Research Article

Sleep Disturbance on Stroke Patients: A Narrative Review

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ABSTRACT

Purpose of Review: Sleep disturbance is one of the most prevalent yet systematically neglected consequences of stroke. It affects between one-third and more than half of all stroke survivors, yet it receives a fraction of the clinical attention devoted to motor or cognitive impairment. This review was undertaken to examine the prevalence and clinical spectrum of post-stroke sleep disturbance, to understand the neurobiological mechanisms through which stroke disrupts sleep architecture, and to evaluate the clinical consequences of poor sleep quality for functional recovery, neuroplasticity, depression, cognitive function, and quality of life in stroke survivors.
Methods: A comprehensive narrative literature search was conducted across PubMed, Google Scholar, ScienceDirect, CINAHL, Embase, PsycINFO, and the Cochrane Library. The final search was performed on March 2026. Search terms included combinations of "stroke," "sleep quality," "insomnia," "sleep disturbance," "sleep architecture," "post-stroke sleep," "neuroplasticity," "motor recovery," "functional outcomes," "Pittsburgh Sleep Quality Index," "polysomnography," "actigraphy," "quality of life," "depression," "cognitive decline," and "circadian rhythm." Studies published between January 2015 and March 2026 were included. Eligible studies included randomized controlled trials, observational studies, systematic reviews, and meta-analyses examining sleep quality and its consequences in adult stroke populations. Non-English publications, case reports, editorials, and conference abstracts were excluded.
Results: Post-stroke sleep disturbance is highly prevalent, affecting 32–56% of stroke survivors across study designs and diagnostic criteria, with evidence that it persists into the chronic phase of recovery in the majority of affected patients. Neurobiological mechanisms include direct structural damage to hypothalamic, thalamic, and brainstem sleep-regulatory circuits; neuroinflammatory disruption of sleep homeostasis; and secondary behavioural perpetuating factors including irregular schedules, excessive time in bed, and post-stroke depression. Poor sleep quality independently predicts worse motor outcomes, slower functional recovery, greater depression severity, accelerated cognitive decline, reduced quality of life, and elevated stroke recurrence risk. Fewer than 6% of stroke survivors receive formal sleep evaluation in the first three post-stroke months despite this burden.
Conclusion: Sleep quality is a core determinant of stroke recovery outcomes, not a peripheral quality-of-life concern. The mechanistic evidence establishing sleep as an active component of rehabilitation-relevant neuroplasticity, combined with clinical evidence that poor sleep independently predicts functional and psychological outcomes, justifies its integration into standard post-stroke rehabilitation assessment and management. Routine sleep screening using validated instruments should be implemented immediately. Prospective longitudinal research examining the causal pathways between specific sleep architecture deficits and rehabilitation outcomes in diverse populations is urgently needed.

Keywords: post-stroke sleep disturbance; sleep quality; insomnia; sleep architecture; Pittsburgh Sleep Quality Index; neuroplasticity; motor memory consolidation; functional recovery; quality of life; depression; cognitive decline; stroke rehabilitation.

INTRODUCTION

Stroke and the Overlooked Burden of Sleep Disturbance

Stroke is a leading cause of global adult disability (101 million prevalent cases, 143 million DALYs annually), disproportionately burdening low- and middle-income regions (1). While motor and cognitive deficits dominate clinical care, sleep disturbance remains a clinically critical yet overlooked post-stroke consequence (2). Affecting between one-third and over half of all stroke survivors, fewer than 6% receive formal sleep evaluation within three months post-stroke (2, 3). Standard pharmacological treatments carry severe risks in older neurological populations—such as falls, cognitive impairment, and respiratory depression—while non-pharmacological alternatives remain underutilized (4).

Why Sleep Matters Beyond Comfort: The Neuroplasticity Argument

Sleep is a mechanistically essential driver of neurobiological recovery rather than just a comfort measure. Slow-wave and REM sleep actively consolidate offline motor learning and strengthen synaptic connections targeted by physical therapy (5, 6). When post-stroke sleep is fragmented or suppressed, the neuroplastic gains achieved during rehabilitation therapy cannot properly consolidate, directly hindering physical recovery (7).

