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Analysis of Various Sleep Architecture Parameters Using Polysomnography among Patients with Recurrent Stroke: A Cross-Sectional Study at A Tertiary Care Hospital in Rajasthan

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ABSTRACT

Background: Sleep-disturbance is an increasingly recognized disorder that is particularly prevalent among stroke patients. Despite the known association between sleep disturbances and stroke, the prevalence and pattern of sleep disturbances among patients with recurrent strokes is not well defined.
Objectives: The present study was conducted to evaluate sleep architecture in patients with recurrent stroke and to study correlations of various sleep parameters with number of stroke among the patients of recurrent strokes.
Subjects & Method: This hospital based observational cross-sectional study was conducted under the Department of Neurology in a tertiary care hospital setting, on the patients with recurrent stroke (ischemic as well as hemorrhagic stroke). Sixty five patients with diagnosis of more than one stroke within 7 days of last stroke were included in the study after obtaining informed written consent and satisfying inclusion and exclusion criteria. The patients were divided into three groups on the basis of number of strokes, i.e., 2, 3 and 4 strokes. Detailed history and clinical examinations were performed. Patient underwent one night polysomnography to obtain sleep parameters; following which they were enquired about sleep quality and daytime sleepiness. Correlations between sleep parameters and number of strokes were established using IBM SPSS software version 23.0. P value of less than 0.05 was considered significant.
Results: Mean Total Sleep Time (TST), Mean Sleep Period Time (SPT) and Sleep Efficiency were found to have an inverse and statistically significant correlation with number of strokes (P<0.0001); while Sleep Latency showed direct correlation with number of strokes (P<0.0001). Only 43.08% (28/65) patients could attain REM stage. All those were from 2 stroke group and none from 3 or 4 stroke groups. Apnea Hypopnea Index and Arousal Index were found to be increasing with higher number of strokes; while Mean average saturation and Mean minimum saturation were found to be decreasing with higher number of strokes among the study population (P<0.0001). It was observed that with higher number of strokes, Epworth Sleepiness Scale (ESS) score also increases (P<0.0001).
Conclusion: Number of strokes shows statistically significant correlation with sleep architecture. Increasing number of strokes adversely affect sleep parameters.

Keywords: *Recurrent strokes, Polysomnography, Sleep architecture, TST, SPT, Sleep Efficiency, Sleep latency, Apnes Hypopnea Index, Arousal Index, Epworth Sleepiness Scale.*

INTRODUCTION

Sleep architecture is defined as the structure and pattern of the different sleep stages experienced by the subject. Lesions affecting the central nervous system, whether focal or diffuse, may disturb sleep structure and patterns**.** Sleep architecture is a significant aspect to understand the basic structural organization of normal sleep.[1] Over a period of time there could be disturbances in the normal sleep pattern which could be mainly due to stress, stroke and these are considerable disabilities in a person's life which he/she tries to adjust/manage throughout their life.[2] Post-stroke effects leads to sleep-

apnea which is usually noticed as obstructive sleep apnea (OSA) that partially or completely blocks the breath in the upper airway during sleep.[3]

Stroke is the most common source of disability-adjusted life-years (7.3million, 35%). Globally, stroke is one of the leading causes of morbidity and mortality in adults**.** Changes in sleep architecture following ischemic stroke have been poorly investigated. Although sleep disorders are common in most neurological disorders, they are often poorly recognized as adjunctive risk factors.[4] The American Heart Association and American Stroke Association guidelines recommend screening for obstructive sleep apnea for the prevention of **recurrent stroke** or transient ischemic attack (TIA).[5]

Therefore, it is essential to evaluate the association across the sleep architectureparameters and stroke which scope the early diagnosis and appropriate treatment underlying the breathing sleep disorder to both improve the chances of recovery from stroke in the short term and to reduce the risk of recurrent strokes in the long term.

Neurobiology of Sleep Disturbance and Recurrent Stroke:[6]

- Sleep disturbance can increase the risk of recurrent stroke by:
- Promoting the accumulation of toxic proteins in the brain
- Increasing inflammation and oxidative stress
- Elevating levels of the wake-stabilizing hormone orexin
- Recurrent stroke can lead to sleep disturbances by:
- Damaging brain regions involved in sleep regulation
- Causing pain and discomfort
- Disrupting circadian rhythms

Although a number of studies have studied the association between sleep disturbances and "stroke", **none have looked into such an association with "recurrent stroke**". Hence, the present "**novel"** study was carried out to evaluate sleep architecture using polysomnography in patients with recurrent stroke including the various sleep parameters, quality of sleep and day time sleepiness. The study provides valuable insights into the sleep architecture of patients with **recurrent strokes**, shedding light on various sleep indices, sleep duration, and characteristics of sleep.

