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Research Article

Sleep Changes After Acquired Brain Injury and its Impact on Recovery Outcomes: A Scoping Review of Existing Literature

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ABSTRACT

Background: While sleep and Acquired Brain Injuries (ABI) have an intimate relationship, there are limitations to elucidating the underlying aetiology of sleep disturbances and the effects of sleep on ABI outcomes. Differing disease trajectory, iatrogenic influences and the numerous challenges in sleep research further confound the relationship between sleep and ABI.

Objectives: To summarize the research findings of the relationship between ABI and sleep, and the relationship between post-ABI sleep disturbances and ABI outcomes as well as the methodological limitations that would affect the applicability of the results.

Design: A scoping review was conducted from research published within 5 years from 2019-2023.

Method: Sources of evidence were PubMed, Cumulative Index of Nursing and Allied Health Literature (CINAHL), Cochrane and Embase. MeSH terms used were "sleep and stroke or brain injury" in PubMed and adapted accordingly to other search engines. Studies were included if they met the criteria: Adult sample >21 years old; papers with objective or subjective measures of sleep; papers published within 5 years from 2019-2023; papers that measured sleep after stroke or brain injury; unpublished papers (conference abstracts); translated papers (official translations from other languages); observational or epidemiological quantitative studies.

Result: The search yielded 4336 articles. After 1472 duplicates were removed, 2864 abstracts were screened. 93 articles were found to meet the inclusion criteria. There were ample descriptions of sleep disturbances measured both subjectively and objectively occurring after stroke, but no convincing elucidation of the extent of causality and causal mechanisms of stroke and disturbed sleep.

There are also convincing correlations between sleep disturbance and poorer stroke outcomes, however the findings suffer from a lack of clarity of which type of sleep disturbance causes which specific outcome and the lack of a plausible causal mechanism.

There is a clear relationship between Traumatic Brain Injuries (TBIs) and sleep related disorders and symptoms. The wide variety of sleep measures and their combinations allowed for a greater breadth of evidence but the heterogeneity in measures used, and methodologies poses a challenge for direct comparison between studies.

There is also a general direction towards sleep disturbances having significant effects on patient outcomes, of particular interest would be the time taken to recover post-TBI. Patients were also more likely to experience psychological issues like depression and anxiety with poor sleep.

Conclusion: Future research and interventional trials can only proceed after the dilemma between small low-powered studies using precise but resource-heavy measurements and large high-powered studies using imprecise measurements can be satisfactorily resolved and there is more detailed understanding of post-ABI sleep pathophysiology in humans.

Keywords: Sleep; Stroke; Brain injury; Traumatic; Haemorrhagic

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INTRODUCTION

Sleep is a complex neurological phenomenon that involves the whole brain at structural, cellular and molecular levels and is not yet completely understood. The sleep state occurs as a function of the interplay between the homeostatic sleep drive (Process S) and the circadian rhythm (Process C). The neuroanatomical locus of Process S has yet to be identified but is postulated to involve the inhibition of arousal regions of the brain by the anterior hypothalamus. On the other hand, the suprachiasmatic nucleus of the hypothalamus has been identified as the "body clock" that reacts to light input and other environmental cues to regulate the onset and timing of the sleep-wake cycle. In addition, the sleep state is composed of cyclical stages of Non-Rapid Eye Movement (NREM) and Rapid Eye Movement (REM) sleep that alternate with each other as sleep progresses, usually at night. NREM sleep is a function of the network connecting the brainstem, thalamus and cortex, whereas Rapid Eye Movement (REM) sleep is a function of the pontine neurons [1].

Sleep is believed to be a vital process for survival as it has important functions of energy conservation, body repair and restoration and neural reorganization and growth [2]. Indeed, the evidence base for the health effects of poor sleep quality and or quantity is broad. A review of 85 meta-analyses by Gao et al., showed that long sleep duration was associated with an increase in all-cause mortality and stroke, short sleep duration was associated with increased risk of obesity and poor sleep quality was associated with increased risk of diabetes mellitus [3]. Sleep problems has also been shown to be associated with all-cause cognitive disorders and also believed to be a causal factor in various major mental disorders [4,5]. Hence, the growing interest in sleep as a potential therapeutic target to improve outcomes in many different medical disorders.

However, it can be anticipated that in such a complex neurological process as sleep, there can be a myriad of structural, functional, chemical and or environmental contributors to disturbances in sleep quantity and quality both in silo and in combination. Therefore, a clear understanding of the relationships between and among contributing factors and the nature of sleep disturbances is necessary to inform targeted and appropriate therapeutic interventions.

