RESEARCH PAPER

Preoperative Sleep Disorders in Older Adults: Implications for Postoperative Cognitive Recovery

Mohamed Yaseen Mohamed Moawad[1], Prof. Amany Khairy Abu Elhussein[2], Prof. Haidy Salah Mansour[3], Dr. Mohamed Mostafa Ali[4]

[1]Assistant Lecturer of Anesthesiology & Intensive Care Department, Faculty of medicine – Minia University, Mohamed.Yaseen@mu.edu.eg

[2]Prof. of Anesthesiology & Intensive Care Department, Faculty of medicine – Minia University

[3]Prof. of Anesthesiology & Intensive Care Department, Faculty of medicine – Minia University

[4]Lecturer of Anesthesiology & Intensive Care Department, Faculty of Medicine, Minia University

Received 27 July 2026, Revised 22 August 2026, Accepted 31 August 2026, Available Online 02 September 2026

Abstract

Sleep disturbance is common in older adults presenting for surgery, yet it is frequently treated as a comfort issue rather than a component of perioperative brain health. Age-related reductions in slow-wave sleep, circadian amplitude, and sleep continuity coexist with a high prevalence of insomnia and obstructive sleep apnea, creating a vulnerable preoperative state that may interact with surgical stress, anesthesia, pain, inflammation, hypoxemia, and environmental disruption. This narrative review examines the implications of preoperative sleep disorders for postoperative cognitive recovery in older adults. Current evidence is strongest for postoperative delirium, with recent prospective studies and meta-analyses showing that poor preoperative sleep, short sleep duration, altered slow-wave sleep, and obstructive sleep apnea are associated with greater delirium risk. Emerging data also link preoperative sleep disturbance to delayed neurocognitive recovery and postoperative cognitive dysfunction over weeks to months. Proposed mechanisms include exaggerated neuroinflammation, blood-brain barrier dysfunction, circadian and hypothalamic-pituitary-adrenal dysregulation, intermittent hypoxemia, impaired synaptic homeostasis, and disruption of sleep-dependent metabolic clearance. Preoperative assessment can combine a brief sleep history with validated instruments such as the Pittsburgh Sleep Quality Index, Insomnia Severity Index, and STOP-Bang questionnaire. Management should prioritize identification of untreated sleep apnea, continuation of positive airway pressure therapy, nonpharmacological sleep optimization, medication review, pain control, and preservation of circadian cues. Melatonin, ramelteon, and dexmedetomidine have shown preventive signals for delirium in selected settings, but current evidence does not justify treating them as universal sleep-directed neuroprotection. Preoperative sleep disturbance should therefore be viewed as both a marker of reduced cognitive reserve and a potentially modifiable perioperative risk domain. Well-designed trials are needed to determine whether targeted preoperative sleep treatment can causally improve postoperative cognitive trajectories.

Keywords: Sleep disorders; Older adults; Postoperative delirium; Perioperative neurocognitive disorders; Obstructive sleep apnea; Cognitive recovery

How to cite this article: Mohamed Yaseen Mohamed Moawad1 Prof. Amany Khairy Abu Elhussein2 Prof. Haidy Salah Mansour3 Dr. Mohamed Mostafa Ali4. Preoperative Sleep Disorders in Older Adults: Implications for Postoperative Cognitive Recovery

Mohamed Yaseen Mohamed Moawad1, Prof. Amany Khairy Abu Elhussein2, Prof. Haidy Salah Mansour3, Dr. Mohamed Mostafa Ali4. Int J Drug Deliv Technol. 2026;16(80s):605-615. DOI: 10.25258/ijddt.16.80s.75.

1. Introduction

Sleep changes substantially across the adult lifespan. Healthy aging is associated with earlier sleep timing, more fragmented nocturnal sleep, reduced sleep efficiency, and a marked reduction in slow-wave sleep, while the ability of the circadian and homeostatic systems to maintain consolidated sleep becomes less robust **[1,2]**. These physiological changes do not necessarily constitute disease, but they narrow the margin by which older adults can tolerate additional sleep disruption from pain, anxiety, hospitalization, medication exposure, and surgery.

