereby linking orexin signaling to noradrenergic arousal circuits[22]. Among orexin receptors, orexin receptor 2 (OX2R) plays a dominant role in sustaining arousal[30]. Reduced orexin levels or disruption of orexin signaling produce excessive daytime sleepiness and are causally associated with narcolepsy with cataplexy[31]. Conversely, heightened sympathetic activity may potentiate orexin-driven wake stabilization, reinforcing hyperarousal states[23].

By attenuating sympathetic tone, stellate ganglion block (SGB) may indirectly rebalance orexin–

noradrenergic interactions, reducing persistent wake drive and facilitating more physiologic sleep–wake transitions. Clinical evidence supports this framework: SGB has been explored in disorders of excessive daytime sleepiness (EDS), with Xu reporting significant improvement in wakefulness and daytime functioning following cervical sympathetic blockade[14]. The proposed mechanism involves reduction of sympathetic hyperactivity and stabilization of hypothalamic and brainstem arousal circuits, suggesting that SGB may modulate broader sleep–wake regulatory networks beyond nocturnal insomnia.

Limitations

Several limitations should be acknowledged. First, this review is narrative rather than systematic, and therefore does not follow PRISMA methodology or include formal risk-of-bias assessment. Although relevant literature was identified through structured database searches, selection bias cannot be excluded. Second, the clinical evidence base for stellate ganglion block (SGB) in sleep disorders remains heterogeneous and limited in scale. Existing studies include small randomized trials, case series, and case reports, with variability in patient populations, procedural protocols (e.g., anesthetic agent, volume, laterality, frequency), and outcome measures.

Third, many studies rely on subjective sleep metrics such as the Pittsburgh Sleep Quality Index (PSQI) or Insomnia Severity Index (ISI), with relatively limited incorporation of objective measures such as polysomnography or actigraphy. Fourth, long-term durability data are sparse, and optimal dosing intervals and maintenance strategies remain undefined. Fifth, mechanistic inferences regarding autonomic, neuroendocrine, and neuroimmune modulation are based on indirect evidence and translational models rather than direct human neurophysiologic measurement.

Finally, publication bias toward positive outcomes cannot be excluded, and larger multicenter randomized controlled trials with standardized protocols and objective sleep endpoints are required to confirm reproducibility, durability, and safety across diverse patient populations.

Conclusion

Sleep disorders arise from complex dysregulation across autonomic, circadian, neuroimmune, and stress-response systems. Stellate ganglion block targets these convergent pathways by modulating sympathetic tone, restoring melatonin signaling, and reducing inflammatory and stress-mediated arousal. Existing evidence supports SGB as a safe and effective adjunctive therapy for sleep dysfunction, warranting further prospective investigation in rigorously designed controlled trials.

Conflict of Interest:

The authors have no conflicts of interest to declare.

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