OBJECTIVES:

1. To study various sleep parameters using Polysomnography in patients with recurrent stroke.
2. To study co-relation of number of strokes with various sleep parameters.

SUBJECTS & METHODS

This hospital based cross sectional study was conducted under the Department of Neurology, Govt Medical College, Kota, which is a tertiary care hospital setting that provides neurosurgical, neurodiagnostic and intensive care services in addition to neurologic evaluation during the period from July 2022 to June 2023 among the patients with recurrent stroke (ischemic as well as hemorrhagic stroke).

Inclusion Criteria: Patients with recurrent stroke (>1 stroke either ischemic or hemorrhagic).
Exclusion Criteria: Patients on sedatives, patients with addiction, patients known case of OSA, patients with chronic cardio pulmonary disease, pregnant females, patients currently on mechanical ventilation or tracheostomy, patients requiring current treatment with pressors, patients currently using CPAP, or use within one month prior to stroke, patients currently having contact of respiratory tract infection and patients in the state of coma/stupor were excluded from the study.

Methodology

All the patients with diagnosis of more than one stroke within 7 days of last stroke were included in the study Informed consent was taken from patients fulfilling inclusion and exclusion criteria. Detailed history was taken and clinical examination was conducted. Patient underwent one night polysomnography (PSG) following which he/she was enquired about sleep quality and daytime sleepiness by P.S.Q.I. and E.S.S. scale respectively. Sleep parameters were gained from P3SG and the data were analysed.

Statistical analysis

Data were arranged in the form of descriptive tables and self explanatory charts/graphs. Descriptive and frequency analysis was done by using Microsoft Excel version 7.0. Frequencies were expressed in the form of percentage. Distribution of continuous data was represented in the form of mean and standard deviation. Appropriate statistical tests of significance like Chi-square test and ANOVA test were applied to establish the correlations between various sleep parameters and number of strokes. Statistical analysis was performed by using IBM SPSS software version 23.0. P value of less than 0.05 was considered significant.

RESULTS

Total 65 patients were enrolled in the present study based on inclusion and exclusion criteria. Baseline epidemiological characteristics have been summarized in Table 1 and 2 below. Mean Age in the study group was 70.92±11.84 years. Majority were Males (75.38%) while Females were 24.62%. While a majority (53.85%) had 2 episodes of stroke, 33.85% had 3 episodes, and 12.30% of patients had 4 episodes (recurrent strokes). No significant difference in number of strokes on the basis of site of stroke (supra or infra tentorium) (Table 3)

Mean Total Sleep Time (TST) among the study subjects was 348.43±39.77minutes. Mean Total Sleep Time was found to have a statistically significant correlation with number of strokes (P<0.0001). It was found to be the maximum (377.34 min) among those patients who had a history of two strokes while it was found to be the least (273 min) among those patients who had a history of 4 strokes. The correlation was established to be statistically significant (P<0.0001).

Mean Sleep Period Time (SPT) was 182.94±48.52minutes. Mean Sleep Period Time was also observed to be having a statistically significant correlation with number of strokes (P<0.0001). It was found to be the maximum (218.94 min) among those patients who had a history of two strokes while it was found to be the least (112.25 min) among those patients who had a history of 4 strokes. The correlation was established to be statistically significant (P<0.0001).

Mean Sleep Efficiency was 51.01±8.097 %. Sleep Efficiency (the percentage of time spent asleep while in bed) was calculated among the study patients. Sleep efficiency was categorized into four categories, i.e., <40%; 41-50%; 51-60% and 61-70% to analyze further. It was observed to have a statistically significant correlation with number of strokes (P<0.0001). We observed that in the subgroup of patients with a history of two strokes, the majority (80%, 28 out of 35) exhibited sleep efficiency within the range of 51-70%. Conversely, in the group with three strokes, all patients (100%, 22 out of 22) demonstrated sleep efficiency falling within the 41-50% range. Among 4 strokes group 62.5% (5/8) patients had sleep efficiency between 41 to 50% min while 37.5% (3/8) has sleep efficiency of even less than 40%. Thus, with increasing number of strokes, sleep efficiency was observed to be decreasing. The correlation was established to be statistically significant (P<0.0001).