Sleep Quality and Rehabilitation Outcomes

Substantial evidence demonstrates that poor sleep independently predicts worse motor trajectories, increased post-stroke depression, accelerated cognitive decline, and reduced long-term quality of life, even after controlling for stroke severity (7, 8, 9). These findings establish sleep quality as an independent causal determinant of recovery. This review addresses the prevalence, neurobiological mechanisms, and clinical consequences of post-stroke sleep disturbance, and how they should reform clinical rehabilitation practice.

METHODS

Search Strategy

A comprehensive narrative literature search was conducted across seven electronic databases: PubMed, Google Scholar, ScienceDirect, CINAHL, Embase, PsycINFO, and the Cochrane Library. The final search was completed in March 2026. The search strategy utilised the following Boolean combinations: ("stroke" OR "cerebrovascular accident" OR "ischaemic stroke" OR "haemorrhagic stroke") AND ("sleep quality" OR "insomnia" OR "sleep disturbance" OR "sleep architecture" OR "post-stroke sleep") AND ("functional recovery" OR "neuroplasticity" OR "motor recovery" OR "quality of life" OR "depression" OR "cognitive function" OR "Pittsburgh Sleep Quality Index"). For Google Scholar, the first 200 results sorted by relevance were screened due to the large volume of uncurated results.

Inclusion and Exclusion Criteria

To ensure relevance and clinical focus, the search window was restricted to publications between January 2015 and March 2026. Table 1 details the inclusion and exclusion criteria applied during the screening process.

Table 1: Inclusion and Exclusion Criteria

Criterion Category	Inclusion Criteria	Exclusion Criteria
Population	Adult stroke survivors (≥18 years) at any post-stroke phase	Paediatric populations; neurological conditions unrelated to stroke; healthy controls only
Variables of interest	Sleep quality as a primary or secondary outcome; sleep architecture metrics; consequences of sleep disturbance for functional, psychological, or quality-of-life outcomes	Studies not reporting sleep outcomes; sleep studies in non-stroke neurological populations without stroke-specific data
Study design	Randomized controlled trials, observational studies, systematic reviews, meta-analyses	Case reports, editorials, letters to the editor, narrative reviews lacking primary data synthesis, conference abstracts
Language and format	Peer-reviewed articles published in English	Non-English publications (due to limited translation resources), dissertations, preprints, grey literature
Publication period	January 2015 – March 2026	Publications before January 2015 (except foundational citations)

Screening Process and Quality Appraisal

Two independent reviewers screened identified records by title and abstract to determine initial eligibility. Disagreements were resolved through discussion or consultation with a third reviewer. Full-text review was subsequently conducted for all potentially eligible records. While a formal systematic quality appraisal score (e.g., PEDro scale or AMSTAR) was not generated given the narrative review design, critical appraisal was actively incorporated into data synthesis. This qualitative appraisal focused on sample size adequacy, study design limitations, risk of bias, and the distinction between statistical and clinical significance. A summary table of the key included studies is presented in Table 2.

