

Original Research Article

ALTERATIONS IN POLYSOMNOGRAPHIC PROFILE IN ISCHAEMIC STROKE PATIENTS

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ABSTRACT

Background: Ischaemic stroke may disrupt normal sleep and cause changes in sleep architecture, respiratory events, arousals, oxygenation, and limb movements. Polysomnography (PSG) provides objective assessment of these changes. This study aimed to evaluate alterations in PSG parameters among patients with ischaemic stroke. **Materials and Methods:** This prospective study included 30 patients admitted to the Neurology ward, TVMCH, Tirunelveli, from February 2019 to April 2019, after ethical approval and written informed consent. Overnight PSG was performed from 10 pm to 6 am using EEG, EOG, EMG, ECG, nasal airflow, respiratory effort, pulse oximetry, snoring, and limb movement monitoring. Sleep and cardiorespiratory parameters were assessed during recording. **Result:** Mean age was 54.3 ± 9.1 years. Sleep efficiency was $<85\%$ in 25 (83.3%) and $\geq 85\%$ in 5 (16.7%) patients; mean efficiency was $63.8 \pm 18.1\%$. AHI <5 occurred in 19 (63.3%) and ≥ 5 in 11 (36.7%); mean AHI was 8.1 ± 10.9 . PLM index ≥ 5 occurred in 16 (53.3%) and <5 in 14 (46.7%), with mean 25.78 ± 48.12 . Mean sleep-onset latency was 43.4 ± 61.0 min, stage 2 sleep $35.6 \pm 12.9\%$, slow-wave sleep $5.1 \pm 3.8\%$, REM latency 184.7 ± 111.2 min, and arousal index 22.4 ± 25.7 . Oxygen saturation was $94.5 \pm 2.8\%$, desaturation index 14.86 ± 19.61 events/h, desaturation duration 31.7 ± 36.1 s, maximum desaturation $6.6 \pm 3.7\%$, and nocturnal heart rate 68.7 ± 8.7 beats/min. Male patients comprised 24 (80.0%) and females 6 (20.0%). **Conclusion:** PSG showed that sleep disturbance after ischaemic stroke extends beyond sleep-disordered breathing, involving sleep continuity, architecture, arousals, limb movements, oxygenation, and heart rate.

INTRODUCTION

Ischaemic stroke is one of the most important causes of neurological morbidity and mortality and can result in long term disability, cognitive impairment and diminished quality of life. The consequences of stroke are not only those of the actual brain injury but there is a number of physiological changes that can continue to influence recovery and long-term outcomes. These changes could therefore be significant for stroke management and recovery, and the understanding of these changes may be important in this context.^[1,2] Sleep plays an important role for normal brain functioning and recovery. It is linked to the processes of neuroplasticity, memory consolidation, the regulation of autonomic functions, cardiovascular functions, and metabolic processes. They can be impacted during sleep disturbances by alterations in autonomic function, inflammation, vascular regulation, and oxidative stress. Poor cerebrovascular and brain health are also linked to sleep-disordered breathing, insomnia, and circadian rhythm disturbances.^[3,4] This is especially true

following a stroke, where damage to the brain may impact the pathways involved with typical rest.

The link between stroke and sleep is complex and appears to be bidirectional. Cerebral ischaemia can disrupt sleep by impairing sleep-regulatory networks and by the associated autonomic dysfunction, motor impairments, dysphagia, airway abnormalities, mood changes, and decreased levels of physical activity. All these can contribute to poor sleep patterns, inability to sleep through the night and sleep architecture and circadian rhythm changes. Disturbed sleep, however, can also affect cerebrovascular and neurophysiological function via intermittent hypoxaemia, sympathetic activation, blood-pressure regulation, endothelial dysfunction and impaired cardiovascular control. Sleep may thus be a sequela of stroke and a risk factor for recovery and rehabilitation that may therefore be influenced by sleep disturbance.^[5-8] Sleep disturbances following stroke can occur in the areas of sleep continuity, sleep efficiency, sleep latency, sleep architecture, arousals, respiratory events, oxygenation, and movement-related abnormalities. These features can be

measured objectively by polysomnography (PSG) and thus can be valuable to assess sleep alterations post-stroke.^[2,4,9]