Mean Sleep Latency observed was 18.54±5.74 minutes. Sleep Latency (time to go from fully awake to sleeping) was calculated among the three groups of patients, i.e., patients having 2, 3 and 4 strokes. Sleep latency was then distributed into four categories, i.e., <10; 11-20; 21-30 and >31 min to analyze further. It was observed to have a statistically significant correlation with number of strokes (P<0.0001). We observed that the maximum patients (32/35; 91.43%) from the patient group having H/o two strokes showed to have sleep latency between the range of 11-20 min. Most patients (25/30; 83.33%) from 3 and 4 strokes group showed to have sleep latency of 21-30 min. Among 3 strokes group 100% patients (22/22) had sleep latency between 11 to 30 min; while among 4 strokes group 87.5% (7/8) patients had sleep latency between 21 to 30 min and 12.5% (1/8) patients had sleep latency of >31 min also, which was still higher. Thus, with increasing number of strokes, sleep latency was also found to be increasing. The correlation was established to be statistically significant (P<0.0001).

Means of N1%, N2%, N3% and REM% were calculated for the study population. Statistical analysis shows that all the stages had significant decrease among those having more number of strokes (P<0.01). Mean of REM% stage was found to be absent among the patients having history of three or four strokes.

Mean REM Latency was 104.89±10.36 minutes. Mean Arousal Index was 17.65±4.48 minutes. Apnea Hypopnea Index of 16-30 was seen in 37 (56.92%) followed by 6-15 in 22(33.85%) and >31 in 6(9.23%). REM Latency (time elapsed between the onset of sleep to the first REM stage) was calculated among the study patients. 43.08% (28/65) patients could attain REM stage. All 100% were from 2 stroke group and none from 3 or 4 stroke groups. A statistically significant correlation was established between REM latency and number of strokes (P<0.0001). The correlation was established to be statistically significant (P<0.0001).

Sleep parameters like **Apnea Hypopnea Index; Arousal Index;** Average saturation and Minimum saturation were observed to be statistically significantly associated with number of strokes (P<0.0001). Apnea Hypopnea Index and Arousal Index were found to be increasing with higher number of strokes; while Mean average saturation and Mean minimum saturation were found to be decreasing with higher number of strokes among the study population.

Average Saturation of 96-100% was observed in 30(46.15%) patients, 81-90% in 19 (29.23%), 91-95% in 14 (21.54%) and <80% in only 2 (3.08%) patients. While a minimum saturation of 51-60% was seen in 35 (53.84%), 61-70% was seen in 28 (43.08%) and <50% only in 2 (3.08%).

Epworth Sleepiness Scale (ESS) 0-7 was not seen in any of the patients. Out of the total patients, 49.23% had a scale of 8-9, followed by 35.38% with a scale 10-15 and 15.38%,16-24. With higher number of strokes, Epworth Sleepiness Scale score also increases. 100% patients from 4 strokes group had 16-24 ESS score. 100% of patients from 3 strokes group had 10-15 ESS score; while 91.43% (32/35) patients had 8-9 ESS score. The correlation was established to be statistically significant (P<0.0001).

Table 1: Baseline epidemiological characteristics

Gender	Number of patients	Percentage
Male	49	75.38%
Female	16	24.62%
Total	65	100%
Number of strokes		
2	35	53.85%
3	22	33.85%
4	8	12.30%
Total	65	100%
Modified Rankins Scale		
0	**0**	**0%**
1	**0**	**0%**
2	44	67.69%
3	19	29.23%
4	2	3.08%
5	0	0%
Total	65	100%

Table 2: Site of Stroke wise distribution: Supratentorial versus Infratentorial

Site of stroke	First Stroke (N=65)		Second Stroke (N=65)		Third stroke (N=30)		Fourth stroke (N=8)	
	No. of Pts.	%	No. of Pts.	%	No. of Pts.	%	No. of Pts.	%
Supra tentorial	59	**90.77%**	49	**75.38%**	24	**80%**	5	**62.5%**
Infra tentorial	6	9.23%	16	24.62%	6	20%	3	37.5%
Total	65	100%	65	100%	30	100%	8	100%
			Chi sq = 1.064 with 2 degrees of freedom; P = 0.588 (NS)					
	Chi sq = 7.291 with 3 degrees of freedom; P = 0.082 (NS)							

Table 3: Co-relation of Number of strokes with Sleep parameters:

Parameters		Number of Strokes			P value	
		2	**3**	**4**		
Mean Total Sleep Time TST (min)		377.34	329.86	273	<0.0001(S)**	
Mean Sleep Period Time SPT (min)		218.94	151.36	112.25	<0.0001(S)**	
Sleep Latency	<10 min (2)	2	0	0	Chi sq= 53.180 with df= 6	<0.0001 (S)*
	11-20 min (36)	32	4	0		
	21-30 min (26)	1	18	7		
	>31 min (1)	0	0	1		
	Total	35	22	8		
Sleep Efficiency	<40% (3)	0	0	3	Chi sq= 62.085 with df=6	<0.0001 (S)*
	41-50% (34)	7	22	5		
	51-60% (15)	15	0	0		
	61-70% (13)	13	0	0		
	Total	35	22	8		
Mean of N1% stage		4.54	5.18	3.63	0.0015(S)**	
Mean of N2% stage		39.43	35	35.5	<0.0001(S)**	
Mean of N3% stage		7.74	5.64	1.5	<0.0001(S)**	
Mean of REM% stage		2.65	0	0	-	
REM Latency (N=28)	<100 min (2)	2	0	0	42.162 with df=6	<0.0001 (S)*
	101-110 (22)	22	0	0		
	110-120 (4)	4	0	0		

***Chi square; **ANOVA; NS=Not Significant; S=Significant**

Table 4: Co-relation of Number of strokes with Sleep parameters:

Parameters		Number of Strokes			Chi square test	P value
		2	3	4		
Mean AHI		14.86	24.77	31.38	<0.0001(S)**	
Apnea Hypopnea Index	0-5	0	0	0	72.617 with df= 4	<0.0001 (S)*
	6-15	22	0	0		
	16-30	13	22	2		
	>31	0	0	6		
Arousal Index	<10(5)	5	0	0	37.019	<0.0001 (S)*
	11-20(39)	29	10	0		
	21-30(21)	1	12	8		
Mean Average Saturation		97.63	89.95	82.63		<0.0001(S)**
Mean Minimum Saturation		64.91	57.09	52.13		<0.0001(S)**

***Chi square; **ANOVA; NS=Not Significant; S=Significant**

Table 5: Co-relation of Number of strokes with ESS:

Parameters		**Number of Stroke**			**Chi square test**	**P value**
		2	**3**	**4**		
Epworth Sleepiness Scale	**0-7**	**0**	**0**	**0**	**109.43 with df= 4**	**<0.0001(S)**
	8-9	**32**	**0**	**0**		
	10-15	**1**	**22**	**0**		
	16-24	**2**	**0**	**8**		

DISCUSSION

Several hypotheses have been advanced to understand how sleep disruption might cause or increase the incidence of stroke. Among them, sympathetic activation, procoagulatory and inflammatory pathway activation, acceleration of atherosclerosis, and consequent changes in brain hemodynamics and oxygenation are the ones mostly debated in literature.[7-9]

A recent meta-analysis confirmed that prevalence of insomnia is considerably higher in stroke survivors compared to the general population (ranging from 32.2 to 40.7% of cases depending on the tools of investigation). Greater insomnia symptoms were indicated in those with comorbid depression and anxiety.[10]

This present study was a cross sectional observational study conducted in the Department of Neurology, Government Medical College and attached group of hospitals, Kota involving **patients with recurrent stroke i.e., >1 stroke**, either ischemic or haemorrhagic. Different statistical tests such as, descriptive analysis, frequency analysis, cross tabulation, chi-square test and correlation analysis using the SPSS version 25 were used to study the changes in sleep architecture among the patients with recurrent strokes. A p value of less than 0.05 was considered significant.

This study confirms a severe sleep disruption inthe acute phase of stroke, as well as a significant improvement ofsleep quality among the patients having history of recurrent strokes as per inclusion criteria. In particular, sleepefficiency and REM sleep have been observed to be particularly reduced (Table 3). The amelioration of sleep quality found to be more significant in the subgroup of patients with history of four strokes when compared with those having history of 2 or 3 strokes. In particular, a lower REM sleep percentage was accompanied by a higher number of strokes, and a lower sleep efficiency (Table 3). Interestingly, this subgroup of patients (having history of four strokes) also had a statistically higher AHI, consistent with the hypothesis that sleep isaltered globally in this group (Table 4).

We hypothesize that the wellknown increased incidence of insomnia and OSA in may play a significant role in recurrence of stroke. **Miano S, et al.**[11] **(2022)**, a study published in 2022, also reported similar results in their study entitled “SAS CARE 1: Sleep architecture changes in a cohort of patients withIschemic Stroke/TIA”, however this study was not solely focused on **recurrent stroke**.The observed alterations of sleep architecture are in agreement with the high prevalence of acute insomnia in stroke patients.[12] The insomnia may be a directconsequence of either the infarct, the result of environmental factors(light, noise on stroke units), or comorbidities (SDB, depression,pain).[10,12-14]The fact supports the results of present study that insomnia has shown statistically significant association with increasing number of strokes among the patients under study. **Kim J, et al.**[10] **(2015)** reported that reduced sleep duration has been shown to bestrongly associated with poor functional outcomes and neurocognitivedysfunction among stroke survivors,[10]which is consistent with the results of present study also (Table 3), sleep duration was observed to lesser among those having more number of strokes.