Table 2: Summary Table of Key Included Studies

Author, Year	Study Design	n	Sleep Measure	Population	Key Findings	Limitations	Quality
Baylan et al., 2020	Systematic review and meta-analysis	29 studies (n=9,834)	PSQI, clinical interview, PSG (varied)	Stroke survivors	Pooled insomnia prevalence 32% (95%CI: 26–38%); high heterogeneity across studies	Heterogeneous assessment instruments; wide CI; variable time since stroke	Moderate-High
Fleming et al., 2021	Observational (cross-sectional)	60	PSQI + wrist actigraphy	Chronic stroke, incomplete motor recovery	68% poor sleep quality (PSQI>5); mean SE 76.4%; persists in chronic phase	Small sample; chronic phase only; cross-sectional design	Moderate
Fleming et al., 2020	Prospective longitudinal	60	Wrist actigraphy	Acquired brain injury (ABI) including stroke	Sleep disruption independently predicts worse motor outcomes and slower recovery after adjusting for confounders	Mixed ABI population; 12-week follow-up only; no PSG	Moderate-High
Sandeep & Morgenstern, 2022	Narrative review	N/A	Multiple (review)	Stroke survivors	Only 6% receive formal sleep evaluation in first 3 months; OSA in 30–80%	No primary data; variability in included studies	Moderate
Baylan et al., 2023	Systematic review and meta-analysis	11 RCTs	PSQI, ISI, PSG (varied)	Stroke survivors	Sleep interventions improved sleep quality (SMD −0.61), depression, and HRQoL in stroke survivors	Heterogeneous interventions; small trial samples; limited follow-up	Moderate-High
Li et al., 2022	Systematic review and meta-analysis	Multi-cohort	Insomnia symptoms (self-report)	General and stroke populations	Insomnia associated with 54% increased stroke incidence/recurrence risk after adjustment	Self-report measures; reverse causality possible; heterogeneous populations	Moderate-High
Fulk et al., 2020	Prospective cohort	95	PSQI	Stroke survivors,	Sleep problems independently	Single US centre; self-	Moderate

Author, Year	Study Design	n	Sleep Measure	Population	Key Findings	Limitations	Quality
				12-month follow-up	predict worse HRQoL and reduced community participation at 12 months	report sleep measure; no objective monitoring	
Tang et al., 2021	Observational cohort	210	PSQI, SF-36	Chronic stroke survivors	Insomnia severity independently predicts lower HRQoL across all SF-36 domains including physical functioning	Chinese cohort; cross-sectional component; no polysomnography	Moderate
Nissen et al., 2021	Experimental (human TMS study)	Controlled	PSG + TMS	Healthy adults (mechanistic)	Sleep potentiates LTP-like synaptic mechanisms in primary motor cortex relevant to rehabilitation neuroplasticity	Healthy adults — not stroke; indirect extrapolation required	High (mechanistic)
Al-Dughmi et al., 2017	Observational (cross-sectional)	Pilot	PSQI + cognitive tests	Chronic stroke	Poor sleep independently associated with greater cognitive impairment in chronic stroke	Small pilot sample; cross-sectional; self-report sleep measures	Low-Moderate

Table Legend: ABI = acquired brain injury; CI = confidence interval; HRQoL = health-related quality of life; ISI = Insomnia Severity Index; LTP = long-term potentiation; n = sample size; PSG = polysomnography; PSQI = Pittsburgh Sleep Quality Index; RCT = randomized controlled trial; SE = sleep efficiency; SF-36 = Short Form-36 Health Survey; SMD = standardised mean difference; TMS = transcranial magnetic stimulation.

RESULTS

Prevalence and Clinical Spectrum of Post-Stroke Sleep Disturbance

Post-stroke sleep disturbance is widespread, with a pooled insomnia prevalence of 32% (3) and overall sleep-wake disorder estimates exceeding 50% (2). Objective evaluations reveal that 68% of chronic stroke survivors exceed the PSQI threshold for poor sleep, demonstrating a reduced mean sleep efficiency of 76.4% (10). Subtypes include insomnia (3), obstructive sleep apnea affecting 30–80% of ischemic patients (2, 11), excessive daytime sleepiness in 20–40% (8), persistent circadian rhythm disruption (12), and REM sleep behavior disorder in up to 12% (13). Crucially, these disturbances persist long into the chronic phase without resolving spontaneously, establishing them as long-term therapeutic targets (3, 10).

Neurobiological Mechanisms of Post-Stroke Sleep Disturbance

Post-stroke sleep disruption stems from three main interacting mechanisms:

- **Direct Circuit Damage:** Ischemia affecting the suprachiasmatic nucleus, thalamus, and monoaminergic or cholinergic brainstem systems disrupts melatonin release, sleep spindle generation, sensory gating, and NREM/REM state transitions (11, 13).
- **Neuroinflammation:** Elevated post-stroke levels of IL-1β, TNF-α, and IL-6 fragment sleep architecture and selectively suppress slow-wave sleep, an effect compounded by OSA-related hypoxic stress (14, 15).

