

# A COMPARATIVE POLYSOMNOGRAPHIC STUDY BETWEEN PATIENTS WITH WAKE UP AND DAYTIME ISCHEMIC STROKE ADMITTED AT ALEXANDRIA UNIVERSITY HOSPITALS

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## Introduction

Cerebrovascular stroke and sleep are closely correlated.<sup>1</sup> Several pathophysiological mechanisms have been proposed in the literature that explain the occurrence of a stroke at high rates with certain sleep disorders.<sup>2</sup>

Obstructive sleep apnea (OSA) is on the top of the list of sleep disorders increasing the risk for cerebrovascular stroke.<sup>3</sup> Obstructive sleep apnea increases stroke risk via several direct and indirect mechanisms. Direct mechanisms include increased sympathetic activity, nocturnal hypertension, intermittent hypoxia, arousal responses, hemodynamic instability, oxidative stress, and endothelial dysfunction.<sup>4</sup> Indirect mechanisms include increasing the risk for other stroke risk factors such as diabetes mellitus, hypertension, and cardiac arrhythmias. Insomnia, periodic limb movements (PLM), and restless leg syndrome (RLS) are other sleep disorders that increase stroke risk. They were proposed to increase the stroke risk via increasing sympathetic activity, micro-arousals, oxidative stress, oxidative stress, hypoxia, inflammation, and metabolic dysregulation. Sleep duration was also reported to be related to stroke. Both long and short sleep duration were reported to increase sleep risk.<sup>5</sup>

Though the relationship between stroke and sleep is well established, data is scarce about the correlation between sleep architecture and stroke time onset. The sleep architecture was compared between patients with wake-up stroke (WUS) and patients with daytime stroke (DTS) in few studies literature studies, and the data from these studies are conflicting.<sup>6</sup>

These studies included all types of cerebrovascular strokes and did not specify a certain type. A common finding reported by these studies was that OSA was the only risk factor for WUS. However, the data are scarce about whether there is a difference between the polysomnographic architecture of patients with DTS and WUS. Therefore, we addressed this point in this study. We aim at studying the difference in sleep architecture among patients with DTS and patients with WUS ischemic thrombotic cerebrovascular stroke among a sample of Egyptian population.

## Aim of work

To compare the polysomnographic profile between patients with wake-up thrombotic cerebrovascular stroke and patients with daytime thrombotic cerebrovascular stroke.

