High Prevalence of Supine Sleep in Ischemic Stroke Patients

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**Background and Purpose**—Sleep apnea is very common after stroke and is associated with poor outcome. Supine sleep is known to exacerbate apneas in the general sleep apnea population. We therefore investigated the pattern of sleep positions in the acute stroke period.

**Methods**—Inpatients with acute ischemic stroke underwent full polysomnography that included continuous monitoring of sleep positions. Sleep apnea severity was measured using the apnea-hypopnea index (AHI). Stroke severity was measured by the NIH Stroke Scale (NIHSS) at the time of study enrollment by certified study personnel. Percent total sleep time spent in the supine position was calculated and compared by stroke severity based on a median split of NIHSS using a Wilcoxon rank-sum test.

**Results**—Of the 30 patients, the median age was 67. The median AHI was 23 (IQR: 6, 47). Twenty-two patients (73%) had sleep apnea with an AHI ≥5. The vast majority of sleep time among the stroke cases was spent supine, with a median percent sleep time spent supine of 100 (IQR: 62, 100). The majority (63%) of subjects spent no time asleep in any of the nonsupine positions (prone, left, right). Median percent sleep time supine was 100 (IQR: 100, 100) in those with a higher NIHSS and 63 (IQR: 51, 100) in those with a lower NIHSS ($P<0.01$).

**Conclusions**—Given the high prevalence of supine sleep identified, research into positional therapy for stroke patients with sleep apnea seems warranted. (Stroke. 2008;39:2511-2514.)

**Key Words:** sleep apnea ■ obstructive ■ cerebrovascular accident ■ supine position

Obstructive sleep apnea (OSA) is very common after stroke, affecting more than half of ischemic stroke patients.1 OSA is an important condition in stroke patients not only because of its high prevalence, but also because of its association with poor outcomes after stroke including mortality and functional outcome.2,3 Although there does appear to be an improvement in OSA severity months after stroke,4-6 OSA prevalence remains high months and even years after stroke.4-7

There is a well-established relationship between OSA severity and body position during sleep. Supine positioning is associated with an increase in upper airway collapsibility8 and thus an increase in apnea frequency and duration.9 Supine sleep also necessitates a higher pressure from continuous positive airway pressure (CPAP) devices for effective OSA treatment.10 Subjects in the acute stroke period are more disabled than in the later months after stroke,11 which may affect their sleep positioning. Because of the greater disability, stroke patients in the early poststroke period may have a higher prevalence of supine sleep than in the chronic poststroke phase, contributing to a higher apnea-hypopnea index (AHI) early in the poststroke period. The contribution of sleep position to OSA severity also has implications for OSA treatment in stroke patients. We therefore sought to characterize the pattern of sleep positions in the acute stroke period and to compare the sleep positions in stroke patients with and without OSA.

**Materials and Methods**

**Subjects**

Subjects were recruited from the University of Michigan inpatient Neurology service. Subjects were eligible for the study if they were over 18 years with an ischemic stroke resulting in some degree of disability (modified Rankin Scale score ≥1). Exclusion criteria were: decompensated heart failure, cardiac or respiratory arrest or myocardial infarction within the prior 3 months, severe pneumonia, hypertension refractory to treatment, prior exposure to CPAP, previous pneumothorax, bulblous emphysema, and acute sinus or ear infection. These criteria were part of a parent clinical trial.

**Study Procedures**

Subjects had nocturnal polysomnography performed within the first 7 days of stroke symptom onset. Studies were performed during the stroke hospitalization or inpatient rehabilitation stay, or during a readmission to the General Clinical Research Center (GCRC). Polysomnography consisted of 4 EEG leads (C3-A2, C4-A1, O1-A2, and O2-A1 of the international electrode placement system), 2 electro-oculographic leads, chin and bilateral anterior tibialis surface
electromyograms, 3 ECG leads, nasal and oral thermocouples and nasal pressure cannula (oral and nasal airflow measures), thoracic and abdominal piezo-electric bands (thoracic and abdominal excursion), snoring monitor, and finger pulse oximetry. An apnea was defined as ≥10 seconds of complete airflow cessation. An hypopnea was defined as a reduction in airflow, chest excursion, or abdominal excursion that led to ≥4% oxygen desaturation, awakening, or arousal. OSA was defined as ≥5 apneas or hypopneas per hour of sleep.12 Split-night studies, in which the second half (approximately) of the study was used to titrate positive airway pressure, were performed when the AHI during the first half appeared to be >20 and ≤60.13 Time spent in various sleep positions was documented continuously by the sleep technologist and classified as prone, supine, left, or right side. An electronic position sensor was not used. For split-night studies, the entire night was considered. No instructions were given to the subjects about sleep position. Sleep stage scoring was performed by one experienced registered polysomnographic technologist for all studies to decrease variability. Positional OSA was defined as an overall AHI ≥5 and at least a 50% lower AHI in the lateral positions (left or right) than the supine position.14 Possible positional OSA was defined as an AHI ≥5 in the supine position and no sleep recorded in any other position. Nonpositional OSA was defined as an AHI ≥5 with less than a 50% reduction in AHI in the lateral positions (left or right) compared with the supine position (thus requiring sleep in a nonsupine position). Baseline clinical characteristics were documented from the medical record. Stroke severity was measured by the NIH Stroke Scale (NIHSS) at the time of study enrollment by certified study personnel. Ability to perform activities of daily living was quantified by the Barthel Index at the time of study enrollment.

Statistical Analysis

Frequencies and percentages were calculated for demographics, baseline characteristics, and sleep study results. Medians and interquartile ranges (IQR) were calculated for continuous variables. Total sleep time spent in the supine position (minutes) and percent total sleep time spent in the supine position were compared by sleep apnea status and by stroke severity based on a median split of NIHSS using Wilcoxon rank-sum tests. Analyses were performed using S-plus 7.0 for Windows. This study is approved by the University of Michigan Institutional Review Board.