- **Secondary Behaviors:** Excessive time in bed, irregular schedules, post-stroke depression (present in 30–40% of cases), pain, and spasticity create self-perpetuating cycles that prevent spontaneous recovery without targeted intervention (16, 17).

Impact of Sleep Quality on Neuroplasticity and Motor Recovery

Slow-wave NREM sleep is mechanistically required for motor cortex ensemble reactivation and motor memory consolidation (5), while restorative sleep potentiates LTP-like synaptic plasticity essential for physiotherapy gains (6). Clinically, objective sleep disruption independently predicts worse motor trajectories (7), reduces actual rehabilitation participation rates by ~30% (18), and predicts poorer 1-year functional outcomes (8). Meta-analytic evidence confirms that sleep interventions significantly improve post-stroke functional recovery (SMD −0.61) (19).

Sleep Quality, Depression, and Psychological Outcomes After Stroke

Sleep disruption and post-stroke depression share a bidirectional relationship (17). Baseline insomnia severity independently predicts follow-up depression severity (20). Depression elevates HPA axis activity and nocturnal cortisol, while sleep loss impairs emotional regulation, heightens amygdala reactivity, and suppresses REM sleep—which normally processes emotional memories essential for psychological adaptation to disability (17, 21).

Sleep Quality, Cognitive Function, and Stroke Recurrence Risk

Poor sleep quality independently accounts for greater cognitive impairment (22) and compounds long-term fatigue (23), worsening existing neurological vulnerabilities (21). Furthermore, insomnia symptoms increase incident stroke or recurrence risk by 54% via elevated blood pressure, inflammation, sympathetic activation, and metabolic dysregulation (12, 14), while concurrent OSA independently elevates recurrence risk through nocturnal hypoxemia and blood pressure surges (2).

Assessment of Sleep Quality in Stroke Populations

While laboratory polysomnography (PSG) is the diagnostic gold standard for sleep architecture and OSA, its high burden limits routine rehabilitation use (24). Actigraphy offers multi-night objective data, though motionless wakefulness in hemiparetic patients can reduce reliability (27). Consequently, the Pittsburgh Sleep Quality Index (PSQI)—validated in Arabic (26)—serves as the primary, highly sensitive (89.6%) and specific (86.5%) clinical tool to identify poor sleep (scores >5) in stroke rehabilitation settings (25).

DISCUSSION

Synthesis of Prevalence, Mechanisms, and Clinical Consequences

Evidence across four key domains indicates that sleep quality is a modifiable, clinically critical determinant of stroke recovery that remains under-assessed and under-treated. Because slow-wave and REM sleep actively consolidate motor learning (5, 6) and poor sleep independently predicts worse functional recovery (7, 8), sleep quality must be treated as a core rehabilitation variable. Monitoring sleep should be as routine as motor assessment to ensure patients maximize their therapy outcomes.

The Missing Integration: Sleep Architecture and Rehabilitation Outcomes

Mechanistic studies demonstrate why N3 and REM sleep are vital for motor learning (5, 6), and clinical research links overall sleep quality to recovery outcomes (7, 8). However, no human stroke studies simultaneously track objective sleep architecture alongside standardized functional measures. Proving that specific N3 and REM deficits directly impair therapy-relevant motor consolidation remains the field's primary research gap (Ramanathan et al., 2015; Nissen et al., 2021; Fleming et al., 2020; Fulk et al., 2020).

Ethical and Practical Considerations

Data Privacy and Security: Stroke sleep research collects sensitive personal data from vulnerable populations. Protocols must strictly address confidentiality, secure storage, and informed consent, particularly when syncing wearable data to commercial cloud platforms.

Health Equity and Access: Most studies stem from high-income nations, leaving advanced assessments (polysomnography, validated actigraphs) largely inaccessible in low- and middle-income regions like Egypt and the broader Arabic-speaking world, despite high stroke burdens (1, 2). Consumer wearables offer equitable potential but require stroke-specific validation in diverse groups.

