Original Research

Association Between Sleep Duration and Functional Disability in Inpatient Stroke Rehabilitation: A Pilot Observational Study

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KEYWORDS
Rehabilitation; Sleep; Stroke

Abstract  Objective: To describe the change in sleep duration during inpatient rehabilitation and to determine if sleep quality and sleep duration is associated with functional disability for individuals after stroke. It was hypothesized that participants who experienced optimal sleep during inpatient rehabilitation would have greater functional ability at discharge.

Design: Longitudinal observation study.

Setting: Inpatient rehabilitation unit at a large, urban hospital.

Participants: Thirty-seven individuals with acute stroke (N=37; mean age, 62.5±11.8y, male=20, female=17) were recruited from September 2018 to September 2019. Participants were invited to participate in the study by clinical personnel associated with their usual care as they were admitted to inpatient rehabilitation.

Interventions: Not applicable.

Main Outcome Measures: Participants were asked to wear an actigraph for the duration of their rehabilitation program to assess sleep. The first 3 nights of actigraphy data were averaged to obtain total sleep time (TST) and sleep efficiency (SE) at admission, and the last 3 nights were averaged for TST and SE at discharge. Functional disability (primary outcome was FIM) at admission and discharge was gathered from the participants’ medical records. One-way analysis of variance and chi-square analyses assessed for group differences, and regression modeling was used to determine if sleep was associated with functional ability at discharge.

List of abbreviations: ADL, activities of daily living; REM, rapid eye movement; SE, sleep efficiency; TST, total sleep time.

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Changes in sleep outcomes often occur in individuals after a stroke. These changes can include change in total sleep time (TST), sleep efficiency (SE), sleep quality, and alterations in sleep architecture. For some individuals poststroke, TST increases during the acute stage, whereas others experience a decrease in TST. For some individuals, this decrease in TST may be because of alterations in neural control of sleep-wake centers in the brain or breathing during sleep and production or release of melatonin. Given 7 or more hours of sleep is the recommended TST for healthy adults, it is evident that a stroke may be deleterious on sleep duration and quality. However, an increase in TST in the acute stage of stroke may be conducive to healing because sleep has been shown to be crucial for neuroplasticity, removal of toxins from the brain, and recovery. For individuals who experience a reduction in TST and sleep quality in the acute stage of stroke, it is plausible that a return to optimal sleep duration and quality would be associated with recovery.

Because learning new and/or relearning motor skills is a main goal of inpatient stroke rehabilitation, it is plausible that sleep would be a key factor in recovery for persons after a stroke. Siengsukon et al found that individuals after a stroke who slept after practicing a task had an improvement in performance on the task compared with individuals who stayed awake. Furthermore, research has shown that as a survivor of stroke experiences greater levels of insomnia during rehabilitation, their subsequent levels of daily activity decreased at discharge and 1 month after discharge. Also, sleep apnea and sleep-disordered breathing have been associated with poorer functional recovery after inpatient rehabilitation, length of rehabilitation stay, more dependence with activities of daily living (ADL), and worse functional recovery at 3 months but not 6 months poststroke. Furthermore, treatment of sleep apnea by continuous positive airway pressure resulted in improved impairment severity and functional ability in individuals in inpatient rehabilitation compared with those not treated by continuous positive airway pressure. Individuals with stroke who self-reported poor sleep quality or higher sleep disturbance demonstrated less functional improvement during inpatient rehabilitation than those with good sleep quality or less sleep disturbance.

Two studies to our knowledge have used actigraphy to assess if sleep outcomes are associated with stroke recovery. One study reported that higher wake after sleep onset assessed via actigraphy in the acute phase of stroke was associated with lower ADL function at 6 months poststroke after controlling for demographic and clinical variables, but they did not examine if sleep outcomes were associated with functional ability or recovery at discharge from inpatient rehabilitation. Another study reported total sleep time and sleep efficiency assessed using actigraphy within 2 weeks of admission to inpatient rehabilitation after stroke were associated with functional ability at discharge from inpatient rehabilitation. However, only 14 individuals with stroke were enrolled in this study, and sleep was assessed only at admission (within 2 weeks) of inpatient rehabilitation.