A recent meta analysis by **Baylan S, et al.**[15] **(2020)**confirmed that prevalence of insomnia is considerablyhigher in stroke survivors compared to the general population(ranging from 32.2 to 40.7% of cases depending on the tools

ofinvestigation) and greater insomnia symptoms were indicated in thosewith comorbid depression and anxiety,[15] the fact supports the results obtained in the present study that greater degree of insomnia were observed statistically significant with the recurrence of stroke and found to be more profound among those having a history of more number of strokes as evident in Table 3 and 4.

We observed statistically significantly reducing mean N3% and absence of REM sleep among those having history of 3 or 4 strokes (30 out of 30 patients) as shown in table 3. Similarly, **Hofmeijer J, et al.**[13] **(2019)** also found in their pilot study an absence of REM sleep during night-time in 10 out of 13 patients and lack of N3 sleep in those with secondary deterioration. **Miano S, et al.**[11] **(2022)** also concluded similarly reporting a negative correlation between the Stroke Scale measured (NIHSS) and N3%, especiallyin patients with the highest sleep disruption. **Hermann and Bassetti**[16] **(2016)** stated that insomnia may be directly related to brain damage,[16] which can be a potential explanantion of recurrent strokes in such patients.

In present study, we observed no significant difference in number of strokes on the basis of site of stroke (supra or infra tentorium) (Table 2). However, according to **Ferre A, et al.**[17] **(2013)**, patients with strokes within the paramedian thalamus can also develop insomnia due to an inability to generate sleep spindles due to the disruption of the thalamoreticular system.[17]But on the other hand, most other studies confirmed that no significantdifferences in sleep architecture between patients with brainstem (infratentorial) lesions versus those with hemispheric (supratentorial) lesions were found.[11,18-20]

In present study, we observed and analyzed the patterns of Stages of sleep (N1, N2, N3, REM means along with REM latency), sleep duration parameters (TST, SPT), sleep latency and efficiency, parameters like AHI, Arousal index, mean saturation and Epworth Sleepiness Scale) to assess the degree of impact of snoring, gasping or choking for air during sleep, insomnia, daytime hypersomnia and other contributory factors of OSA (Obstructive Sleep Apnea) /SDB (Sleep Disordered Breathing), among the study population having history of recurrent strokes (two, three and four times). We found strong statistical correlations between increasing number of strokes (**recurrences**) and decreasing overall sleep duration (TST, SPT sleep efficiency) as well as NREM sleep and mere absence of REM sleep. We also observed strong statistical correlations between increasing number of strokes (**recurrences**) and increasing values of sleep parameters like AHI, Arousal Index, Sleep latency and Epworth Sleepiness Scale.

Koo DL, et al.[21] **(2018)** supports our results with the explanation that any cause of **sleep disruption, including OSA, insomnia,** shift work, **sleep-wake disorder**, periodiclimb movement disorder, or restless legs syndrome(RLS), have been linked to increased risk of cardiovascular and cerebrovascularevents.[21]Impaired cardiovascular restoration, through a **reduction in cardioprotective stable non–rapid eye movement (non-REM) sleep**,and derangement of normal autonomic and sympathetic nervous systemactivation are thought to be the main mechanisms that **increase the risk ofstroke and its recurrence**.

McDermott M, et al.[22] **(2018)** study furtheradds to the justifications by stating that **Sleep duration** alsoseems to play a role in the propagation of metabolic and cardiovascular disease. Long sleep duration is associated with diabetes and coronary heart disease, whileshort sleep duration is associated with diabetes, hypertension, and coronaryheart disease.[22]

The American Heart Association recommends OSA screeningwith a thoroughhistory to assess for suggestive symptoms (i.e., snoring, gasping or choking for airduring sleep, **insomnia,** and **daytime hypersomnia**), a complete physicalexamination with specific notation of the body mass index and upper airwayanatomy, and implementation of practical questionnaires, such as the STOP-BANG questionnaire, Berlin Questionnaire, or the **Epworth Sleepiness Scale**.[23-25]

Obstructive sleep apnea and Sleep Disordered Breathingultimately leading to insomnia is a recognized, well-established independent risk factor of **Stroke**, in additionto other risk factors including hypertension, diabetes, hyperlipidemia, tobaccouse, and sedentary lifestyles.[26]OSA is said to be associated with a two-fold increased risk of incident stroke(relative risk 2.10; 95% confidence interval 1.50 to 2.93).[27]A large prospectivepopulation study, the Sleep Heart Health Study, followed approximately 5000patients for 8 years and found that OSA increased the risk of stroke(adjusted hazard ratio of 2.86; 95%confidence interval 1.1 to 7.4).[28]