PSG can measure sleep architecture and physiological changes during sleep simultaneously, while subjective symptom or screening questionnaires do not. This is very significant because the level of sleep disturbance may not always be apparent from clinical symptoms alone. PSG is also recommended when a detailed assessment of sleep-disordered breathing is required in patients with stroke.^[2,4,9] Previous studies have shown that sleep-disordered breathing (SDB), especially OHS is prevalent in stroke patients. The sleep disturbances following stroke, however, do not only present with breathing disturbances. Changes in sleep efficiency, sleep fragmentation, arousals, rapid eye movement and non-rapid eye movement sleep, and sleep oxygenation also have been reported and may be of relevance for functional recovery.^[2,4,10,11]

Despite these findings, different studies report different PSG profiles in stroke patients. Sleep architecture, sleep continuity, respiratory disturbances, oxygenation, and movement-related abnormalities have been found to be different. This variation could be due to differences in stroke characteristics, lesion location, severity of the clinical signs, time of PSG assessment, demographic factors, comorbidities, functional status, and PSG methodology and scoring.^[2,10,12] Much of the available research has focused primarily towards sleep disordered breathing and obstructive sleep apnoea. These are important, but not all of the other sleep alterations post ischaemic stroke may be assessed by this focus. A broader assessment of other PSG parameters such as sleep, respiratory and oxygenation parameters, arousals and movement-related changes may help to understand the sleep profile in these patients better.^[2,4] A thorough objective evaluation of PSG is, therefore, important to gain better insight into the spectrum of sleep disorders with ischaemic stroke.

Aim: This study aimed to evaluate alterations in PSG parameters among patients with ischaemic stroke.

MATERIALS AND METHODS

This prospective study was conducted in 30 patients admitted to the Neurology ward of TVMCH, Tirunelveli from February 2019 to April 2019. Ethical approval was obtained from the Institutional Ethical Committee and written informed consent was obtained from participants before study initiation.

Inclusion and Exclusion criteria: This study included patients aged above 18 years with clinically and radiologically confirmed ischaemic stroke who were able to provide informed consent. Patients with severe stroke (NIHSS >24), ventilator dependence, pre-existing sleep disorders, haemorrhagic stroke, cortical venous thrombosis, and other major neurological illnesses were excluded from the study.

Materials: The materials used in the study included a polysomnography (PSG) system with electroencephalography, electrooculography, electromyography, electrocardiography, nasal airflow monitoring, thoracic and abdominal respiratory effort monitoring, pulse oximetry, snoring monitoring, and limb movement monitoring.

Methods: Thirty patients undergone overnight polysomnography during their hospital stay. PSG recordings were performed from 10 pm to 6 am and included electroencephalography, electrooculography, electromyography, electrocardiography, nasal airflow, thoracic and abdominal effort, pulse oximetry, snoring, and limb movements. Sleep parameters including sleep efficiency, sleep onset latency, sleep stages, rapid eye movement latency, respiratory abnormalities, periodic limb movements, arousal index, oxygen saturation, desaturation index, duration of desaturation episodes, and nocturnal heart rate were assessed. Data were presented as mean, standard deviation, frequency, and percentage.

RESULTS

Male patients were more common than female patients, with 24 (80.0%) males and 6 (20.0%) females. Sleep efficiency was <85% in 25 (83.3%) patients, while 5 (16.7%) had sleep efficiency ≥85%. An AHI <5 was seen in 19 (63.3%) patients, compared with 11 (36.7%) patients with an AHI ≥5. For periodic limb movements, 16 (53.3%) patients had an index ≥5 and 14 (46.7%) had an index <5 [Table 1].

Table 1: Distribution of patients according to sex, sleep efficiency, apnoea-hypopnoea index, and periodic limb movement index

		N (%)
Sex	Male	24 (80.0%)
	Female	6 (20.0%)
Sleep efficiency	<85%	25 (83.3%)
	≥85%	5 (16.7%)
Apnoea-hypopnoea index (AHI)	≥5	11 (36.7%)
	<5	19 (63.3%)
Periodic limb movement index	≥5	16 (53.3%)
	<5	14 (46.7%)

The mean age of the patients was 54.3 ± 9.1 years. Mean sleep efficiency was $63.8 \pm 18.1\%$, with stage 2 sleep accounting for $35.6 \pm 12.9\%$ of total sleep time. Slow-wave sleep was $5.1 \pm 3.8\%$. Sleep onset latency averaged 43.4 ± 61.0 minutes, while REM

latency was 184.7 ± 111.2 minutes. The mean periodic limb movement index was 25.78 ± 48.12 , and the mean arousal index was 22.4 ± 25.7 . The mean AHI was 8.1 ± 10.9 [Table 2].