The purpose of this longitudinal observation study was to describe change in sleep duration during inpatient rehabilitation and to determine if sleep duration and sleep quality during acute inpatient rehabilitation is associated with functional disability for individuals after stroke. Given previous research that has shown optimal sleep duration may improve functional ability, that insomnia has been negatively associated with poststroke ADL levels, and that sleep outcomes may affect functional ability after stroke, we hypothesized participants who experienced optimal sleep duration (7-9 h/night) during inpatient rehabilitation would have greater functional ability at discharge.

**Methods**

**Design and ethics**

This was a longitudinal observational study. This study was conducted according to the standards of the Declaration of Helsinki and with approval of the St. Luke’s Health System institutional review board and the University of Kansas Medical Center’s institutional review board.

**Participants**

Individuals with stroke who were admitted for inpatient rehabilitation at a large, urban hospital were enrolled in this study. Participants were invited from September 2018 to September 2019 to participate in the study by clinical personnel associated with their usual care as they were admitted to inpatient rehabilitation. Participants were included if they were aged between 18-80 years, had a stroke diagnosed by a physician, were admitted to inpatient rehabilitation, spoke English, and were able to provide consent to admission to inpatient rehabilitation. Participants were excluded if they had prior history of a stroke, had nervous system disorders other than a stroke, or had infectious diseases.

**Results**

Sixteen participants (43%) were categorized as “good sleepers” and 21 (57%) were “poor sleepers” based on their TST at admission. Of the poor sleepers, 14 participants (66%) remained short sleepers (<7 h at admission and discharge). Sleep outcomes did not significantly predict FIM score at discharge.

**Conclusions**

Most participants had less than optimal sleep duration during inpatient rehabilitation. Efforts may be warranted to optimize sleep during inpatient rehabilitation.
Data collection

After completing informed consent, an actigraph was placed on the participant’s less affected wrist by study personnel to ensure proper placement and fit. Participants were instructed to wear the device continuously for the duration of their inpatient rehabilitation stay to objectively assess TST and SE (defined as TST/time in bed × 100). Actigraphy has been shown to be a valid and reliable method to assess sleep behavior. The FIM score at admission and discharge conducted by clinicians as part of standard of care, when the stroke occurred, stroke location (if magnetic resonance imaging or computed tomography scan was available), current medications, and relevant past medical history were obtained from the participant’s medical record. The FIM is an 18-item instrument that measures and assesses functional independence on a 1-7 scale. Higher scores indicate greater functional independence across 6 domains (self-care, sphincter control, transfers [mobility], locomotion, communication, social cognition).

Data processing

A trained research assistant reviewed all actigraphy output to verify as per expert guidelines 24-h/d wear for the length of inpatient rehabilitation. Actigraphy data were analyzed using the Cole-Kripke algorithm within ActiLife software (version 6.11.9). The trained research assistant then reviewed each actigraph report for irregularities following automated scoring and adjusted scoring as per expert guidelines. The sleep variables of interest were TST and SE. Each participant’s first 3 nights of actigraphy data were averaged to obtain TST and SE at admission, and the last 3 nights were averaged for TST and SE at discharge.

Participants were dichotomized into a “good sleeper” group or a “poor sleeper” group depending on how their sleep changed over the course of their inpatient rehabilitation stay. They were considered “good sleepers” if their sleep duration was (1) optimal (7-9h) at admission and remained 7-9 hours at discharge; (2) long duration (>9h) at admission but normalized to 7-9 hours at discharge; or (3) short duration (<7h) at admission but normalized to 7-9 hours at discharge. Participants were considered “poor sleepers” if their sleep duration was (1) optimal (7-9h) at admission but changed to a longer sleep duration (>9h) at discharge; (2) optimal (7-9h) at admission but changed to a shorter sleep period (<7h); (3) short sleep duration (<7h) at admission and remained short duration (<7h) at discharge; or (4) long duration (>9h) at admission and remained long (>9h) at discharge.

Sample size

Using G*Power, we determined a total of 34 participants was needed to detect a medium effect size ($\eta^2$) of 0.25 with 1 predictor in the linear regression models for 80% power and allowing for a type I error of 0.05.