The International Classification of Sleep Disorders recognizes insomnia, sleep-related breathing disorders, circadian rhythm sleep-wake disorders, movement disorders, parasomnias, and hypersomnolence disorders as distinct clinical entities **[3]**. In surgical populations, however, these diagnoses often present as overlapping symptoms such as prolonged sleep latency, short sleep duration, frequent awakenings, nonrestorative sleep, daytime fatigue, or witnessed apneas. A systematic review found that sleep disturbance is common before and after surgery and is influenced by demographic, psychological, clinical, and procedural factors **[4]**. Chronic insomnia is especially relevant because contemporary guidelines emphasize cognitive-behavioral therapy for insomnia as first-line treatment and recommend careful use of pharmacological therapy, particularly in older people **[5]**.

At the same time, postoperative cognitive complications remain among the most consequential adverse outcomes of surgery in later life. Delirium can emerge within hours to days, while more subtle deficits in memory, attention, executive function, and processing speed may persist for weeks or months. Sleep is biologically linked to each of these domains through memory consolidation, synaptic plasticity, circadian organization, metabolic homeostasis, and immune regulation. The practical question is therefore no longer whether sleep and cognition are related, but whether the preoperative sleep state identifies a modifiable vulnerability that can be incorporated into perioperative brain-health pathways.

2. Preoperative Sleep Disorders in Older Adults

2.1 Age-related sleep changes and insomnia

Normal aging shifts sleep architecture toward lighter and more fragmented sleep. Older adults tend to have less stage N3 sleep, more nocturnal awakenings, an earlier circadian phase, and more daytime napping than younger adults **[1,2]**. Comorbid pain, nocturia, cardiopulmonary disease, depression, anxiety, polypharmacy, reduced daytime activity, and environmental light exposure can convert these physiological changes into clinically significant insomnia or irregular sleep-wake patterns. In the preoperative period, uncertainty about surgery, fasting instructions, travel, and unfamiliar sleeping environments may further reduce sleep duration even in patients without chronic insomnia.

The distinction between chronic sleep disorder and transient preoperative sleep loss is clinically useful. Chronic insomnia is defined by persistent difficulty initiating or maintaining sleep, or early awakening, together with daytime impairment despite adequate opportunity for sleep **[3,5]**. By contrast, one or two short nights before surgery may represent an acute stress response. Both patterns can matter, but they likely differ in mechanism: chronic insomnia may reflect sustained hyperarousal and comorbidity burden, whereas acute sleep restriction may transiently impair attention, executive control, pain tolerance, and stress regulation.

2.2 Practical preoperative sleep assessment

A structured sleep history should ask about habitual bedtime and wake time, sleep duration, sleep latency, awakenings, naps, snoring, witnessed apneas, nocturnal choking, restless legs symptoms, sedative use, alcohol, and daytime sleepiness. The Pittsburgh Sleep Quality Index (PSQI) remains one of the most widely used multidimensional tools for subjective sleep quality over the preceding month **[6]**. The Insomnia Severity Index provides a brief measure of insomnia symptoms and their daytime impact **[7]**. Neither tool is a substitute for a formal sleep diagnosis, but both are useful for documenting a baseline and identifying patients whose sleep complaints are clinically meaningful.

Obstructive sleep apnea (OSA) deserves separate attention because it is common, underdiagnosed, and relevant to both respiratory and neurocognitive risk. The STOP questionnaire and its expanded STOP-Bang version were developed for rapid perioperative screening **[8]**. Society of Anesthesia and Sleep Medicine guidance supports systematic preoperative identification of known or suspected OSA and individualized planning according to comorbidities, surgery, opioid requirements, and monitoring resources **[9]**. A meta-analysis of surgical cohorts confirmed that STOP-Bang has high sensitivity for moderate-to-severe and severe OSA, although specificity is limited and a positive screen is not equivalent to polysomnographic diagnosis **[10]**. The American Society of Anesthesiologists likewise recommends risk-stratified perioperative management, continuation of positive airway pressure when appropriate, and attention to postoperative respiratory depressant exposure **[11]**.

The goal of screening is not to create unnecessary surgical delay. Rather, it is to identify untreated severe disease, document home positive-airway-pressure use, anticipate airway and opioid sensitivity, and recognize that fragmented sleep and intermittent hypoxemia may coexist with reduced cognitive reserve.

3. Postoperative Cognitive Recovery in Older Adults

3.1 Terminology and clinical spectrum

The 2018 consensus nomenclature proposed the umbrella term perioperative neurocognitive disorders (PND) to align perioperative cognitive changes with standard neurocognitive diagnostic frameworks **[12]**. Within this framework, delirium is an acute fluctuating disorder of attention and awareness occurring in the early postoperative period; delayed neurocognitive recovery refers to objectively measured decline within approximately 30 days; and postoperative neurocognitive disorder describes impairment persisting from 30 days to 12 months. A recent clinical guide emphasizes the importance of preoperative cognitive baseline, validated delirium assessment, and appropriately timed postoperative cognitive testing **[13]**.