We observed statistically significant correlations between increasing number of strokes (**recurrences**) and increasing values of sleep parameters like AHI, Arousal Index, Sleep latency and Epworth Sleepiness Scale. The association of higher apnea-hypopnea and arousal indices with an increased number of strokes corroborates the bidirectional relationship between sleep apnea and stroke. Sleep apnea is not only a consequence but also a potential contributor to stroke risk.The significant correlation between arousal index and the number of strokes implies that as the number of strokes increases, there are changes in the frequency of arousals from sleep.Arousal index increased with an increasing number of strokes. Patients with two strokes had the highest proportion with an arousal index of 11-20. This suggests that

the cumulative neurological burden may contribute to increased sleep fragmentation and arousal events, impacting the overall quality of sleep.

Table 4 depicts that sleep parameters like **Apnea Hypopnea Index; Arousal Index**; **Average saturation and Minimum saturation** were observed to be statistically significantly associated with number of strokes (P<0.0001). Apnea Hypopnea Index and Arousal Index were found to be increasing with higher number of strokes; while Mean average saturation and Mean minimum saturation were found to be decreasing with higher number of strokes among the study population. The significant correlation between apnea hypopnea index and the number of strokes indicates that as the number of strokes increases, there are changes in the frequency of apneas and hypopneas during sleep. Apnea hypopnea index increased with an increasing number of strokes. This suggests that sleep-disordered breathing, such as apneas and hypopneas, may become more prevalent with a higher stroke burden. The neurological consequences of recurrent strokes could contribute to respiratory disturbances during sleep.

Sleep-disordered breathing, defined as anapnea-hypopnea index score of greater than 5 events per hour, was present in upto 72% of patients with ischemic and hemorrhagic stroke and transient ischemicattack, compared to a prevalence range of 9% to 38% in the general population.[29] Similar to present study, **Johnson & Johnson**[29] **(2010)** also reported that subgroup analysis found that sleep-disordered breathing occurred morefrequently in those with **recurrent rather than initial stroke**, clearly supporting the results of the present study.

The pathophysiologic mechanisms of OSA as a risk factor for stroke areincreasingly understood through the evolving literature. The **proposedmechanisms of stroke and OSA** include endothelial dysfunction, **oxidative stress**, systemic inflammation, development of atherosclerosis linked to intrathoracic pressure swings, **recurrent arousals and intermittent hypoxia** resulting in sympathetic activation.[30-33]

In present study, we observed that the significant correlations between oxygen saturation levels and the number of strokes (Table 4), which suggests that as the number of strokes increase, there are changes in respiratory and cardiovascular parameters during sleep. Average and minimum saturation levels decreased with an increasing number of strokes. This implies that as the neurological burden accumulates, there may be an increased likelihood of respiratory challenges during sleep. The decline in oxygen saturation levels could be linked to a combination of stroke-related respiratory muscle weakness and the presence of sleep-disordered breathing.

Brown DL, et al.[34] **(2015)** concluded that untreated OSA is also linked to higher incidences of **recurrent strokes**. In theBrain Attack Surveillance in Corpus Christi (BASIC) project, 526 of 842participants (63%) with sleep apnea (apnea-hypopnea index score >10 events/hour) suffered recurrent strokes with an adjusted hazard ratio of 1.02 perone-point increase in the apnea-hypopnea index score (95% confidence interval1.01 to 1.03).[34]Another study supporting present study (**Yaggi HK, et al.**[35]2005) states that the presence of OSA also increases post stroke mortality, with a trend analysis finding a correlation between increasing OSA severity and pooreroutcomes.[35]A similar finding of increased mortality in patients with moderate OSA (apnea-hypopnea index score >15 events/hour) admitted for in-hospital stroke rehabilitation was seen in a 10-year longitudinal study conducted by **Sachin C, et al.**[36] **(2008)**.

CONCLUSION

Patients with recurrent stroke have significant sleep disturbances, with time indices and changes in architecture being worse among patients with more number of strokes. Strategies to promote sleep quality following stroke and their subsequent impact on stroke outcome should be a focus of future investigations. A better understanding of sleep after stroke is not only relevant for rehabilitation and long-term outcome, but also for mental health and quality of life.

The present “novel study on the Sleep Architecture in patients with Recurrent Stroke” adds a valuable layer to our understanding of the complex relationship between recurrent strokes and sleep architecture. It not only contributes to the existing body of literature but also has practical implications for the holistic care of stroke survivors. The correlations observed in the present study provide a comprehensive understanding of how the cumulative burden of recurrent strokes influences various aspects of sleep architecture. These findings not only contribute to the existing knowledge but also have implications for the holistic care and management of individuals with multiple strokes.