Statistical analyses

Data were analyzed using SPSS with an $\alpha$ of 0.05. The average and SDs or frequency were calculated for demographic variables for each group. One-way analyses of variance were analyzed for differences between the good sleepers group and the poor sleepers group at admission for number of nights the actigraph was worn, age, body mass index (because of association with sleep apnea), number of comorbidities, and number of medications. One-way analyses of variance were also used to determine if there were group differences for admission and discharge on TST, SE, and FIM. Chi-square analyses were used to assess for group differences on race, highest degree earned, marital status, employment status, type of medications taken, type of stroke, side of the brain where the stroke occurred, and location of the stroke. Four linear regression models were used to determine if TST and SE at admission or discharge is associated with functional independence at discharge from inpatient rehabilitation. TST at admission, TST at discharge, SE at admission, and SE at discharge were used as the predictor variable in the regression models, and FIM at discharge was the dependent variable.

Results

Forty-seven individuals enrolled in this study. The average age was 61 years old, and 36.2% were married; most were White (68.1%), have high school–level education (51.1%), and either work full time (44.7%) or are retired (44.7%) (table 1). Also, the average number of comorbidities was 4.3, the average number of medications was 9.2, and most had an ischemic stroke (74.5%) affecting the left side of the brain (57.4%) with a variety of locations (see table 1). Ten people were excluded from data analysis because of incomplete data: 1 individual died while in inpatient rehabilitation, 2 people were discharged to the intensive care unit, 1 participant was discharged to acute care, 5 people experienced issues with the actigraph device (ie, uncomfortable, device programmed incorrectly), and 1 person dropped out of the study for an unknown reason. Therefore, 37 participants completed the study and were included in data analysis.

Of the 37 participants, 16 were “good sleepers” (mean age, 63.2±13.5y) and 21 were “poor sleepers” (mean age, 61.9±10.6y). Of the “good sleepers,” 5 participants had optimal sleep duration at admission and at discharge, 2 participants changed from long sleep duration to optimal sleep duration, and 9 participants changed from short sleep duration to optimal sleep duration. For the “poor sleepers,” 1 individual changed from optimal sleep duration to long sleep duration, 6 participants changed from optimal sleep duration to short sleep duration, and 14 participants remained short duration sleepers. No participants remained long duration sleepers from admission to discharge (table 2), and there were no evident daytime naps. There were no statistically significant differences between the “poor sleepers” and “good sleepers” on number of nights the actigraph was worn, demographic variables, stroke variables (table 1), or functional ability at admission or discharge.
Simple linear regression models found TST at discharge ($R^2=0.000$, $F_{1.35}=0.011$, $P=.916$), SE at admission ($R^2=0.063$, $F_{1.35}=2.351$, $P=.134$), and SE at discharge ($R^2=0.024$, $F_{1.35}=0.846$, $P=.364$) were not significant predictors of FIM scores at discharge (Table 3). TST at admission ($R^2=0.098$, $F_{1.35}=3.815$, $P=.059$) was approaching statistical significance and explained 9.8% of the variance in FIM discharge score (see Table 3; fig 1).

### Discussion

The results of this study support prior research that TST can both increase and decrease in individuals after a stroke. Over half of the participants had poor sleep from admission to inpatient rehabilitation to discharge, and sleep duration at admission may contribute to functional ability at
discharge of inpatient rehabilitation. Future studies may consider if interventions to optimize sleep duration during inpatient rehabilitation might improve functional outcomes.

It is concerning that over half of the participants in this study (57%) were poor sleepers with sleep that either remained short duration from admission to discharge, changed from optimal sleep duration to long sleep duration, or changed from optimal sleep duration to short sleep duration. It is particularly concerning that the largest subcategory of poor sleep (66%) was those whose sleep remained short (<7h) from admission to discharge. It is not clear why the “poor sleep” individuals did not have sleep duration within the optimal range of 7-9 hours. It is likely multifactorial and because of primary factors (the stroke affecting sleep centers within the brain and neural excitability) and secondary factors (behavioral, environmental, socioeconomical, psychosocial, emotional). Sleep duration during inpatient rehabilitation after stroke can be affected because of changes in one’s sleep schedule, an unfamiliar sleep environment, an environment less conducive for sleep (temperature, light, noise), worry about medical condition, finances, loneliness, anxiety, and post-stroke fatigue.4,34-37 Given that sleep is critical for neuroplasticity and optimal functioning of the body,17,38-47 it seems prudent to investigate factors (personal, environmental, health system) that are contributing to maladaptive changes to sleep.