This distinction matters when interpreting sleep research. Many older studies used the term postoperative cognitive dysfunction (POCD) without uniform diagnostic thresholds, while newer work increasingly separates delirium from longer-term decline. Sleep disturbance may influence both, but the strength and quality of evidence differ substantially across outcomes.

3.2 Postoperative delirium

Postoperative delirium remains common after major surgery in older adults, with incidence varying widely according to age, baseline cognition, urgency, surgical type, frailty, and the method and frequency of assessment **[14]**. Delirium is usually multifactorial: predisposing factors such as cognitive impairment, sensory loss, frailty, multimorbidity, and sleep disturbance interact with precipitating factors such as surgery, infection, pain, medications, hypoxemia, and metabolic derangement **[15]**.

The clinical consequences extend beyond a transient episode of confusion. Meta-analytic data associate postoperative delirium with mortality, complications, prolonged hospitalization, institutional discharge, and functional decline **[16]**. Delirium is also associated with worse subsequent cognitive outcomes **[17]**, and across hospitalized older adults it is linked to an increased risk of later dementia **[18]**. These associations support the concept that delirium may be both an acute manifestation of cerebral vulnerability and a contributor to longer-term cognitive deterioration.

3.3 Delayed neurocognitive recovery and longer-term decline

The landmark ISPOCD1 study established that measurable cognitive decline can persist after major noncardiac surgery in older adults **[19]**. Subsequent work identified advanced age, lower educational attainment, pre-existing disease, postoperative complications, and other markers of reduced reserve as important predictors **[20]**. Modern interpretation is more cautious than the early concept of a single anesthesia-induced syndrome; postoperative cognitive trajectories reflect the combined effects of baseline cognition, surgical inflammation, perioperative complications, sleep and circadian disruption, pain, medication exposure, and recovery environment.

For sleep research, the key methodological requirement is therefore a preoperative cognitive baseline. Without baseline testing, poor postoperative scores may reflect unrecognized pre-existing impairment rather than new decline. Likewise, a single sleep questionnaire cannot fully separate chronic insomnia, OSA, circadian disruption, depression, and acute preoperative anxiety.

4. Clinical Evidence Linking Preoperative Sleep to Cognitive Recovery

4.1 Preoperative sleep disturbance and postoperative delirium

The clinical literature increasingly supports an association between disturbed preoperative sleep and postoperative delirium. A 2026 systematic review and meta-analysis of older surgical patients reported that preoperative sleep disturbance was associated with substantially higher odds of postoperative delirium across cardiac, orthopedic, and other noncardiac procedures **[21]**. Although definitions of sleep disturbance varied across included studies, the pooled evidence strengthens the argument that sleep is a meaningful preoperative risk domain rather than only a postoperative symptom.

The strongest recent prospective evidence comes from a multicenter cohort of 535 adults aged 60 years or older undergoing major noncardiac surgery. Preoperative sleep disturbance defined using the PSQI was associated not only with more early postoperative delirium but also with higher rates of postoperative cognitive dysfunction at 7, 30, 90, and 180 days **[22]**. The study also identified a higher PSQI threshold as a practical marker of cognitive risk. Because this was observational rather than interventional, it does not prove that treating sleep disturbance will prevent cognitive decline, but it provides a clinically relevant link between baseline sleep and a prolonged recovery trajectory.

Earlier systematic reviews reached broadly similar conclusions. One meta-analysis found an association between perioperative sleep disturbance and postoperative delirium, while another analysis of clinical studies likewise supported sleep disruption as a delirium risk factor **[23,24]**. Heterogeneity remains important: studies have used different sleep questionnaires, timing windows, surgical populations, anesthesia techniques, delirium instruments, and covariate adjustments. Nevertheless, consistency across designs makes preoperative sleep a reasonable component of risk stratification.