Results

Thirty-two subjects were studied. Of these, 2 self-terminated the diagnostic study within 2 hours of initiation and were therefore excluded from analysis because of lack of usable data. There were 30 subjects remaining for analysis. Demographics and vascular risk factor information are found in Table 1. No subject was intubated during the sleep studies. Most subjects had no prestroke disability: 26 (87%) had a modified Rankin Scale score 0 to 1 before stroke. Strokes were nonlacunar in 22 (73%). Median baseline NIHSS scores was 7 (IQR: 3, 10). The median baseline NIHSS scores were as follows: 2: 27%, 3: 17%, 4: 40%, 5: 17%. There was clinical suspicion of dysphagia, and therefore a speech pathology swallow evaluation in 19 (64%). Thirteen (43%) had dysphagia based on this evaluation. Six (20%) subjects had dohhoff feeding tubes in place during the polysomnogram.

Most patients had their sleep studies performed during their hospitalization (n = 27), while 3 (10%) were studied after readmission to the GCRC. Split night studies were performed in 7 (23%); the remainder had full night diagnostic studies. Median minimal oxygen saturation was 88% (IQR: 82, 91). The median AHI was 23 (IQR: 6, 47). Twenty-two patients (73%) had OSA.

Median recorded total sleep time per stroke case was 262 minutes (IQR: 218, 322) of a median total record time of 415 minutes (IQR: 384, 447). Median total sleep time supine per stroke case was 209 minutes (IQR: 131, 270). The majority of sleep time among the stroke cases was spent supine, with a median percent total sleep time spent supine of 100 (62, 100). The majority (63%) of subjects spent no time asleep in any of the nonsupine positions (prone, left, right). Twenty-nine cases (97%) spent no time asleep in the prone position, 23 (77%) spent no time asleep on the left side, and 24 (80%) spent no time asleep on the right side. Median total sleep time and median percent total sleep time supine were significantly higher in those with a higher NIHSS compared with lower NIHSS, and nonsignificantly higher in those with OSA compared with those without OSA (Table 2). Of the 23 subjects who had full night diagnostic studies, 4 (17%) did not have OSA, 4 (17%) had positional

<table>
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<th>Table 2. Total Sleep Time and Percent Total Sleep Time Spent Supine by NIHSS and OSA Status</th>
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<td>NIHSS Status</td>
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<td>Median sleep time supine (minutes, IQR)</td>
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OSA indicates obstructive sleep apnea; NIHSS, NIH stroke scale; IQR, interquartile range.
OSA, and 15 (65%) had possible positional OSA with no sleep at all in the nonsupine positions. No subject clearly had nonpositional OSA.

**Discussion**

This study suggests that acute stroke patients with some disability spend the vast majority of their sleep in the supine position. Regardless of the cause, given the known association between supine posture and AHI, this finding has important implications for the assessment of OSA severity in the acute stroke period, as well as possible postural treatment options for stroke patients with OSA. The effects of supine positioning may be even more important in the more severely disabled patients, as those with a higher NIHSS spend an even greater percent of sleep time in the supine position.

Supine sleep was much more prominent (median 100%) in our stroke population than other reported populations. Other studies have reported supine sleep to represent 25% of sleep in those without a sleep disorder, 30% to 50% in those with positional OSA,15–17 27% in those with nonpositional OSA,15 and 42% in those being screened for OSA in an outpatient laboratory where subjects were encouraged to sleep supine.18

Positional OSA is found in more than half of OSA patients.15,19,20 Nonpositional OSA appears to be uncommon after stroke based on the current data, although this finding requires confirmation in larger studies. All of our subjects with adequate supine and nonsupine sleep showed positional apnea, wholly consistent with the hypothesis that acute stroke patients may be particularly vulnerable to any effects of position on airway patency during sleep. Any motor deficit involving upper airway muscles, as suggested by the moderate frequency of dysphagia in the current study, could well reduce airway patency during sleep. This effect could be exacerbated in the supine position, when the weight of the genioglossus muscle may further compromise airflow.21 Although the effect of posture on AHI in stroke patients has not been well studied, supine posture exacerbates apneas in general OSA patients and also likely does so in stroke patients.22,23

Overall, acute stroke patients in this study slept for only 4.4 hours, a value in keeping with the limited other reports of sleep duration in the early stroke period.24,25 Aside from the influence of environmental factors in the hospital, it is possible that stroke leads to relative insomnia.26 It is unknown whether lack of sleep affects stroke recovery.

Limitations of this study include the sample size. The results of this study are not generalizable to patients with very mild stroke. Although subjects in the current study were not given any instructions on sleep positioning, it is possible that the act of performing polysomnography altered sleep positioning. Polysomnography was shown in one study to increase supine positioning from 31% to 49% of time spent in bed of subjects with OSA.27 It is possible that some transient shifts in position were missed because of sleep technologist inattention. It seems unlikely that this would have biased the results, however. Unfortunately, we do not have results on sleep positioning in the subacute period with which to compare the current results. We also do not have information on the angle of the head of the bed, or the number of pillows used to elevate the head. Thus no comment can be made about the relationship between these factors and the severity of sleep apnea.

Postural therapy has been tried in the general OSA population and has been found to have a beneficial effect,16,20,28,29 although it is not as great an effect as CPAP.30 Given the low tolerance of stroke patients to CPAP,31,32 if our finding of stroke patients’ propensity for supine sleep is confirmed, research into positional therapy for stroke patients with OSA seems warranted.

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**Disclosures**

None.