Although several systematic reviews of the literature about sleep after specific types of brain injury have been performed, it is believed that in view of the reasons explained above, a scoping review of the literature was still necessary to understand and summarize the research findings of the relationship between ABI and sleep and the relationship between post-ABI sleep disturbances and ABI outcomes as well as the methodological limitations that would affect the applicability of the results.

This scoping review was guided by the following research questions:

1. What is the spectrum of both subjective and objective sleep disturbances after the major forms of ABI, namely trauma and stroke (both ischaemic and haemorrhagic)?
2. What is the postulated mechanism that leads to sleep disturbances after ABI?
3. What is the impact of post-ABI sleep disturbances on ABI outcomes?
4. What is the postulated mechanism that leads to poor outcomes in post-ABI individuals with sleep disturbances?
5. What were the methodological strengths and weaknesses of the reviewed research and how do they affect future research and clinical practice?

The objective of this review was to aid clinicians into adopting a more informed approach towards the management of ABI overall and ABI-related sleep disturbances and to aid future researchers to improve and innovate study designs that further our understanding of sleep with or without ABI and the potential of treating sleep disturbances to improve ABI outcomes. The combined magnitude of the impact of ABI on population health is obvious and needs no elaboration, adding to the importance of this review [6,7].

MATERIALS AND METHODS

A scoping review was conducted in accordance with the Arksey et al., [8]. The five steps adopted here was first described by Levac et al., and modified accordingly [9]. The scoping review was done with these rationales in mind: To investigate the scope, breadth and type of pre-existing literature on a topic or question; to assess the need and value of furthering research with a systematic review; to unify, summarise and share the findings to relevant research bodies; and to identify any potential research gaps to help in the conception and design of future research [8,9]. Furthermore, we adapted our scoping review according to the Preferred Reporting Items for Systematic reviews and Meta-Analyses extension for Scoping Reviews (PRISMA-ScR).

Selection of patients

Identifying research question: All authors were involved in writing the research questions through discussions and review of pre-existing literature on the relationship between ABI and sleep. These studies included the effects of ABI on sleep, associated sleep disturbances and the impact of sleep problems on the outcome of ABI recovery. Specifically, we sought to uncover (i) The prevalence and variety of sleep disturbances after ABI, (ii) Subjective and objective sleep disturbances after ABI, (iii) Relationship between sleep disturbances and ABI outcomes and recovery, (iv) Neuroimaging characteristics of abnormal sleep after ABI and (v) The function of sleep in post ABI recovery. With those questions as guidelines, our primary research question was designed as "What is the effect of ABI on patients sleep and how do sleep disturbances affect the outcomes of ABI recovery?"

Identifying relevant studies: Our search strategy was designed to include important keywords that best answered our research questions. We used "sleep and stroke or brain injury" in PubMed and adapted this search strategy to other search engines. Sources of evidence that we selected from were PubMed, CINAHL, Cochrane and Embase. The quality of these databases and breadth of their medical content were relevant to our study. Papers were selected if they met the following criteria: Adult sample>21 years old; papers with objective or subjective measures of sleep; papers published within 5 years from 2019-2023; papers that measured sleep after stroke or brain injury; unpublished papers (conference abstracts); translated papers

(official translations from other languages); observational or epidemiological quantitative studies.

Exclusion criteria were as follows: Paediatric population; interventional trials; animal studies; papers that measured pre-injury sleep; narrative reviews; expert opinions; validation studies for questionnaires; statements; study protocols; studies reporting psychometric properties of tools screening for depression, cognitive impairment and or sleep; studies that used depression, cognitive impairment, or abnormal sleep as a subject exclusion criterion; studies that include other neurological diseases.

Selecting the studies: Our literature search yielded 2864 abstracts after removing 1472 duplicates. To assess the eligibility of the articles, we derived a two-stage screening process. At the first level of screening, two authors reviewed all abstracts briefly and excluded 1871 articles that did not meet our requirements. A third and fourth author were included at the next level of screening to filter the remaining 993 abstracts and exclude 900 articles at this stage.

The final 93 papers were selected for our scoping review and read in full prior to data charting. The screening algorithm is described in Figure 1, which was generated by Covidence. Covidence, a web-based collaboration software platform that streamlines the production of systematic and other literature reviews, was extensively used as a database and in the screening process. Covidence was first used to filter and tag the papers, in which the reviewers then reconvened to assess the ones that were ambiguous.