Our hypothesis that optimal sleep duration during inpatient rehabilitation would be associated with greater functional ability at discharge was not supported. Our results are contrary to a study by Bakken et al1 that found sleep outcomes explained 7.1% of the variance in ADL performance at 6 months poststroke and Cherkasskey et al19 who reported greater respiratory disturbance because of sleep-related breathing disorder explained nearly 21% of the variance in change in functional ability from admission to inpatient rehabilitation to discharge. Animal research has demonstrated rapid-eye movement (REM) sleep 24 hours after an ischemic injury was capable of reducing the infarct volume in rats 7 days after stroke.48 Furthermore, human research has shown higher wake after sleep onset and non-REM stage 1 and lower sleep efficiency5 and lower REM latency4 that are associated with poorer outcomes after stroke. Also, slow wave sleep, REM, and sleep efficiency were associated with cognitive function during the acute and subacute phases after stroke.49 Taken together, it appears possible that early sleep patterns after stroke may affect functional ability and recovery after a stroke. Future studies may consider if screening for sleep disorders as well as interventions at a personal or organizational level to optimize sleep duration.

| Participants | TST at Admission (h) | TST at Discharge (h) | SE at Admission (%) | SE at Discharge (%) | FIM at Admission | FIM at Discharge |
|--------------|----------------------|---------------------|---------------------|---------------------|-----------------|-----------------|
| Good sleepers (n=16) | 7.9±0.27 | 8.0±0.78 | 86.6±3.9 | 86.2±2.8 | 76.8±16.8 | 102.2±8.8 |
| Remained Optimal (7-9h) (n=5) | 9.2±0.27 | 8.7±0.17 | 92.2±5.2 | 89.9±8.0 | 76.0±1.4 | 108.0±8.5 |
| Short sleep (<7h) to optimal sleep (n=9) | 5.9±1.3 | 7.6±0.58 | 75.9±13.8 | 86.7±6.5 | 49.5±14.3 | 81.3±23.9 |
| Poor sleepers (n=21) | 8.9±0.0 | 9.5±0.0 | 95.5±0.0 | 95.8±0.0 | 71.0±0.0 | 105.0±0.0 |
| Optimal sleep then long sleep (>9h) (n=1) | 7.3±0.28 | 6.4±0.60 | 88.0±4.0 | 83.3±6.5 | 65.5±13.3 | 95.7±10.8 |
| Remained short (<7h) (n=14) | 5.9±0.68 | 5.7±1.0 | 76.5±9.1 | 76.4±16.3 | 60.4±16.0 | 93.6±19.9 |

NOTE. Data reported as mean ± SD.

| Variables | Unstandardized Coefficients | Standardized Coefficients | Significance (P Value) | R² |
|-----------|-----------------------------|---------------------------|------------------------|----|
| Model 1 TST at admission | 0.076 | 0.039 | 0.314 | 1.95 | .059 | 0.098 |
| Model 2 TST at discharge | −0.044 | 0.039 | −0.018 | −0.106 | .916 | 0.000 |
| Model 3 SE at admission | 0.441 | 0.288 | 0.251 | 1.533 | .134 | 0.063 |
| Model 4 SE at discharge | −0.244 | 0.265 | −0.154 | −0.920 | .364 | 0.024 |
during inpatient rehabilitation might improve functional outcomes, enhance recovery, and reduce length of stay.

