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Sleep-Disordered Breathing and Poor Functional Outcome After Stroke

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Background and Purpose We objectively evaluated patients with recent stroke to determine the prevalence of sleep-disordered breathing (SDB) and whether SDB was associated with unfavorable clinical outcomes.

Methods Forty-seven patients with recent ischemic stroke (median, 13 days) were studied with computerized overnight oximetry for evidence of arterial oxyhemoglobin desaturation (Sa_{O_2}). Polysomnography was also performed on 19 patients. Medical history, sleep history, location of stroke, and severity of neurological deficit were recorded, and patients were observed by staff for evidence of snoring and excessive daytime sleepiness. Functional abilities were measured with the use of the Barthel Index (BI). Outcome variables included ability to return home at discharge, continued residence at home at 3 and 12 months, BI at discharge, BI at 3 and 12 months, and death from any cause at 12 months.

Results Mean Sa_{O_2} during oximetry was 94.0±1.7%, and percentage of recording time spent at <90% Sa_{O_2} was 4.3±5.7%. The number of desaturation events per hour of recording time (desaturation index [DI]) was 9.5±9.67, with 15 of 47 (32%) having DI >10 and 6 of 47 (13%) having DI >20. Oximetry measures of SDB correlated with lower BI scores at discharge and lower BI at 3- and 12-month follow-ups ($P \leq .05$, Pearson coefficients). Oximetry measures correlated with return home after discharge, but the association between oximetry measures and living at home was lost at 12 months. Two oximetry variables correlated with death at 1 year. Brain stem location correlated with higher DI and time at <90% Sa_{O_2}, but patients with hemispheric stroke and oximetry abnormalities also had worse functional outcome. No correlation was found between oximetry values and sex, age, preexisting medical conditions (except previous stroke), or severity of neurological deficit. Oximetry abnormalities were associated with a history of snoring. Polysomnography on 19 patients confirmed oximetry evidence of severe SDB. Eighteen of 19 patients (95%) had an apnea-hypopnea index (AHI) of >10 events per hour of recording, 13 of 19 (68%) had an AHI >20, and 10 of 19 (53%) had an AHI >30. Desaturation events were largely due to obstructive apneas.

Conclusions SDB accompanied by arterial oxyhemoglobin desaturation is common in patients undergoing rehabilitation after stroke and is associated with higher mortality at 1 year and lower BI scores at discharge and at 3 and 12 months after stroke. SDB may be an independent predictor of worse functional outcome. Obstructive sleep apnea appeared to be the most common form of SDB, and the frequent history of snoring suggests that SDB preceded the stroke in most patients. (***Stroke.* 1996;27:252-259.**)

Key Words • activities of daily living • rehabilitation • sleep apnea syndromes • stroke outcome

Sleep-disordered breathing is a frequent clinical phenomenon,[1,2] and the prevalence increases with age.[3-5] Sleep apnea, characterized by recurrent cessation of airflow, is the most common type of SDB and may be either obstructive or central, with many patients having both types.[6,7] Other periodic breathing patterns, including CSR, also occur during sleep.[8,9] Recurrent hypopneas and apneas accompany all forms of SDB, often causing OxyHb desaturation, frequent arousals, and disruption of sleep architecture. Fatigue, inattention, and excessive daytime sleepiness may result. Cardiopulmonary consequences associated with hypoxemia include pulmonary hypertension with right heart failure,[2,6] cardiac arrhythmias,[2,10-13] systemic hypertension,[2,13-18] and ischemic heart disease.[13,17]

There are several reasons to suspect that the prevalence of SDB is high after stroke, although few studies using objective measures have been reported. Stroke is a condition primarily of the elderly, and OSA, the most common form of SDB,[6] increases with age. A history of snoring (which usually accompanies OSA) is more common in stroke patients compared with age- and sex-matched patients admitted for other conditions and is an independent risk factor for stroke.[17,19-22] In a prospective study of 4368 middle-aged men, habitual or frequent snorers had an increased risk of combined heart disease and stroke, even when adjustments were made for age, obesity, history of hypertension, smoking, and alcohol use.[23]