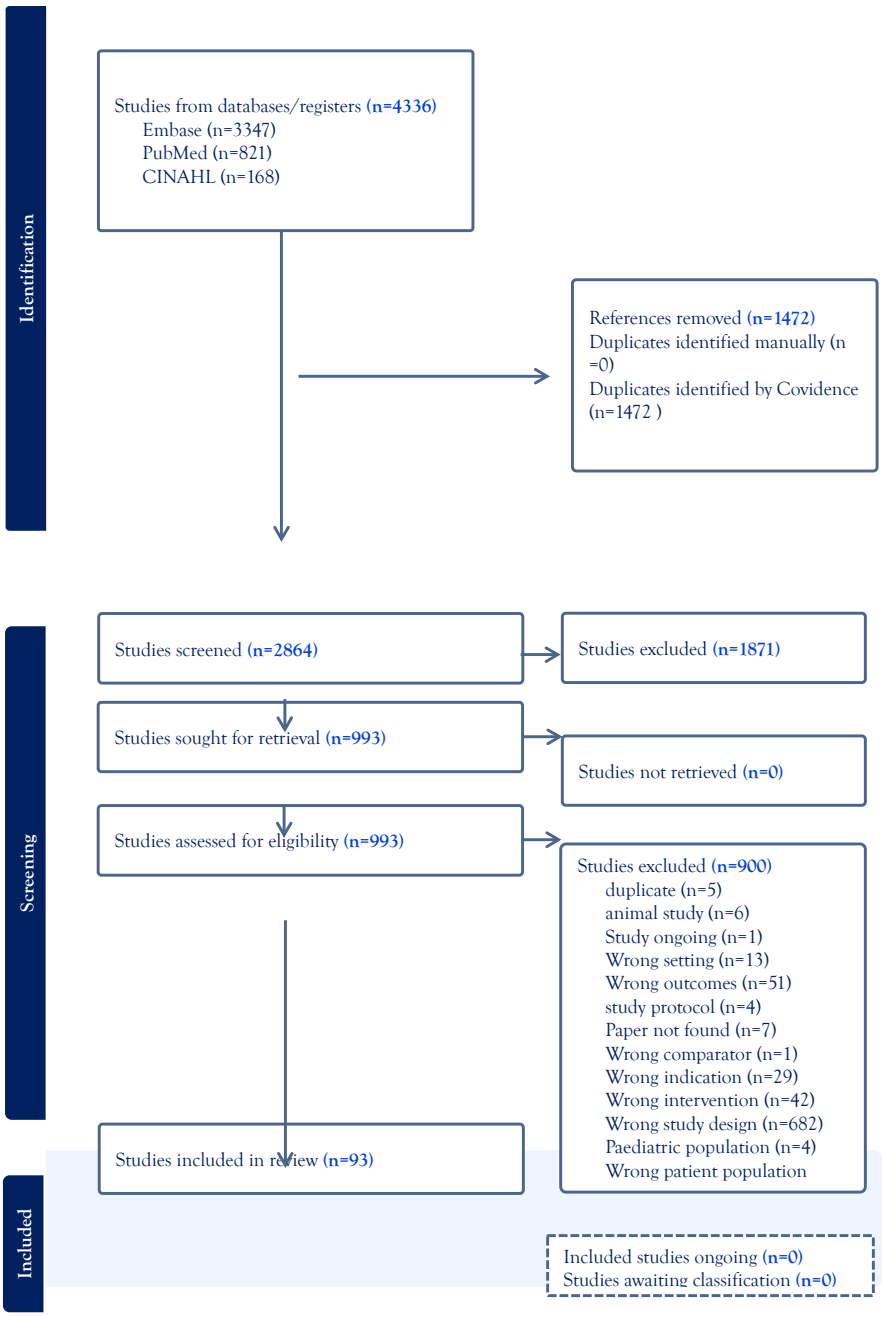

Figure 1: PRISMA flow chart describing the identification and screening protocol for the selection of literature and articles.

Charting the data: According to our research question, we sieved and categorized the papers into two main themes: Stroke and Traumatic Brain Injury (TBIs). We then further subdivided the papers into two more groups: Effect of stroke/TBIs and ABIs on sleep and effect of post-ABI sleep disturbances on ABI outcomes. Finally, we then split the papers according to objective and subjective measurements of sleep. We recorded information as follows: Author and year of study; title; study design and main objective; study sample inclusion/exclusion, sample size; primary outcome measure and other measures; summary of results. The included articles were described in the summary tables below Supplementary Tables 1-4.