**Study limitations**

There were several limitations to this study. First, the small sample size limits interpretation of the results. Also, because of lack of information about the participants’ sleep prior to their stroke, it is unclear if the stroke or inpatient rehabilitation environment contributed to change in sleep duration or sleep efficiency at admission to inpatient rehabilitation. There are other factors that may mediate the relationship between sleep and functionality poststroke rehabilitation. Types of medications,50,51 social support,52,53 severity54 and type55 of comorbid conditions, and socioeconomic status56 have been shown to influence recovery after stroke. However, there were no group difference between the “good sleepers” and “poor sleepers” in number of medications, use of stimulant or sleep aid, number of comorbidities, marital status, and highest degree earned. Also, there is limited generalizability because all participants attended the same inpatient rehabilitation facility.

Another limitation is we reported functional ability using 1 outcome. Although the FIM has been shown to be a valid and reliable measure of functional independence,29 there may be a functional outcome measure or another domain that would be more sensitive to change in sleep duration or quality. Measuring changes in cognition may have been more sensitive to alterations in sleep duration and quality given cognition performance is highly affected by sleep disturbances. Previous animal and human research shows cognition is one of the first functions to change when sleep deprived15,38,39; thus it may be plausible cognition may be more sensitive to changes in sleep during inpatient rehabilitation after stroke and would be a preferable functional outcome to assess. Also, the Centers for Medicare and Medicaid Services recently removed the FIM from the inpatient rehabilitation facility patient assessment instrument.

**Conclusions**

This study demonstrated TST can both increase and decrease from admission to inpatient rehabilitation to discharge, and most participants had less than optimal sleep duration during inpatient rehabilitation. Although it remains unclear why some participants experience poor sleep during inpatient rehabilitation, it suggests the need for further research to examine what factors may aid in contributing to optimal sleep during the inpatient rehabilitation setting.

**Suppliers**

a. ActiLife software version 6.11.9; ActiGraph Corp, Pensacola, FL.

b. G*Power 3; Heinrich Heine University Düsseldorf.

c. SPSS; IBM.
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References

1. Sterr A, Herron K, Dijk DJ, Ellis J. Time to wake-up: sleep problems and daytime sleepiness in long-term stroke survivors. Brain Inj 2008;22:575–9.
2. Alberti A. Sleep changes. Front Neurol Neurosci 2012;30:38–40.
3. Terzoudi A, Vorvolakos T, Heliopolous I, Livaditis M, Vadikolias K, Piberidou H. Sleep architecture in stroke and relation to outcome. Eur Neurol 2009;61:16–22.
4. Bakken LN, Lee KA, Kim HS, Finset A, Lerdal A. Sleep-wake patterns during the acute phase after first-ever stroke. Stroke Res Treat 2011;2011:936298.
5. Bakken LN, Kim HS, Finset A, Lerdal A. Stroke patients’ functions in personal activities of daily living in relation to sleep and socio-demographic and clinical variables in the acute phase after first-time stroke and at six months of follow-up. J Clin Nurs 2012;21:1886–95.
6. Bassetti CL, Aldrich MS. Sleep electroencephalogram changes in acute hemispheric stroke. Sleep Med 2001;2:185–94.
7. Muller C, Achermann P, Bischof M, Nirkko AC, Roth C, Bassetti CL. Visual and spectral analysis of sleep EEG in acute hemispheric stroke. Eur Neurol 2002;48:164–71.
8. Vock J, Achermann P, Bischof M, et al. Evolution of sleep and sleep EEG after hemispheric stroke. J Sleep Res 2002;11:331–8.
9. Lyons OD, Ryan CM. Sleep apnea and stroke. Can J Cardiol 2015;31:918–27.
10. Fiorina P, Lattuada G, Silvestrini C, Ponari O, Dall’Ago P. Disruption of nocturnal melatonin rhythm and immunological involvement in ischaemic stroke patients. Scand J Immunol 1999;50:228–31.
11. Watson NF, Badr MS, Belenky G, et al. Recommended amount of sleep for a healthy adult: a joint consensus statement of the American Academy of Sleep Medicine and Sleep Research Society. Sleep 2015;38:843–4.
12. Duss SB, Seiler A, Schmidt MH, et al. The role of sleep in recovery following ischemic stroke: a review of human and animal data. Neurobiol Sleep Circadian Rhythms 2017;2:94:105.
13. Xie L, Kang H, Xu Q, et al. Sleep drives metabolite clearance from the adult brain. Science 2013;342:373–7.
14. Fultz NE, Bonmassar G, Setsompop K, et al. Coupled electrophysiological, hemodynamic, and cerebrospinal fluid oscillations in human sleep. Science 2019;366:628–31.
15. Rechtschaffen A, Bergmann BM, Everson CA, Kushida CA, Gilliland MA. Sleep deprivation in the rat: X. Integration and discussion of the findings. Sleep 1989;12:68–87.
16. Siengsukon C, Al-Dughmi M, Al-Sharman A, Stevens S. Sleep parameters, functional status, and time post-stroke are associated with offline motor skill learning in people with chronic stroke. Front Neurol 2015;6:225.
17. Siengsukon CF, Boyd LA. Sleep to learn after stroke: implicit and explicit off-line motor learning. Neurosci Lett 2009;451:1–5.
18. Huang RJ, Lai CH, Lee SD, et al. Objective sleep measures in inpatients with subacute stroke associated with levels and improvements in activities of daily living. Arch Phys Med Rehabil 2018;99:699–706.
19. Cherkassky T, Oksenberg A, Froom P, Ring H. Sleep-related breathing disorders and rehabilitation outcome of stroke patients: a prospective study. Am J Phys Med Rehabil 2003;82:452–5.
43. Heffner KL, France CR, Trost Z, Ng HM, Pigeon WR. Chronic low back pain, sleep disturbance, and interleukin-6. Clin J Pain 2011;27:35-41.