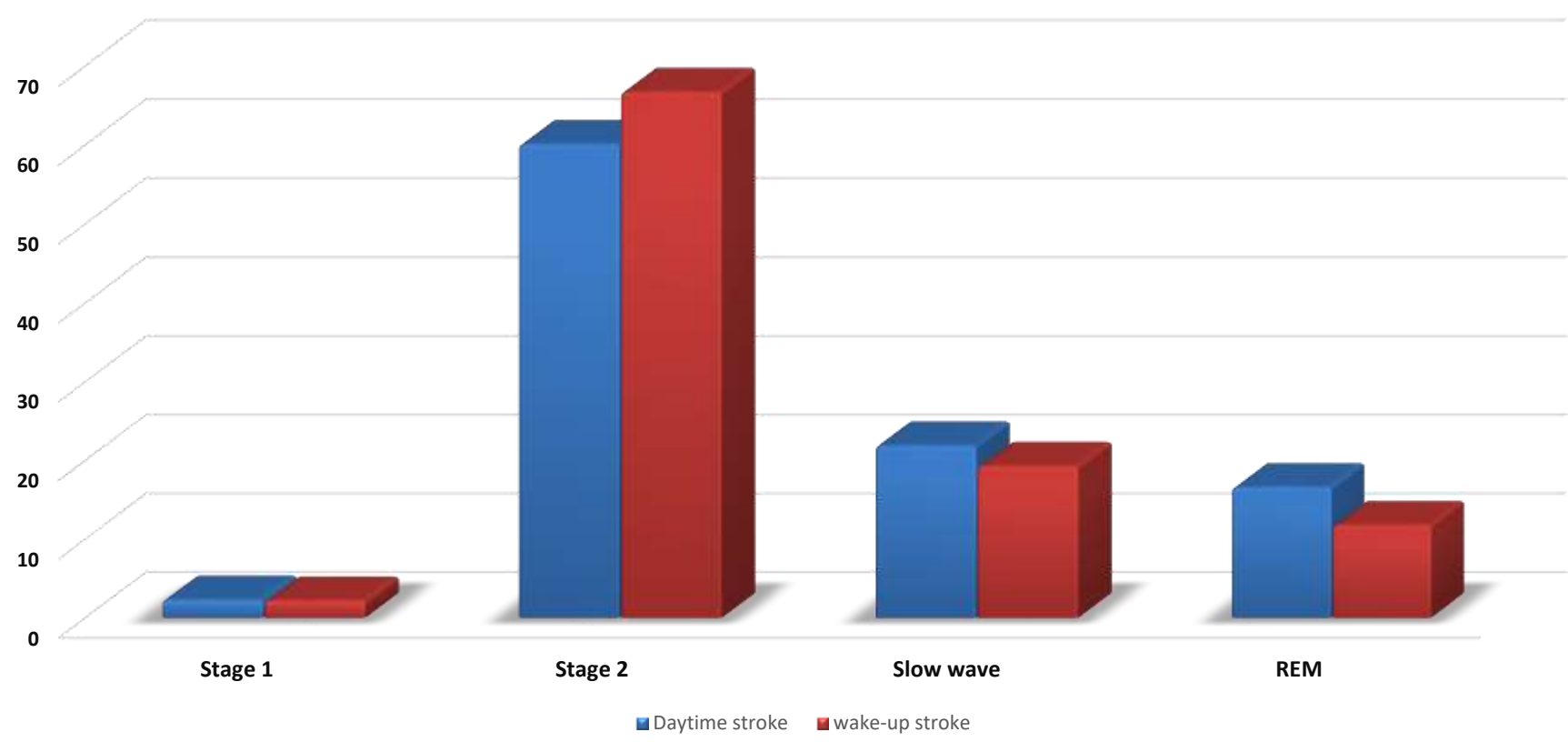
## Methods and Materials

Forty patients with cerebrovascular stroke who were admitted at the department of neurology at Alexandria university hospitals. Twenty of these patients had their stroke onset on awakening, and the remaining 20 had their stroke onset during the daytime. Inclusion criteria included patients older than 45 years diagnosed with ischemic thrombotic cerebrovascular stroke within one week with a National Institutes of Health Stroke Scale (NIHSS) of 20 or less. Patients with stroke in young, embolic stroke, or Glasgow coma scale less than 11 were excluded from the study. Patients with co-morbid psychiatric illness affecting sleep; like depression or anxiety, patients on medications that affect sleep e.g. hypnotics or stimulants, and patients with pre-existing documented sleep disorders were excluded from this study.