Collating, summarising and reporting the results: According to the devised themes, a structured framework was designed and adopted for the presentation of the sieved articles. The articles were then independently classified into the groups identified in step 4 by two reviewers. The focus of the presentation of these results were on the bimodal relationship of sleep and ABIs and the nature of sleep quality assessment. All authors and reviewers contributed to the decision of themes and summarizing the results of the papers.

RESULTS

Sleep disturbances after stroke

A total of 39 papers were reviewed that described the sleep changes and disturbances that occurred after a stroke. Only questionnaires for subjective report of sleep or clinical interviews according to diagnostic criteria were used as the primary sleep measures for 7 of the studies and 6 studies used a combination of both subjective and objective sleep measures as shown in Table 1. The remaining 26 studies that used objective measures mostly used either fully attended or portable overnight PSG to assess for Sleep-Disordered Breathing (SDB) and a handful of studies used Electroencephalogram (EEG) to assess for sleep architecture, or actigraphy to assess sleep-activity rhythms and only 1 study used a combination of PSG, actigraphy, skin temperature thermochromes and 24-hour blood sampling for melatonin and clock-gene profiles for a comprehensive measurement of sleep pathology. Majority of the study designs were either cross-sectional or retrospective chart reviews which are prone to limitations in addressing causal relationships or introducing selection bias. The few longitudinal studies were limited by a short duration of follow-up ranging from 3 months to 2 years post-stroke.

Among the 14 studies reviewed that used subjective sleep measures, the most used was the Pittsburgh Sleep Quality Index (PSQI) that measured overall sleep quality and screened for sleep disorders, followed by the Epworth Sleepiness Scale (ESS) which measured daytime sleepiness.

The heterogeneity of measures used meant that overall comparability between studies is limited. Nevertheless, there is general agreement that there is a high prevalence of poor sleep quality and Excessive Daytime Sleepiness (EDS) after stroke except for 1 cross-sectional study of 35 community-dwelling older adults with chronic stroke that did not show a difference in PSQI-measured sleep quality from non-stroke age and gender-matched controls, even though objectively measured actigraphy data showed that the stroke subjects had longer sleep duration and greater sleep fragmentation [10-20]. This raises the question of the reliability of subjective sleep measures perhaps in specific subgroups. Also, there does not seem to be a structural correlation with sleep disturbances in most studies except for possible increased daytime sleepiness in thalamic strokes and poorer sleep quality in right-sided middle cerebral artery strokes [13-16].

An important issue to address is whether stroke itself can cause incident sleep problems or disorders. To answer this, the study would have to ensure that study subjects had normal sleep prior to the stroke and had incident sleep disturbances starting from the acute post-stroke period that persists after discharge from hospital to account for the possible confounding effects of the hospital environment on subjects' sleep.

Pajediene et al., studied 66 acute stroke patients within the first 10 days of stroke with a clinical interview and the ESS, and found that 50% of the sample had either new or stroke-exacerbated sleep complaints or symptoms mostly related to Obstructive Sleep Apnea (OSA) or insomnia [21]. On the other hand, the data about insomnia after stroke is conflicting. A study done in 2022 reported that 15.6% of the study sample of Acute Ischemic Stroke (AIS) patients had de-novo insomnia by 3 months after stroke onset [22]. However, a 2-year prospective study of 437 AIS patients showed that insomnia does not occur de novo after stroke and that its severity also improves with time [23-27].

It was observed from the review of the studies that used objective sleep measures that there was a prominent research interest/emphasis on Sleep Disorder Breathing (SDB) after stroke with at least half of the study sample being affected [28-30]. However, a few studies demonstrated that non-SDB sleep disorders were equally prevalent and important, such as Periodic Limb Movement Disorder (PLMD) and Rapid Eye Movement Sleep Behavior Disorder (RBD) [31-34]. The occurrence and severity of SDB appears to be associated with Ischemic Stroke (IS), brainstem and cerebellar lesions and greater extent of cerebral small vessel disease [35-38]. However, the strength of the evidence is limited by the cross-sectional or retrospective study designs.

In terms of changes to sleep architecture and microarchitecture after stroke, this can only be determined by analysis of the PSG or EEG parameters. Consistently, there is reduced slow wave/deep sleep across studies [39-42], reduced proportion of REM sleep and even altered REM microarchitecture and N2 stage microarchitecture [43-47].