Table 2: Mean polysomnographic and clinical parameters among patients

	Mean $\pm$ SD
Age (in years)	54.3 ± 9.1
Sleep efficiency (%)	63.8 ± 18.1
Sleep onset latency (min)	43.4 ± 61.0
Total sleep time occupied by stage 2 sleep (%)	35.6 ± 12.9
Slow-wave sleep (%)	5.1 ± 3.8
REM latency (min)	184.7 ± 111.2
Apnoea-hypopnoea index	8.1 ± 10.9
Periodic limb movement index	25.78 ± 48.12
Arousal index	22.4 ± 25.7

Mean oxygen saturation during sleep was $94.5 \pm 2.8\%$. The mean desaturation index was 14.86 ± 19.61 events/h, and the mean duration of desaturation episodes was 31.7 ± 36.1 seconds. Maximum

desaturation averaged $6.6 \pm 3.7\%$, while the mean nocturnal heart rate was 68.7 ± 8.7 beats/min [Table 3].

Table 3: Nocturnal oxygenation and heart rate parameters among patients

	Mean $\pm$ SD
Oxygen saturation during sleep (%)	94.5 ± 2.8
Desaturation index (events/h)	14.86 ± 19.61
Duration of desaturation episodes (s)	31.7 ± 36.1
Maximum desaturation (%)	6.6 ± 3.7
Nocturnal heart rate (beats/min)	68.7 ± 8.7

DISCUSSION

In the present study, the average age of the patients was 54.3 ± 9.1 years, with a distinct male predominance (24 patients, 80.0% of patients were male, 6 patients 20.0% female). Mekky et al. reported no difference in mean age between 93 patients with first-ever ischaemic stroke and controls (55.05 ± 12.27 years; $p=0.524$), of which 59 (63.4%) were male.¹³ Kim et al. reported patients in acute ischaemic stroke group had a higher mean age and more males (67.7 ± 12.6 years, 54.4%).¹⁴ Study setting and patient characteristics may have contributed to this younger age and higher male:female ratio of our study and should be taken into consideration when comparing PSG findings.

In our study, the mean sleep efficiency was $63.8 \pm 18.1\%$, with 83.3% of patients being sleep efficiencies below 85%. Similarly, Mekky et al. reported a significantly poorer sleep efficiency in 93 patients with first-ever ischaemic stroke compared with 50 age-matched controls ($p=0.001$).¹³ Frequent arousals, respiratory events, limb movements, acute illness and hospital environment may be associated with poor sleep efficiency. Variation may also be due to differences in time of PSG.

In our study the mean AHI was 8.1 ± 10.9 /h with some patients with AHI ≥ 5 /h. Patients with stroke had significantly higher AHI than controls ($p<0.001$) with the highest AHI observed in patients with cortical infarcts. In 119 patients with acute ischaemic stroke, Fu et al. discovered that moderate to severe sleep apnoea was correlated with wake-up stroke (OR

2.879, 95% CI 1.17-7.089; $p=0.021$). Respiratory arousal-threshold phenotype was present in 34.8% of the patients with wake-up stroke and 68.1% of the patients without wake-up stroke ($p=0.008$) among patients with OSA and an AHI ≥ 5.15 . Schutz, et al. identified three OSA phenotypes in patients with ischaemic stroke and REI ≥ 10 /h: 12% had higher NIHSS, 56% were younger with relatively low NIHSS and 32% had severe OSA with larger comorbidity.¹⁶ The results show that respiratory disturbance is present in a significant proportion of stroke patients, but is variable depending on the type of stroke and the subjects studied.

The mean number of periodic limb movements per hour in our study: 25.78 ± 48.12 events/h. Manconi et al. performed a study of PLMS in stroke/TIA patients, which included 169 acute recordings, 191 at 3 months, 210 paired recordings from 105 patients, and 162 matched control recordings. They found the mean PLMS frequencies to be comparable between patients and controls, and similar proportions to have PLMS frequencies >10 and >15 events/h, with PLMS frequency stable from the acute to chronic phase. PLMS was positively correlated with age, BMI and hypertension.¹⁷ Mekky et al., reported a significantly higher LM index in stroke patients than control patients ($p=0.05$), with a significant difference between the patients with sizable and lacunar infarctions ($p=0.038$).¹³ Dharmakulaseelan et al. reported PLM index was also higher in patients with ESUS compared to those with other stroke mechanisms ($p=0.037$).¹² Thus, the results differ between studies. The results from Mekky et al. show

a possible association with infarct burden, and those from Manconi et al. show a possible persistence of PLMS that is not related to the acute phase of stroke.^[13,17]

In the present study, stage 2 sleep was found to compose $35.6 \pm 12.9\%$ of total sleep time, and slow-wave sleep was found to compose $5.1 \pm 3.8\%$ of total sleep time. Mekky et al. found increased slow-wave sleep in stroke patients and increased REM sleep and REM latency in controls. They did not detect any differences in sleep based on stroke severity.^[13] We found that infarct location influenced sleep, however, with brainstem strokes having the greatest REM percentage, and REM latency. This shows that sleep organization can vary depending on the location of brain damage.