4.2 Objective sleep characteristics and delirium vulnerability

Subjective sleep quality may not capture the neurophysiological features most relevant to cognition. In a prospective pilot study of patients undergoing heart valve surgery, lower preoperative slow-wave sleep was associated with postoperative delirium **[25]**. Slow-wave sleep is particularly relevant to memory consolidation, synaptic homeostasis, autonomic regulation, and coordinated cerebrospinal-fluid dynamics. Objective measurements such as polysomnography or wearable-derived sleep continuity may therefore improve mechanistic specificity in future studies.

Outside strictly surgical cohorts, cumulative poor sleep burden in middle-aged and older adults has been associated with delirium during subsequent hospitalization **[26]**. This finding suggests that sleep may function as a longer-term marker of brain vulnerability. It also raises the possibility that the perioperative period unmasks an already fragile sleep-cognition system rather than creating the entire risk de novo.

4.3 Sleep duration and delayed cognitive recovery

Acute sleep loss immediately before surgery may also matter. A prospective cohort study reported that short sleep duration on the night before surgery was associated with postoperative cognitive decline in older patients **[27]**. Another clinical study found a correlation between preoperative sleep disorders and delayed neurocognitive recovery **[28]**. These studies are smaller than the delirium literature and use different cognitive definitions, so their results should be considered hypothesis-generating rather than definitive.

The temporal pattern is biologically plausible. Sleep restriction before surgery may reduce attention and executive reserve at the same time that anesthesia, inflammation, pain, and environmental sleep loss create additional cognitive load. In a patient with mild baseline impairment, a small reduction in reserve may be sufficient to cross the clinical threshold for delirium or measurable postoperative decline.

4.4 Obstructive sleep apnea

OSA is a particularly important phenotype because it combines sleep fragmentation with recurrent intermittent hypoxemia, sympathetic activation, vascular stress, and frequent cardiometabolic comorbidity. In a prospective study of older adults undergoing elective knee replacement, pre-existing OSA was associated with a markedly higher incidence of postoperative delirium **[29]**. The study was relatively small, but it established a clinically plausible signal that has influenced subsequent perioperative research.

Evidence that perioperative positive-airway-pressure therapy specifically prevents delirium is less certain. In the PODESA randomized trial, older adults with newly diagnosed OSA undergoing hip or knee arthroplasty were assigned to auto-titrating positive airway pressure or routine care. Delirium incidence was unexpectedly low, treatment adherence declined over successive postoperative nights, and the trial was unable to demonstrate a statistically significant reduction in delirium **[30]**. More broadly, a systematic review and meta-analysis found that perioperative CPAP improves sleep-apnea severity and may favor some postoperative outcomes, but the evidence was insufficient to establish a clear effect on all complications **[31]**. Thus, OSA should be treated for established respiratory and cardiovascular indications, while its cognitive-protective benefit remains an important research question.

5. Biological Mechanisms

5.1 Sleep-dependent brain clearance and synaptic homeostasis

Sleep supports multiple restorative processes that may become especially important in the aging brain. Experimental work demonstrated that sleep increases interstitial fluid exchange and accelerates clearance of metabolites, including beta-amyloid, from the brain **[32]**. Human imaging has since shown large, coupled oscillations in neural activity, hemodynamics, and cerebrospinal fluid during non-rapid-eye-movement sleep, providing a physiological link between deep sleep and fluid dynamics **[33]**. These findings do not establish that one night of poor sleep causes postoperative cognitive dysfunction, but they support the broader concept that repeated loss of consolidated sleep may reduce the brain's ability to maintain metabolic homeostasis.

Sleep also participates in synaptic renormalization and memory integration. The synaptic homeostasis framework proposes that wakefulness is associated with net synaptic potentiation and that sleep restores efficiency while preserving salient memory traces **[34]**. An older adult entering surgery after fragmented or curtailed sleep may therefore begin the perioperative period with impaired memory encoding, reduced attentional stability, and less capacity to compensate for acute physiological stress.

5.2 Sleep, cognitive reserve, and aging

Population studies show that both very short and very long habitual sleep are associated with worse cognitive trajectories, although reverse causation and comorbidity remain difficult to exclude. A longitudinal analysis found an association between sleep duration and subsequent cognitive decline **[35]**, while another large cohort linked shorter sleep in midlife and later life with higher dementia incidence **[36]**. In perioperative interpretation, these data support sleep as a marker of brain health rather than a simple dose-response exposure. Chronic sleep disturbance may identify patients with neurodegenerative pathology, depression, vascular disease, pain, or frailty that independently increase postoperative cognitive risk.