However, it was also observed that Hofmeijer et al., study using continuous EEG from patients in acute stroke units shows that the physical environment can potentially confound disturbed sleep patterns in a major way [48]. Pajediene et al., study was the only study found that also examined circadian abnormalities in the acute stroke period and showed possible circadian desynchrony in stroke subjects, but this may also have been confounded by external environmental factors [49].

In summary, there are ample descriptions of sleep disturbances measured both subjectively and objectively occurring after stroke, but the extent of causality by the stroke event and the causal mechanism between stroke and disturbed sleep has not been convincingly elucidated.

Impact of post-stroke sleep disturbances on outcomes

To review and summarize the findings on the impact of post-stroke sleep disturbances and stroke outcomes, a total of 24 studies were reviewed-9 using only subjective sleep measures, 12 using objective measures and 3 using both. Once again, the studies were heterogenous due to the varied use of sleep measures and different stroke outcomes of interest.

All except 2 of the studies using subjective sleep measures were cross-sectional in design and used either the PSQI alone or in combination with the ESS, Insomnia Severity Index (ISI) and or self-report. These studies generally reported a variety of poorer outcomes associated with sleep disturbances. These poorer outcomes were in terms of depression and anxiety specifically ESS score being associated with anxiety and insomnia being associated with more severe depression, physical function specifically mobility and activity levels and global outcomes that include future vascular events and death in the 3 years following index stroke [50-53]. Fatigue appears to be associated with poor sleep quality cross-sectionally but this association was not shown in a 2-year follow-up study where fatigue scores remained stable over time [23,54].

The data regarding the effect of sleep disturbances on cognitive performance were inconsistent despite that brief, albeit different, cognitive screens were used-Mini Mental State Examination (MMSE), Montreal Cognitive Assessment (MOCA) and ADAS-Cog. Davis et al., study used all 3 brief measures but failed to show a relationship between poor sleep quality and cognitive impairment in contrast with 2 other studies [50,55]. Interestingly, out of the 2 studies, Falck et al., showed that only older adults with stroke and poor sleep quality experienced larger deficits in cognitive performance [20]. The only study that used a neurocognitive battery of tests to measure cognition also failed to show a relationship, however this was likely because the study was a comparison of left and right hemispheric strokes [16].

The studies using objective measures of PSG, EEG and actigraphy were similarly mostly cross-sectional in design. The findings are more heterogenous not just because of the variety of outcome measures used, but also because different methods of sleep measurement produced different sleep variables ranging from the presence/absence of sleep disorder to different indices of sleep architecture. In terms of cognitive outcomes, shorter Total Sleep Time (TST) and degree of hypoxia are associated with cognitive impairment within 14 days after minor stroke [55]. Jaramillo et al., showed in a small sample of thalamic stroke patients that reduced slow wave activity on PSG/EEG was not associated with performance on a rigorous cognitive battery but Howell et al., showed that shorter continuous periods of REM sleep after infratentorial stroke was associated with poorer verbal learning/memory performance [13,44]. Older adults with chronic stroke with higher sleep fragmentation or low sleep efficiency on actigraphy were also shown to have poorer cognitive performance on the ADAS-Cog [20].

With regard to physical function, upper limb function does not appear to be associated with sleep measures by actigraphy, but other studies that measured the presence or severity of SDB generally showed an association with poorer outcomes in physical function both cross-sectionally as well as on follow-up between 3 to 12 months [56,57]. REM duration and poor overall sleep quality showed a similar association with physical outcomes [19,58]. This association of sleep fragmentation and lower sleep efficiency with poor physical outcomes was present in actigraphy studies as well [19,57]. A related outcome of physical fatigue was found to be associated with lower sleep efficiency as measured by an accelerometer at 9 months after stroke [59].

Conversely, improved outcomes were shown in 2 studies. An analysis of a medical records database showed that OSA patients with AIS were more likely to receive hyperacute stroke treatments, less likely to die during hospitalization and more likely to be discharged home [60]. In a different study of critically ill stroke patients, the presence of sleep phases on EEG monitoring tended towards improved outcomes even though the result was not statistically significant [45].

In summary, although the correlation between sleep disturbance and poorer stroke outcomes appears convincing, the findings suffer from a lack of clarity of which type of sleep disturbance causes which specific outcome and the lack of a plausible causal mechanism. In fact, a common limitation observed was that stroke type, aetiology, location, severity, and the presence of comorbidities were not adjusted for in study design and statistical analysis.