Sleep-onset latency in our study was 43.4 ± 61.0 min. For sleep-onset latency, Dharmakulaseelan et al. found that the duration was significantly longer for ESUS than other stroke mechanisms, $p=0.0166$.^[12] Mekky et al. also found that stroke patients had higher sleep latency than controls, with no significant difference between the three types of strokes (cortical, subcortical, or brainstem) ($p=0.084$).^[13] These suggest that sleep initiation deficits are likely to be present following stroke. Illness, hospital environment, stress, and medication administration can also have an impact on sleep onset.

In our study, REM latency was 184.7 ± 111.2 minutes. Mekky et al. showed that the REM latency was significantly longer in brainstem stroke ($p=0.001$) and REM percentage was significantly higher in brainstem stroke ($p=0.043$) when compared to the other strokes. They also found that the controls had a longer REM latency than the stroke patients. The results of this study show that REM sleep could be disturbed following ischaemic stroke, especially when brainstem is involved.^[13] The spread is large, and may be leads to the differences in the location of the lesions and level of sleep fragmentation, as well as neurological involvement, and drug exposure.

The mean arousal index (events/h) was 22.4 ± 25.7 in our study. The same results were reported by Šiarnik et al., who found the arousal index was higher in the stroke patients compared to the controls ($p=0.0001$), and higher in large-artery stroke than in small-vessel stroke ($p=0.013$), cardioembolic stroke ($p=0.007$), and other or unknown causes ($p=0.027$) of stroke.¹⁸ The close similarity between the values reported by Mekky et al. and those in our study shows that frequent nocturnal arousals is an important aspect of post-stroke sleep disturbance.^[13] These arousals can be associated with respiratory events, movement of the limbs, environmental disturbance or changes of cortical regulation.

The mean sleep oxygen saturation, desaturation index and mean desaturation duration in our study were $94.5 \pm 2.8\%$, 14.86 ± 19.61 events/h and 31.7 ± 36.1 seconds, respectively, and the maximum desaturation was $6.6 \pm 3.7\%$. Kim et al. reported that stroke patients who desaturated at night were more likely to have nocturnal desaturation (29.1% vs. 12.3%, OR

3.25; 95% CI 1.63–6.46, $p=0.001$) compared with those who did not have a stroke.^[14] Šiarnik et al. showed that the desaturation index differed between stroke aetiological types and was independently associated with atrial fibrillation (OR 1.037; 95% CI 1.004–1.071; $p=0.026$).^[18] In a study by Chen et al., the difference in mean desaturation between those with and without atrial fibrillation was not significant ($p=0.079$) but adjusted mean desaturation was associated with atrial fibrillation (OR 1.19, 95% CI 1.05–1.35; $p=0.008$).^[19]

Mekky et al. also reported an increase in the desaturation indices and a decrease in lowest oxygen saturation in stroke patients when compared to controls. The desaturation index was highest in sizable infarctions and the lowest in the lowest oxygen saturation; the lower the oxygen saturation, the higher the NIHSS score at 1 month ($p=0.006$); ($p=0.047$).^[13] In 204 patients, Huhtakangas et al. observed the following differences between non survivors and survivors: lower minimum oxygen saturation, higher T90, and higher central-apnoea index; minimum saturation and T90 were related to mortality ($p=0.007$ and $p=0.005$).^[20] These results showed that nocturnal oxygenation is an important component of sleep disturbance after ischaemic stroke, and that there are links with wake-up stroke, atrial fibrillation, infarct burden, neurological severity, and mortality.

CONCLUSION

Sleep disturbance following stroke is not restricted to sleep-disordered breathing as the patients had other polysomnographic parameters that were changed. These included decreased sleep efficiency, extended sleep-onset latency, extended REM latency, frequent arousals, periodic limb movements, sleep architecture abnormalities, and oxygenation abnormalities at night. The mean AHI suggested mild sleep-disordered breathing, but the occurrence of limb movements as well as frequent arousals suggested that other aspects of sleep were also affected. PSG was able to provide objective information to help identify these changes, including changes which are not discernible by symptoms alone.

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