5.3 Neuroinflammation

Surgery induces a systemic inflammatory response that can signal to the brain through humoral, neural, endothelial, and immune pathways. Preclinical work in aged mice suggests that preoperative sleep disturbance exaggerates surgery-induced neuroinflammation and neuronal injury **[37]**. Experimental models of perioperative neurocognitive disorder have also identified interleukin-6 as an important mediator of postoperative cognitive change **[38]**. These findings provide a mechanism by which sleep loss and surgical inflammation could interact rather than simply add their effects.

The relevance to older adults is heightened by immunosenescence and 'inflammaging.' A primed neuroimmune system may respond disproportionately to a second hit. Sleep disruption before surgery can therefore be conceptualized as a vulnerability amplifier: it may raise baseline inflammatory tone, alter microglial responsiveness, and make the postoperative inflammatory response more disruptive to attention and memory networks.

5.4 Blood-brain barrier dysfunction

Blood-brain barrier (BBB) integrity is another plausible convergence point. In older adults undergoing noncardiac surgery, biomarkers and imaging-related evidence have implicated BBB dysfunction in postoperative delirium **[39]**. Experimental sleep restriction itself can impair BBB function **[40]**, and sleep loss has been shown to disrupt interactions between brain endothelial cells and pericytes **[41]**. These findings suggest that poor preoperative sleep could reduce barrier resilience before the inflammatory and hemodynamic stresses of surgery.

A more permeable or dysfunctional BBB may increase central exposure to circulating cytokines and other inflammatory mediators. This mechanism remains incompletely validated in humans, but it provides a coherent explanation for why sleep disturbance may be most harmful in patients who also experience infection, hypotension, major blood loss, or other postoperative insults.

5.5 Circadian and neuroendocrine disruption

Delirium is characterized by profound disruption of sleep-wake organization and diurnal behavior. Experimental work has identified circadian transcriptional pathways capable of modulating cognition in delirium models **[42]**. Surgery, anesthetic exposure, nighttime light, frequent observations, pain, and fasting can all alter circadian timing. Older adults already have reduced circadian amplitude, making them more susceptible to these zeitgeber disturbances. Chronic insomnia and recurrent short sleep are also associated with altered hypothalamic-pituitary-adrenal activity, including changes in cortisol regulation **[43]**. Excessive perioperative stress signaling may impair hippocampal function, worsen glucose regulation, and amplify inflammatory pathways. Accordingly, circadian preservation and sleep optimization may be valuable even if no single pathway proves dominant. Prolonged sleep deprivation can trigger broad systemic inflammatory activation in experimental models **[44]**, emphasizing that severe sleep loss is a multisystem stressor rather than an isolated symptom.

6. Preoperative and Perioperative Management

6.1 Treat the sleep disorder, not only the symptom

Management begins with phenotype. A patient with chronic insomnia, a patient with untreated severe OSA, and a patient who slept poorly for two nights because of surgical anxiety should not receive the same intervention. For chronic insomnia, behavioral treatment, regular sleep-wake timing, management of pain and nocturia, reduction of late caffeine and alcohol, and treatment of depression or anxiety are preferable to reflexive sedative prescribing **[5]**. When surgery is imminent, a brief behavioral plan can still focus on stable wake time, adequate daytime light and activity, avoidance of excessive daytime napping, and a realistic rather than perfectionistic expectation of sleep on the night before surgery.

Melatonin-pathway interventions are biologically attractive because they may support circadian organization without the anticholinergic burden of many traditional sedatives. A randomized trial in older hospitalized patients found that ramelteon reduced delirium, although the population was not limited to surgical patients **[45]**. This result helped stimulate perioperative trials but should not be generalized as proof that ramelteon prevents postoperative cognitive disorders in all older adults.

6.2 Sedation strategy and dexmedetomidine

Dexmedetomidine produces a sedative state with some electrophysiological similarities to non-rapid-eye-movement sleep and has been studied extensively for delirium prevention. In a large randomized trial, low-dose postoperative dexmedetomidine reduced delirium in older patients after noncardiac surgery **[46]**. Its benefit is probably multifactorial and cannot be attributed solely to sleep improvement. Bradycardia, hypotension, oversedation, and context-specific monitoring requirements also limit indiscriminate use. Dexmedetomidine should therefore be considered an anesthesia and critical-care strategy rather than a routine preoperative insomnia treatment.