Sleep disturbances after TBI

To investigate the effects of Traumatic Brain Injuries (TBI) on sleep, 28 papers were reviewed in total. Amongst them, 12 papers utilized only objective measures to quantify sleep whereas the other 14 used only subjective measures and 2 used both. Amongst the studies using subjective measures, there was a variety of methodologies employed including cohort studies, longitudinal studies and cross-sectional studies. Most papers employed questionnaires such as the PSQI, ESS, Fatigue Severity Scale (FSS), Night-time Behavioural disturbances domain of the Neuropsychiatric Inventory-Questionnaire (NPI-Q), Sleep and Concussion Questionnaire (SCQ), ISI and/or self-report. However, considering the unique nature of TBIs, several papers also used questionnaires targeted at symptoms of concussion in conjunction with the sleep questionnaires. These included the Rivermead Post-Concussion Symptoms Questionnaire, Behavioural Sleep Intervention (BSI), Brain Age Index (BAI) and the Brain Index Complaint Questionnaire (BICoQ).

The papers reviewed showed that TBIs of all severity have a significant impact on sleep quality and are associated with insomnia or hypersomnia, sleep disturbances, daytime sleepiness, fatigue, general sleep disorders and increased sleep latency [61-71]. Some studies also compared the effects of TBI on different demographics like age and gender which provide interesting perspectives relating to these parameters. For example, poor sleep after TBI was associated with older females [63]. Furthermore, older adults with TBI had higher prevalence of OSA, insomnia and daytime sleepiness and in general, females experienced significantly more severe changes in sleep compared to males [65,66]. Despite the heterogeneity of the subjective measurements used, there is a general agreement that TBIs result in poorer sleep as measured in multiple parameters. Only one paper with both subjective and objective sleep measures described no significant relationships between Sports-Related Concussions (SRC) and

sleep [72]. Furthermore, several papers describe the symptoms of sleep impairment to persist through 4-months, 6-months, 2 years and 5 years after injury with a trajectory of improvement but not back to baseline [64,67,71,73].

The studies using objective measurements used actigraphy, PSG, Magnetic Resonance Imaging (MRI), salivary cortisol and accelerometer (Readiband). Most studies employed formal overnight sleep studies whilst some used portable overnight sleep recording devices to track and analyse sleep architecture post-TBI. Most of the papers described a significant association with poorer sleep quality in terms of increased daytime sleepiness, increased sleep latency, sleep apnea, insomnia, increased quantity of sleep [74-81]. Furthermore, most of these associations were corroborated by sleep architecture disturbances including lower sleep efficiency, lower spindle density and increased proportion of slow-wave sleep and some possible circadian rhythm impairments [78,82-85].

In contrast, one paper described that the patients with TBI were more sleepy initially but their symptoms resolved by day 4 [86]. Nevertheless, most papers agreed that there is significant sleep disruption due to TBIs and structural abnormalities described on neuroimaging. Greater white matter damage was associated with higher slow-wave activity power and more severe complaints of cognitive fatigue. This correlation was only found in TBI patients [87].

Any disruption in sleep architecture can have significant downstream effects on sleep quality, daytime somnolence and systemic disease [88]. Fedele et al., showed that although NREM and REM sleep proportions were not affected but slow-wave sleep was reduced and absent in 35.3% of subacute moderate-severe TBI patients in Post-Traumatic Amnesia (PTA), furthermore, sleep periods were fragmented and median sleep efficiency was reduced [78]. This finding was corroborated in the subtle sleep microarchitecture changes that were found in hospitalized patients with acute moderate to severe TBI which included decreased spindle density and increased slow wave density [83]. However, it also showed contrasting results to Fedele et al., where slow-wave sleep was increased in TBI patients. Hence, the relationship between TBI and slow-wave sleep needs further investigation [78].

Boone et al., paper suggests that TBI can induce dysregulation of the circadian rhythm [89]. Similarly, the results from Ayalon et al., suggested minor TBIs might contribute to some circadian rhythm sleep disorders [90]. Other studies that examined this showed that whilst melatonin levels were higher than the normal range and cortisol levels were elevated compared to controls, there were no disruptions to the circadian rhythm in terms of saliva cortisol measurements and the circadian clock signal for which urinary excretion of 6-sulfoxymelatonin was measured to estimate pineal melatonin production, the main controller of circadian rhythm, suggesting that brain injuries directly alter the sleep-wake cycle rather than the circadian rhythm [84,85]. Taken together, the evidence suggests that there may be other neural mechanisms than the circadian rhythm that cause post-TBI Sleep-Wake Disturbances (SWD).