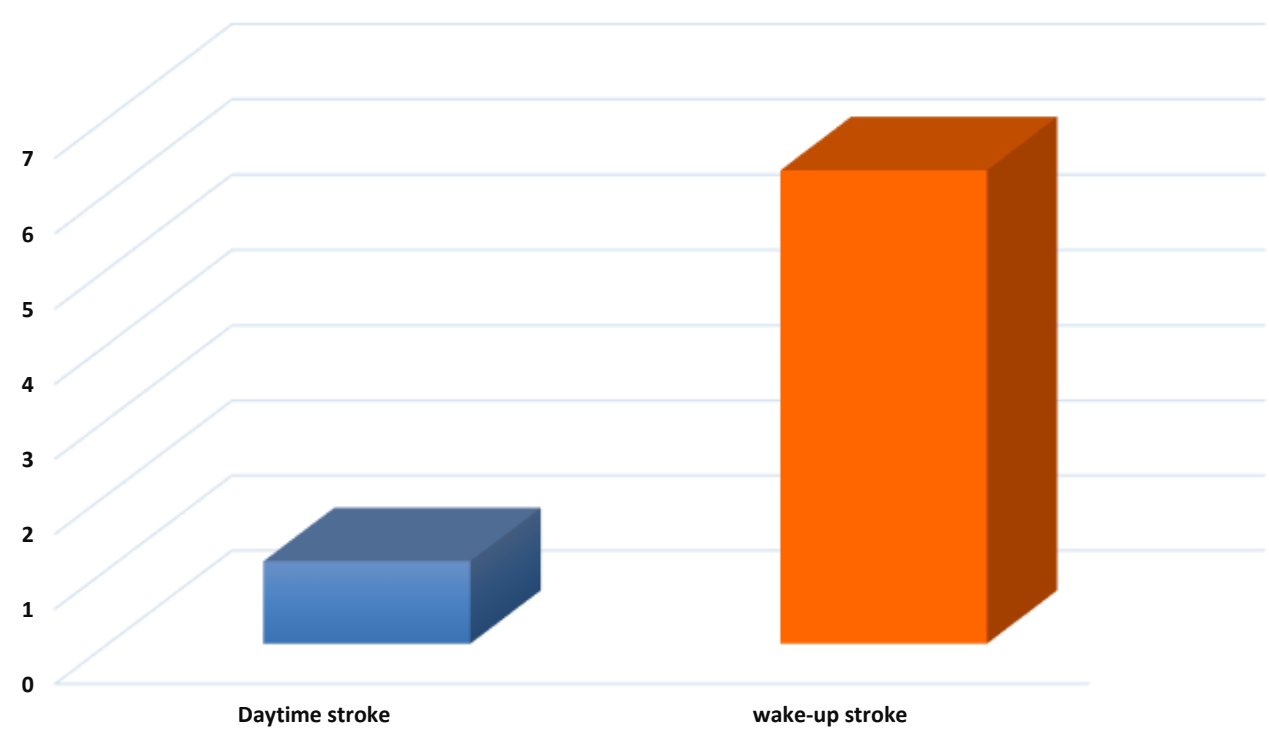
**All patients are subjected to the following :**

- Complete history taking
- Complete neurological examination
- NIHSS assessment and blood pressure assessment
- laboratory profile
- brain imaging
- Arabic validated version of Pittsburg sleep quality index (PSQI) was administered for all patients
- An overnight polysomnographic (PSG) study was performed to all patients .

## Results



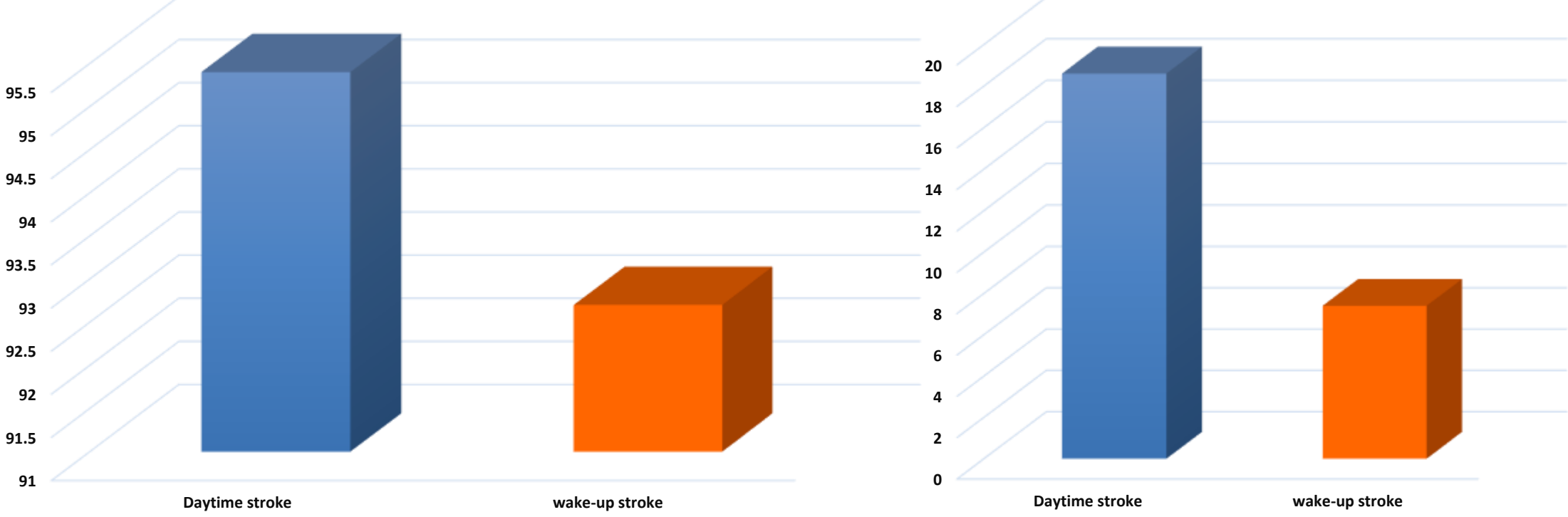
**Figure (1):** A comparative analysis of studied patients according sleep stage