6.3 Melatonin and melatonin-receptor agonists

The overall perioperative evidence for melatonergic therapy remains mixed. A 2026 systematic review and meta-analysis of randomized trials in older surgical patients found a reduction in postoperative delirium with melatonin-containing interventions, while ramelteon did not show a statistically significant pooled benefit; the certainty of evidence was low and heterogeneity was substantial **[47]**. These results support further study but do not justify a universal prophylactic prescription. Dose, timing, baseline sleep phenotype, surgery type, and concomitant delirium-prevention measures may all modify response.

For an individual older patient already taking melatonin safely, continuation may be reasonable when consistent with local practice. Starting a new drug solely to improve cognition should be based on institutional protocols and patient-specific risks rather than the assumption that any hypnotic-induced sleep is physiologically equivalent to natural sleep.

6.4 Delirium prevention and medication review

Major geriatric and anesthesiology guidelines emphasize multicomponent prevention rather than a single pharmacological solution. The American Geriatrics Society guideline recommends identifying high-risk older adults, avoiding deliriogenic medications when possible, optimizing pain control, promoting mobility and orientation, and using nonpharmacological prevention strategies **[48]**. The updated European guideline similarly stresses structured risk assessment, validated delirium screening, and multidisciplinary prevention throughout the perioperative pathway **[49]**.

Medication review is particularly important when a patient reports poor sleep. The 2023 AGS Beers Criteria identify benzodiazepines, nonbenzodiazepine benzodiazepine-receptor agonists, and strongly anticholinergic drugs as potentially inappropriate in many older adults because of risks including cognitive impairment, falls, and delirium **[50]**. First-generation antihistamines should not be treated as benign sleep aids. Likewise, routine escalation of nighttime sedatives in the hospital can worsen respiratory depression, mobility, and cognition even when the patient appears to sleep longer.

6.5 Nonpharmacological sleep protection

Nonpharmacological delirium-prevention programs provide the most transferable model for sleep-centered perioperative care. A classic multicomponent intervention in hospitalized older adults targeted cognitive impairment, sleep deprivation, immobility, dehydration, and sensory impairment and reduced incident delirium **[51]**. Although not a surgical-only study, it established the principle that sleep should be addressed within a broader bundle rather than in isolation.

Simple environmental measures can improve nighttime conditions. Randomized studies in intensive-care populations have reported better sleep quality and lower delirium scores with earplugs and eye masks **[52]**, while polysomnography-based work found that these measures can reduce prolonged awakenings and increase deep-sleep duration when tolerated **[53]**. An American Thoracic Society research statement emphasizes the complex environmental, physiological, and treatment-related causes of sleep and circadian disruption in critical illness and supports systematic sleep-promoting care rather than reliance on sedatives **[54]**.

A perioperative sleep bundle can therefore include daytime mobilization and light exposure, reduction of unnecessary nighttime noise and observations, clustering of care, correction of hearing and visual impairment, orientation cues, comfortable temperature, multimodal analgesia, early removal of catheters when appropriate, and preservation of the patient's usual positive-airway-pressure therapy.

6.6 Obstructive sleep apnea pathway

OSA prevalence rises with age, and meta-analytic evidence indicates a high global burden among older adults **[55]**. A high STOP-Bang score should trigger review of prior sleep studies, severity, symptoms, comorbid pulmonary hypertension or hypoventilation, and home PAP adherence. Patients using CPAP or bilevel PAP should generally be encouraged to bring their equipment or use hospital-compatible therapy. Opioid-sparing analgesia, appropriate positioning, and postoperative oxygenation and ventilation monitoring are central components of care **[9–11]**.

For previously undiagnosed OSA, the urgency of formal sleep testing depends on the clinical scenario. Elective surgery may provide an opportunity for evaluation when severe symptoms or cardiopulmonary instability are present, but most positive screens can be managed using a presumptive risk pathway without automatic cancellation. The cognitive rationale is complementary to the respiratory one: avoiding recurrent hypoxemia and severe sleep fragmentation may preserve attention and reduce physiological stress during the period of greatest delirium vulnerability.

7. A Practical Preoperative Brain-and-Sleep Pathway

A pragmatic approach can be incorporated into routine older-adult preoperative assessment without requiring formal polysomnography for every patient. First, document baseline cognition and ask a brief sleep history. Second, quantify subjective sleep quality with the PSQI when sleep complaints are present and use the Insomnia Severity Index when chronic insomnia is suspected **[6,7]**. Third, screen for OSA with STOP-Bang and identify established PAP users **[8–10]**. Fourth, review sedatives, anticholinergics, alcohol, opioids, and other medications that may worsen sleep architecture, respiration, or delirium risk **[50]**. Fifth, communicate sleep-related risk to the anesthesia, surgical, nursing, and geriatric teams so that postoperative monitoring and delirium prevention begin before the first night after surgery.