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REFERENCES

1. Colrain IM, Nicholas CL, Baker FC. Alcohol and the sleeping brain. Handbook of Clinical Neurology. 2014;125:415–31.
2. Deuschl G, Beghi E, Fazekas F, Varga T, Christoforidi KA, Sipido E, et al. The burden of neurological diseases in Europe: an analysis for the Global Burden of Disease Study 2017. The Lancet Public Health. 2020 Oct;5(10):e551–67.
3. Stevens D, Martins RT, Mukherjee S, Vakulin A. Post-Stroke Sleep-Disordered Breathing—Pathophysiology and Therapy Options. Frontiers in Surgery. 2018 Feb 26;5.
4. Rakusa M, MariuszSieminski, Rakusa S, Cristian Falup-Pecurariu, Fronczek R, Hidalgo H, et al. Awakening to sleep disorders in Europe: Survey on education, knowledge and treatment competence of European residents and neurologists. European Journal of Neurology. 2021 Jul 9;28(9):2863–70.
5. Kleindorfer DO, Towfighi A, Chaturvedi S, Cockroft KM, Gutierrez J, Lombardi-Hill D,et al. 2021 Guideline for the Prevention of Stroke in Patients With Stroke and Transient Ischemic Attack: A Guideline From the American Heart Association/American Stroke Association. Stroke. 2021 May 24;52(7):e364-467.
6. Musiek ES, Holtzman DM. Mechanisms linking circadian clocks, sleep, and neurodegeneration. Science 2016;354(6315):1004–1008. doi:10.1126/ science.aah4968.
7. Arnardottir ES, Mackiewicz M, Gislason T, Teff KL, Pack AI. Molecular Signatures of Obstructive Sleep Apnea in Adults: A Review and Perspective. Sleep. 2009 Apr;32(4):447–70.
8. Libby P. Inflammation in atherosclerosis. Nature. 2002 Dec;420(6917):868–74.
9. Shamsuzzaman ASM, Gersh BJ, Somers VK. Obstructive Sleep Apnea. JAMA. 2003 Oct 8;290(14):1906-14
10. Kim J, Kim Y, Yang KI, Kim DE, Kim SA. The relationship between sleep disturbance and functional status in mild stroke patients. Ann Rehabil Med.2015;39:545-52. https://doi.org/10.5535/arm.2015.39.4.545.
11. Miano S, Fanfulla F, Nobili L, Heinzer R, Haba-Rubio J, Berger M, et al. SAS CARE 1: Sleep architecture changes in a cohort of patients with Ischemic Stroke/TIA. Sleep Medicine. 2022; 98:106-13. https://doi.org/10.1016/j.sleep.2022.06.002.
12. Hasan F, Gordon C, Wu D, Huang HC, Yuliana LT, Susatia B, et al. Dynamic prevalence of sleep disorders following stroke or transient ischemic attack: systematic review and meta-analysis. Stroke. 2021;52:655-63. https://doi.org/10.1161/STROKEAHA.120.029847.
13. Hofmeijer J, van Kaam R, Vermeer SE, van Putten MJAM. Severely disturbed sleep in patients with acute ischemic stroke on stroke units: a pilot study. Front Neurol 2019;10:1109. https://doi.org/10.3389/fneur.2019.01109.
14. Ott SR, Fanfulla F, Miano S, Horvath T, Seiler A, Bernasconi C, et al.. SAS Care 1: Sleepdisordered breathing in acute stroke an transient ischaemic attack - Prevalence, evolution and association with functional outcome at 3 months, A prospective observational polysomnography study. ERJ Open Res. 2020;6. https://doi.org/10.1183/23120541.00334-2019. 00334-2019.
15. Baylan S, Griffiths S, Grant N, Broomfield NM, Evans JJ, Gardani M. Incidence and prevalence of post-stroke insomnia: a systematic review and metaanalysis. Sleep Med Rev. 2020;49:101222. https://doi.org/10.1016/j.smrv.2019.101222.
16. Hermann DM, Bassetti CL. Role of sleep-disordered breathing and sleep-wake disturbances for stroke and stroke recovery. Neurology 2016;87:1407e16. https://doi.org/10.1212/WNL.0000000000003037.
17. Ferre A, Rib_o M, Rodríguez-Luna D, Romero O, Sampol G, Molina CA, et al.. Strokes and their relationship with sleep and sleep disorders. Neurologia 2013;28:103e18. https://doi.org/10.1016/j.nrl.2010.09.016.
18. Terzoudi A, Vorvolakos T, Heliopoulos I, Livaditis M, Vadikolias K, Piperidou H. Sleep architecture in stroke and relation to outcome. EurNeurol 2009;61:16e22. https://doi.org/10.1159/000165344.
19. Bassetti C, Aldrich MS, Quint D. Sleep-disordered breathing in patients with acute supra- and infratentorial strokes. A prospective study of 39 patients. Stroke 1997;28:1765e72. https://doi.org/10.1161/01.str.28.9.1765.
20. Pajediene E, Pajeda A, Urnieziute G, Paulekas E, Liesiene V, Bileviciute-Ljungar I, et al. Subjective and objective features of sleep disorders in patients with acute ischemic or haemorrhagic stroke: It is not only sleep apnoea which is important. Med Hypotheses 2020;136:109512. https://doi.org/10.1016/j.mehy.2019.109512.
21. Koo DL, Nam H, Thomas RJ, Yun CH. Sleep disturbances as a risk factor for stroke. J Stroke. 2018;20(1):12-32. doi:10.5853/jos.2017.02887
22. McDermott M, Brown DL, Chervin RD. Sleep disorders and the risk of stroke. Expert Rev Neurother. 2018;18(7):523-531. doi:10.1080/14737175.2018.1489239
23. Chung F, Yegneswaran B, Liao P, et al. STOP questionnaire: a tool to screen patients for obstructive sleep apnea. Anesthesiology 2008; 108(5):812-821. doi:10.1097/ALN.0b013e31816d83e4
24. Kang K, Park KS, Kim JE, et al. Usefulness of the Berlin questionnaire to identify patients at high risk for obstructive sleep apnea: A population-based door-to-door study. Sleep Breath 2013;17(2):803-810. doi:10.1007/s11325-012-0767-2
25. JohnsMW. A new method for measuring daytime sleepiness: the Epworth sleepiness scale. Sleep. 1991;14(6):540-545. doi:10.1093/sleep/14.6.540
26. Barone DA, Krieger AC. Stroke and obstructive sleep apnea: a review. CurrAtheroscler Rep 2013;15(7):334. doi:10.1007/s11883-013-0334-8