Finally, with the heterogenous nature of TBI populations, patient subgroups also include athletes and veterans who are exposed to high intensity physical training and requirements. Leng et al., cohort study discussed a significantly higher proportion of veterans who have sustained TBIs who developed clinically diagnosed sleep disorders, for which there is a robust 2-year time lag before onset of symptoms. This suggests that the impact of TBI on sleep could be more latent and long drawn [76]. Conversely, there are also studies in athletes with contrasting results for which one study showed athletes with TBIs spend less time asleep and in bed whereas another study showed increased time asleep and longer bedtime despite poorer sleep quality [79,81].

In summary, the wide variety of sleep measures and their combinations allowed for a greater breadth of evidence but the heterogeneity in measures used, and methodologies poses a challenge for direct comparison between studies. Furthermore, the studies reveal that there is a clear relationship between TBIs and sleep related disorders and symptoms. Whilst there are some interesting correlations, the causal mechanism of sleep disturbance in patients with TBI has not been fully elucidated, specifically the causal role of external trauma.

Impact of post-TBI sleep disturbances on outcomes

Finally, to summarize the effect of post-TBI sleep disturbances on TBI outcomes, 24 papers were reviewed. 16 papers used only subjective measurements of sleep whereas 6 used only objective measurements and 3 papers used both. Study methodologies included longitudinal, cohort and cross-sectional studies. More than half of them employed the PSQI for the subjective measurements whereas PSG was the preferred modality in the papers using objective measurements. In terms of the outcomes post-TBI, majority of papers focused on mood and cognition.

In the papers using subjective sleep measures, a variety of questionnaires were used. PSQI, ISI, STOP-BANG, ESS, FSS, self-reported symptoms and BICoQ were commonly used. Most papers described an association of poor sleep in TBI patients with poorer outcomes in multiple domains of assessment. One paper however, described that sleep changes did not mediate associations between TBIs and changes in cognitive performance [91]. Nevertheless, the poorer outcomes in patients post-TBI with sleep disturbances included anxiety and depression, poorer quality of life, fatigue, decline in cognitive ability and OSA with the added effect of its downstream influences [65,92-97]. Sleep apnoea is a common complication described in existing literature for patients with TBI that has been shown to have significant effects on cognition, specifically memory, complex processing speed and executive functioning [98,99].

The studies that employed objective measures used primarily actigraphy with a combination of PSG, other scales and scores and neuroimaging like MRI and Voxel Based Morphometry (VBM) and generally showed poorer outcomes for post-TBI patients with sleep dysfunction. Neuroimaging studies suggest possible structural localization for the pathway between sleep dysfunction and poor outcomes. For instance, poor sleep quality following TBI is significantly associated with lower cerebellar volume [100]. Two other papers described impairments in REM sleep resulting in poorer cognitive performances and quality of life [93,101]. Most significantly, there was one paper that described increased time required for recovery in post-TBI patients with poorer sleep efficiency and quality [102].

However, there were also some reviewed studies that had contrasting results, that despite poorer actigraphic sleep at night in severe TBI-patients, there was no association with functional outcome [103]. Furthermore, another study demonstrated that TST and sleep disturbance was not associated with neurobehavioral impairments after TBI [96]. These contrasting results could be attributed to the nature of the cohort studies having intrinsic bias in patient selection or the heterogenous severities of post-TBI patients that were recruited.

In summary, there is a general direction towards sleep disturbances having significant effects on patient outcomes, of particular interest would be the time taken to recover post-TBI. Patients were also more likely to experience psychological issues like depression and anxiety with poor post-TBI sleep. Furthermore, although there are attempts at elucidating and mapping the mechanism for poorer outcomes, they have yet to show causal relationships. Once again, the heterogeneity of the studies allows for much observational evidence but limited comparative data. Nevertheless, this strengthens the correlation between TBI and poor sleep, and provides much needed baseline data for future research.

DISCUSSION

The results of this scoping review will be discussed according to the research questions that guided the work.

What is the spectrum of both subjective and objective sleep disturbances after the major forms of ABI, namely trauma and stroke (both ischaemic and haemorrhagic)?

It can be generally concluded from this review that ABI patients subjectively experience poorer sleep quality, hypersomnia, insomnia, or daytime sleepiness, but this can be discordant with objectively measured sleep parameters. For stroke, there was abundant literature on SDB and other non-SDB related disorders. In terms of sleep architecture, there was evidence of reduced slow wave sleep in all forms of ABI. The current evidence for circadian rhythm abnormalities after ABI is rather preliminary and relatively scanty, hence firm conclusions cannot be made from this review. Unfortunately, these results do not lend themselves easily to translational applicability except that SDB appears to be the most obvious therapeutic target with an established form of clinical treatment.