**Figure (2):** A comparative analysis between the two studied groups according to their AHI during REM

Table (1): Significant polysomnographic differences between WUS and DTS patients

	WUS (n=20)	DTS (n=20)	P-value
Stage 2% (Mean ± SD)	66.67 ± 6.92	60.09 ± 7.67	0.018*
REM% (Mean ± SD)	11.76 ± 5.48	16.59 ± 5.33	0.008*
Early morning REM duration (Mean ± SD)	25.70 ± 13.13	4.15 ± 4.69	<0.001*
AHI during REM sleep (Mean ± SD)	6.29 ± 10.18	1.10 ± 4.57	0.009*
Mean O <sub>2</sub> during REM sleep	92.70 ± 3.63	95.45 ± 1.35	0.012*
PLM index (Mean ± SD)	7.45 ± 7.30	18.66 ± 24.46	0.043*
PLM-related arousals (Mean ± SD)	0.18 ± 0.24	0.501 ± 0.52	0.019*



**Figure (3) and (4):** Comparative analysis between the studied patients according to Mean O<sub>2</sub> Saturation during REM and PLM

## Conclusions

The polysomnographic parameters are significantly different between patients with DTS and WUS. Shorter disrupted REM might increase the risk to develop WUS through higher apnea-hypopnea index and lower oxygen saturation in REM stage. Longer early morning REM might increase the risk to develop WUS. As autonomic instability increases, Fibrinolytic activity diminishes in the mornings, while platelet aggregability increases. Long stage 2 sleep might have increased the risk of WUS via increasing the transient hypoxic episodes during cyclic alternative pattern(CAP). PLM index could be a risk factor to develop stroke during the morning via sympathetic overactivity, oxidative stress, inflammation, hypoxia, and/or metabolic dysregulation.

## Recommendations

- Further multi-centric well-designed retrospective and prospective studies are needed to observe the relation of REM stage to WUS and DTS.
- Further studies are needed to study the relation of stage 2 to WUS, to analyze analyze (CAP) and its relation to WUS.
- Further studies are needed to study the correlation between periodic limb movement and daytime stroke.

## Contact

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## References

1. Mims KN, Kirsch D. Sleep and Stroke. Sleep Med Clin. 2016;11(1):39-51.
2. Koo DL, Nam H, Thomas RJ, Yun CH. Sleep disturbances as a risk factor for stroke. J Stroke. 2018;20(1):12-32.
3. Yaggi HK, Concato J, Kernan WN, Lichtman JH, Brass LM, Mohsenin V. Obstructive Sleep Apnea as a Risk Factor for Stroke and Death. N Engl J Med. 2005;353(19):2034-2041.
4. Verrier RL, Harper RM, Hobson JA. Cardiovascular Physiology: Central and Autonomic Regulation. In: Principles and Practice of Sleep Medicine. Elsevier Inc.; 2005:192-202.
5. Helbig AK, Stöckl D, Heier M, Ladwig KH, Meisinger C. Symptoms of insomnia and sleep duration and their association with incident strokes: Findings from the population-based MONICA/KORA Augsburg Cohort Study. PLoS One. 2015;10(7).
6. Mohammad Y, Almutlaq A, Al-Ruwaita A, Aldrees A, Alsubaie A, Al-Hussain F. Stroke during sleep and obstructive sleep apnea: there is a link. Neurol Sci. 2019;40(5):1001-1005.