44. Kahan V, Andersen ML, Tomimori J, Tufik S. Stress, immunity and skin collagen integrity: evidence from animal models and clinical conditions. Brain Behav Immun 2009;23:1089-95.

45. Mostaghimi L, Obermeyer WH, Ballamudi B, Martinez-Gonzalez D, Benca RM. Effects of sleep deprivation on wound healing. J Sleep Res 2005;14:213-9.

46. Davies SK, Ang JE, Revell VL, et al. Effect of sleep deprivation on the human metabolome. Proc Natl Acad Sci U S A 2014;111:10761-6.

47. Spiegel K, Leproult R, Van Cauter E. Impact of sleep debt on metabolic and endocrine function. Lancet 1999;354:1435-9.

48. Pace M, Adamantidis A, Facchin L, Bassetti C. Role of REM sleep, melanin concentrating hormone and orexin/hypocretin systems in the sleep deprivation pre-ischemia. PLoS One 2017;12:e0168430.

49. Siccoli MM, Rölli-Baumeler N, Achermann P, Bassetti CL. Correlation between sleep and cognitive functions after hemispheric ischaemic stroke. Eur J Neurol 2008;15:565-72.

50. Dietz MA, McDowell FH. Potentiation of rehabilitation: medication effects on the recovery of function after brain injury and stroke. J Stroke Cerebrovasc Dis 1991;1:37-48.

51. Adams Jr HP, Robinson RG. Improving recovery after stroke: a role for antidepressant medications? Stroke 2012;43:2829-32.

52. Vincent-Onabajo GO, Ihaza LO, Usman Ali M, et al. Impact of social support on participation after stroke in Nigeria. Top Stroke Rehabil 2016;23:305-10.

53. Tsouna-Hadjis E, Vemmos KN, Zakopoulos N, Stamatelopoulos S. First-stroke recovery process: the role of family social support. Arch Phys Med Rehabil 2000;81:881-7.

54. Ergul A, Hafez S, Fouda A, Fagan SC. Impact of comorbidities on acute injury and recovery in preclinical stroke research: focus on hypertension and diabetes. Transl Stroke Res 2016;7:248-60.

55. Tam AKH, Bayley MT. A narrative review of the impact of medical comorbidities on stroke rehabilitation outcomes. Disabil Rehabil 2018;40:1842-8.

56. Ouyang F, Wang Y, Huang W, et al. Association between socioeconomic status and post-stroke functional outcome in deprived rural southern China: a population-based study. BMC Neurol 2018;18:1-9.