What is the postulated mechanism that leads to sleep disturbances after ABI?

Unfortunately, the studies that were reviewed did not identify any postulated mechanism in terms of neuroanatomical correlation or sleep physiology disruptions. Admittedly, the papers included in the review were clinical research papers and not basic science or animal studies that specifically studied sleep pathophysiology. This is an important limitation of this review.

What is the impact of post-ABI sleep disturbances on ABI outcomes?

For both stroke and TBI, subjective poor sleep quality is consistently associated with poorer outcomes especially psychological ones like depression and anxiety. This is unsurprising because impaired sleep is part of the phenomenology of these psychiatric disorders. The association between outcomes such as physical function and cognitive function and objective sleep dysfunction is somewhat inconsistent. This is likely due to methodological limitations including bias in selection of controls, small sample sizes, cross-sectional designs and compromised specificity in the selected measures.

What is the postulated mechanism that leads to poor outcomes in post-ABI individuals with sleep disturbances?

None of the studies reviewed led to an identification of a plausible postulated mechanism, largely due to the common use of cross-sectional designs.

What were the methodological strengths and weaknesses of the reviewed research and how do they affect future research and clinical practice?

This review highlighted the practical challenges in conducting sleep research in the ABI population. Precise and valid objective sleep measurements are labour-intensive, time-consuming and potentially inconvenient and uncomfortable to the research subjects. Hence, studies using these methods typically have small samples sizes and are unable to achieve long follow-up durations for practical and logistic reasons. On the other hand, larger population-based cohort studies or database extraction and analyses can only practically use subjective sleep measures that are more feasible for the research procedure even though there are known discrepancies with objective sleep measures. This dilemma thus directly compromises study power and result validity.

In addition, there is reason to conclude after this review that the knowledge gained from basic science research regarding sleep pathophysiology after ABI has not been similarly found in humans, likely due to the lack of replicable methodology.

For this reason, it can be logically concluded that future research and interventional trials can only proceed after this dilemma can be satisfactorily resolved and there is more detailed understanding of post-ABI sleep pathophysiology in humans.

The strengths of this review were its comprehensiveness, breadth and structure in search strategy as well as having study team members coming to a consensus on disagreements over the inclusion/exclusion of specific papers. On the other hand, some limitations include the exclusion of interventional studies, animal studies, and studies in non-adult samples. This is because the study team felt that interventional trials would not meet the review's objectives, a review of animal studies would have adequate content for a separate review on its own merit, and findings from studies in paediatric populations would not be comparable with studies done in adults due to possible confounding effects of injury to a still-developing brain.

CONCLUSION

The more basic science research is needed to study the impact of sleep dysfunction on an injured brain before appropriate interventional trials can be designed. Moreover, more longitudinal studies with multiple repeated sleep measurements are required

because the ABI-sleep relationship is likely to be dynamic over the recovery trajectory. Ideally, interventional trials should conduct pre-intervention measurements of objective sleep parameters and specific ABI outcomes before designing appropriate sleep interventions that target the specific abnormal sleep parameter and its hypothesized patho-physiological mechanism, so that post-intervention measurements can be more precise and results can have improved internal validity. The increasing use of wearable technology that can capture multiple physiological parameters simultaneously would be a fascinating area of innovation with the potential to address confounding environmental factors.

LIMITATIONS

Nevertheless, sleep research is often challenging and problematic as it is limited by (i) the absence of a single, unifying method of measuring all the different facets of sleep that can reconcile both the subjective and objective components including the regulatory drives and sleep architecture, (ii) the practical challenges in accounting for environmental and psychological confounders, (iii) the lack of understanding of the potential physiological pathways between sleep disturbance and outcomes of interest and (iv) the lack of consensus on what is the best outcome measure in sleep research.

These limitations are exemplified in research studying the relationship between structural brain damage and sleep. Studies of sleep disturbances in humans after brain injury belie the hope of identifying important neuroanatomical correlates and mechanisms of sleep pathophysiology. However, the varying aetiological factors of the different types of ABI plus the different possible medical, neurological and psychological complications along disease-specific recovery trajectories, added with the potential iatrogenic effects of medications and surgery done for treatment, makes the elucidation of such a relationship challenging, if not impossible. Existing interventional studies suffer from the lack of a sound theoretical basis, which also leads to a challenge in designing new treatments for the ABI population.

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CONFLICT OF INTEREST

There is no conflict of interests between the authors.

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