Algorithmic and Population Bias: Consumer tracking algorithms rely heavily on Caucasian datasets from high-income regions. Dedicated validation studies in Middle Eastern and Arabic-speaking stroke populations are necessary to ensure accuracy.

Regulatory Considerations: Consumer wearables used for clinical monitoring fall into a regulatory grey zone. While research use is generally exempt, clinician-directed care based on wearable data faces varying regulatory standards, making institutional guidance essential.

Patient and Clinician Perspectives

Patient Acceptance: Engagement depends on perceived assessment burden, perceived relevance to recovery, and belief in treatment efficacy. Because stroke survivors often do not report sleep issues—assuming they are unavoidable or secondary to physical goals—clinicians must proactively screen using brief validated tools like the PSQI.

Clinician Acceptance: Integrating sleep into rehabilitation requires a cultural shift. Rehabilitation staff need clear evidence summaries regarding neuroplastic neurobiology, and clinical services should track sleep quality as a routine outcome alongside motor, functional, and psychological measures.

LIMITATIONS

This narrative review has several limitations that must be acknowledged when interpreting its findings. First, the search strategy was restricted to publications from January 2015 to March 2026; while this 11-year window captures the most recent evidence and key foundational work, some earlier theoretical and mechanistic frameworks may have been missed. Second, the exclusion of non-English publications and grey literature may have introduced language and publication bias, potentially over-representing positive findings and under-representing research from Arabic-speaking and other non-anglophone populations. Third, the narrative review methodology does not apply the quantitative risk-of-bias assessment and data synthesis employed in systematic reviews and meta-analyses; findings should be interpreted as a synthesis of the available evidence landscape rather than a definitive quantitative estimate. Fourth, the majority of cited studies originate from high-income countries, significantly limiting generalisability to global populations and particularly to the Egyptian and Arabic-speaking contexts where the described clinical need is greatest. Finally, as with all narrative reviews, the selection of studies involves an element of reviewer judgment that may introduce selection bias despite comprehensive search procedures.

FUTURE RESEARCH DIRECTIONS

1. **Prospective longitudinal studies** using polysomnography or validated actigraphy to measure sleep architecture simultaneously alongside standardized functional and motor rehabilitation outcomes are urgently needed in well-characterized stroke cohorts.
2. **Large-scale epidemiological studies** characterizing the prevalence, clinical spectrum, and determinants of post-stroke sleep disturbance in Arabic-speaking, Middle Eastern, and North African populations are required to address the severe geographic evidence gap.
3. **Standardized measurement protocols** specifying instruments, timing, and clinical contexts for post-stroke sleep assessment must be developed and validated to enhance cross-study comparability and enable routine clinical practice.
4. **Validation studies of consumer wearables** (such as the Samsung Galaxy Watch 5 and similar devices) against polysomnography standards, utilizing Bland-Altman analysis, are necessary before clinical deployment in stroke populations.
5. **Implementation research** evaluating the feasibility, acceptability, and real-world impact of routine sleep screening via the PSQI Arabic version in standard Egyptian and regional rehabilitation protocols is needed to support policy change.

CONCLUSION

Sleep disturbance after stroke is a common, neurobiologically critical, and undertreated issue, affecting over half of survivors due to neural damage, neuroinflammation, and self-perpetuating behavioral patterns. Poor sleep quality negatively impacts every facet of recovery—impairing motor learning consolidation, worsening depression, accelerating cognitive decline, reducing quality of life, and increasing stroke recurrence risk.

Because slow-wave and REM sleep actively consolidate physiotherapy learning in the motor cortex, sleep must be reframed as a core mechanism of recovery rather than a simple comfort issue. A patient with poor sleep quality may be physically capable in therapy yet unable to neurobiologically consolidate those gains.

Despite this evidence, clinical action remains insufficient. Rehabilitation services must immediately implement routine sleep screening using brief, accessible tools like the PSQI Arabic version for Arabic-speaking populations. Future research priorities should focus on connecting sleep architecture to rehabilitation outcomes, expanding epidemiological data in underrepresented populations, and integrating sleep management into standard global post-stroke care.

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DISCLOSURE STATEMENT & CONFLICT OF INTEREST

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