The pathway should also establish realistic treatment goals. The objective is not to guarantee a perfect preoperative night of sleep. It is to reduce avoidable sleep loss, treat clinically important disorders, prevent abrupt withdrawal of usual therapies, and avoid iatrogenic attempts to 'fix' sleep with medications that may increase cognitive risk. In high-risk patients, sleep optimization should sit alongside correction of anemia and dehydration, sensory aids, nutrition, pain planning, early mobilization, and delirium surveillance **[48,49,51]**.

8. Obstacles and Limitations

The current literature has several important limitations. First, sleep disturbance is measured inconsistently. Some studies use the PSQI, others a single sleep-duration item, chart diagnoses, STOP-Bang scores, polysomnography, or postoperative sleep ratings. These exposures are not interchangeable. A patient with insomnia, a short sleeper, and a patient with severe OSA may share poor subjective sleep but have different pathophysiology and treatment needs.

Second, residual confounding is substantial. Poor sleep in older adults is associated with pain, depression, anxiety, frailty, vascular disease, neurodegeneration, medication burden, and socioeconomic factors. Many of these variables independently predict delirium and cognitive decline. Even well-adjusted observational studies cannot fully determine whether sleep disturbance is causal, a marker of reduced reserve, or both.

Third, postoperative cognitive outcomes are heterogeneous. Delirium is an acute clinical syndrome requiring repeated validated assessment, while delayed neurocognitive recovery requires baseline and follow-up testing with appropriate normative methods. Studies that label any low postoperative test score as POCD risk misclassification. Fourth, intervention studies often target postoperative sleep rather than preoperative treatment, making it difficult to determine whether modifying the baseline sleep state alters the trajectory of cognitive recovery.

Finally, the biology of sleep is difficult to reproduce pharmacologically. Sedation can increase apparent sleep time without restoring normal sleep architecture or circadian timing. Future studies should therefore measure both clinical outcomes and sleep physiology rather than assume that a sedating intervention is intrinsically sleep restorative.

9. Future Outlooks

The next phase of research should move from association toward mechanism-based intervention. Large prospective cohorts should combine validated subjective tools with actigraphy, wearable oximetry, or targeted polysomnography and should measure baseline cognition, frailty, depression, pain, medication exposure, and biomarkers. This would allow separation of insomnia, circadian disruption, short sleep, OSA, and objectively reduced slow-wave sleep into clinically meaningful phenotypes.

Randomized trials are needed to test whether treating sleep before surgery improves postoperative cognition. Potential strategies include brief preoperative cognitive-behavioral therapy for insomnia, rapid PAP optimization for untreated OSA, personalized circadian interventions using timed light and activity, and multicomponent perioperative sleep bundles. Trials should assess delirium daily for several postoperative days and continue cognitive follow-up through at least 30 and 90 days. They should also report adherence, because a theoretically effective intervention cannot improve outcomes when patients do not or cannot use it.

Precision approaches may ultimately be more effective than universal prophylaxis. A patient with severe OSA and nocturnal hypoxemia may benefit most from respiratory stabilization, while a patient with chronic insomnia and hyperarousal may benefit from behavioral therapy, and a frail patient with fragmented sleep and sensory impairment may benefit most from an environmental and delirium-prevention bundle. Digital sleep monitoring could make such stratification feasible, but algorithms will require external validation in older, multimorbid surgical populations.

Preoperative sleep should therefore become part of perioperative brain-health assessment rather than remain an incidental symptom. The available evidence supports its use for risk identification today, while the question of how much cognitive benefit can be achieved by targeted treatment remains open. If future trials demonstrate causality, sleep optimization could become one of the few modifiable preoperative interventions capable of influencing both immediate delirium and longer-term cognitive recovery.

Disclosure

The authors have no financial interest to declare in relation to the content of this article.

Authorship

All authors have a substantial contribution to the article.

Funding

No Funds.

Conflicts of interest

There are no conflicts of interest.

Acknowledgments

The authors acknowledge the clinicians and researchers whose work has advanced perioperative brain-health and sleep medicine.

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