27. Li M, Hou WS, Zhang XW, Tang ZY. Obstructive sleep apnea and risk of stroke: a meta-analysis of prospective studies. Int J Cardiol 2014;172(2):466-469. doi:10.1016/j.ijcard.2013.12.230
28. Redline S, Yenokyan G, Gottlieb DJ, et al. Obstructive sleep apnea–hypopnea and incident stroke. Am J RespirCrit Care Med 2010;182(2):269-277. doi:10.1164/rccm.200911-1746OC
29. Johnson KG, Johnson DC. Frequency of sleep apnea in stroke and TIA patients: A meta-analysis. J Clin Sleep Med 2010;6(2):131-137.
30. Budhiraja R, Parthasarathy S, Quan SF. Endothelial dysfunction in obstructive sleep apnea. J Clin Sleep Med 2007;3(4):409-415.
31. Jensen MLF, Vestergaard MB, Tønnesen P, Larsson HBW, Jennum PJ. Cerebral blood flow, oxygen metabolism, and lactate during hypoxiain patients with obstructive sleep apnea. Sleep 2018;41(3). doi:10.1093/sleep/zsy001
32. SannerBM, Konermann M, Tepel M, et al. Platelet function in patients with obstructive sleep apnoea syndrome. EurRespir J 2000;16(4):648-652. doi:10.1034/j.1399-3003.2000.16d14.x
33. Parasram M, Segal AZ. Sleep disorders and stroke: does treatment of obstructive sleep apnea decrease risk of ischemic stroke? Curr Treat Options Neurol 2019;21(7):29. doi:10.1007/s11940-019-0575-0
34. Brown DL, Mowla A, McDermott M, et al. Ischemic stroke subtype and presence of sleep-disordered breathing: the BASIC sleep apnea study. J Stroke Cerebrovasc Dis 2015;24(2):388-393. doi:10.1016/j.jstrokecerebrovasdis.2014.09.007
35. Yaggi HK, Concato J, Kernan WN, et al. Obstructive sleep apnea as a risk factor for stroke and death. N Engl J Med 2005;353(19):2034-2041. doi:10.1056/NEJMoa043104
36. Sahlin C, Sandberg O, Gustafson Y, et al. Obstructive sleep apnea is a risk factor for death in patients with stroke: a 10-year follow-up. Arch Inten Med 2008;168(3):297-301. doi:10.1001/archinternmed.2007.70