It is also possible that strokes may cause SDB. Oropharyngeal muscular dysfunction accompanies both hemispheric and brain stem stroke.[24-27] During sleep, functional occlusion of the airway could result in OSA, especially in persons already predisposed to this condition. Disruption of central regulatory pathways may result in CSA, especially in brain stem stroke.[28] Finally, CSR may occur after stroke.[8] All three types of SDB (OSA, CSA, and CSR) may coexist, and all are accompanied by OxyHb desaturation and arousal. Two studies (both published as abstracts) using objective measures

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Selected Abbreviations and Acronyms

AHI = apnea-hypopnea index
BI = Barthel Index
CSA = central sleep apnea
CSR = Cheyne-Stokes respirations
DI = desaturation index
DI group = desaturation group (DI >10/h)
OSA = obstructive sleep apnea
OxyHb = oxyhemoglobin
PSG = polysomnography
Sa_{O_2} = arterial oxyhemoglobin saturation
SDB = sleep-disordered breathing

found that 70% to 72% of patients with recent ischemic stroke had significant SDB, defined by an AHI of ≥10 events per hour.[29,30] Another study found a much higher prevalence of sleep-related respiratory disturbances in 10 patients less than 1 year after stroke compared with matched control subjects.[31]

Regardless of whether SDB precedes or follows a stroke, it may result in daytime sleepiness, inattentiveness, and impaired cognitive functioning. In our clinical experience, apneic spells and snoring are frequently observed on a stroke rehabilitation service. We postulated that SDB might affect ability to participate in a rehabilitation program and contribute to unfavorable outcomes. The purposes of this study were to (1) determine the prevalence of SDB in ischemic stroke patients hospitalized on a rehabilitation service and (2) determine whether SDB was associated with important unfavorable clinical outcomes. Continuous computerized overnight oximetry was used to monitor Sa_{O_2}, and selected patients also received overnight PSG.

Subjects and Methods

Fifty-five patients with recent ischemic stroke admitted to a stroke rehabilitation ward were asked to participate in the study. Eight patients refused, leaving 47 consenting patients. Median age was 69 years (range, 42 to 87 years), there were 26 men and 21 women, and time from stroke until rehabilitation admission was a median of 13 days (range, 4 to 69 days). The study was approved by the appropriate institutional review committee. None of the patients had clinically evident congestive heart failure or required supplemental oxygen. No patient had a prior diagnosis of SDB. No control subjects were studied. Location of stroke was established with the use of clinical criteria and neuroimaging procedures, which were obtained in all patients. Forty-four of 47 patients (94%) had hemispheric strokes. Stroke location was classified as follows: (1) subcortical, (2) cortical, (3) combined cortical plus subcortical, and (4) brain stem or cerebellar.

Fifteen patients (32%) had a history of previous stroke. Thirty-six (77%) had a history of smoking (mean pack-years, 43.1), but only 7 (15%) had a clinical history of chronic pulmonary disease. Other preexisting medical conditions included hypertension in 25 (53%), diabetes in 17 (36%), and prior myocardial infarction in 13 (28%). All patients had been living at home or with relatives before their stroke. Neurological examination by a board-certified neurologist was performed on all patients, and findings were recorded, including the presence or absence of dysarthria or aphasia. Severity of neurological deficit due to hemispheric stroke was classified in the manner of Reding and Potes[32] into pure motor hemiparesis; hemiparesis plus hemisensory loss; and hemiparesis, hemisensory loss, plus homonymous hemianopsia. Patients with brain stem strokes were grouped separately. A Mini-Mental State score[33] was obtained in patients without severe aphasia. A history of sleep habits was obtained from the patient and, when possible, the bed partner. This included a history of snoring (never, occasionally, usually, or always), apneic episodes during sleep, and inappropriate daytime sleepiness. Obesity was measured by calculating the body mass index, defined as weight (kilograms) divided by height (meters) squared.

All patients were evaluated for nocturnal Sa_{O_2} within 1 week of admission to the rehabilitation ward with the use of continuous computerized overnight oximetry (Ohmeda Biox 3700 oximeter with a Laser 128 computer). All hypnotics and nonessential sedating drugs were discontinued at least 3 days before oximetry testing. Antihypertensive medications were not stopped, and seven patients continued to take antidepressants. Computerized (PROFOX) analysis of oximetry data was performed with every 2-second real-time data sampling of Sa_{O_2}, which provides a time resolution fine enough to reveal stereotypic patterns of desaturation associated with SDB. A simultaneous strip chart recording (appropriately calibrated) was also done to help ensure accuracy. Oximetry recordings were edited to eliminate data that appeared to be artifactual (eg, due to probe dislodgment) and might mimic desaturations due to SDB. The computerized analysis defines a desaturation event as a ≥4% change in Sa_{O_2} from baseline. A rise of 4% above the nadir of a desaturation event signals the end of that event. Mean overnight Sa_{O_2}, lowest Sa_{O_2}, total number of desaturation events, and percentage of recording time with Sa_{O_2} <90% were recorded. DI was calculated by dividing the number of desaturation events by the total recording time. Oximetry strip charts were checked frequently during recording by certified sleep laboratory technicians for technical accuracy, and nursing staff observed patients periodically during the night of recording and recorded snoring, apneic episodes, and sleep arousals. Therapists and nurses were questioned weekly regarding the presence of excessive daytime sleepiness.

Selected patients underwent standard overnight PSG (Grass model 78D 12-channel polygraph, scored manually according to established criteria). PSG was performed in an accredited sleep laboratory within 1 week of oximetry and interpreted by a certified sleep laboratory physician. Oximetry criteria for performing PSG were arbitrarily defined and included one or more of the following: (1) any desaturation to a level ≤80%, (2) ≥5% of monitoring time with Sa_{O_2} <90%, and (3) presence of short-duration, repetitive desaturations. Twenty-four patients met these criteria, but three subjects refused PSG, and the sleep laboratory was not available to perform the study within 1 week of oximetry in two additional patients.

Functional abilities were assessed by the BI, a widely accepted multifaceted scale that measures mobility and activities of daily living.[34] BI was scored for all patients on admission and discharge from the rehabilitation unit (median stay, 30 days; range, 8 to 63 days) and by telephone interview at 3 months (±1 week) and 12 months (±1 month) after stroke onset. All personnel scoring BI were blinded to the results of sleep studies. No patients were lost to follow-up.

Primary outcome variables were as follows: (1) ability to return home at discharge, (2) ability to live at home at 3 and 12 months after stroke, (3) BI at discharge, (4) BI at 3 and 12 months after stroke, and (5) death from any cause at 12 months after stroke.

Secondary analysis included assessing the association of OxyHb desaturation with other variables, including age, aspects of medical history, prior stroke, size and location of stroke, and severity of neurological deficit.

A subgroup of 15 patients with the most frequent desaturations (DI >10/h) was also analyzed separately (DI group).

Results were analyzed by χ^2 test, Student's t test, and Pearson correlations. Pearson correlations were chosen for the primary data analysis because of our interest in the relationship between variables. Since the correlation of a continuous variable with a dichotomous variable is a special case of the Pearson correlation, it was appropriate for this study. Proba-

TABLE 1. Mean Overnight Oximetry Values (n=47)

	Mean±SD	Range
Total recording time, min	413±35	309-472
Mean Sao_2, %	94.0±1.7	86.0-96.2
Lowest Sao_2, %	74.8±12.5	40.0-90.0
Time at <90% Sao_2, %	4.3±5.7	0-28.1
Time at <80% Sao_2, %	1.0±3.4	0-21.7
Total No. of desaturation events (≥4% desaturation)	65.0*±66.3	5.0-295.0
Mean low Sao_2 for desaturation events, %	88.5±3.2	71.6-92.1
DI	9.5±9.6	0.7-43.6

*Nine patients had >100 desaturation events.

bility values were used to describe the strength of relationships, and there was no adjustment for multiple comparisons.

Results

Results of overnight oximetry appear in Table 1. The mean overnight Sao_2 was 94.0±1.7%. There was a wide variation in the lowest recorded Sao_2, as shown in Fig 1. Twenty-six of 47 subjects (55%) had at least one desaturation event <80%, and 6 of 47 (13%) had at least one event <60%. The mean number of desaturation events was 65.0±66.3, and this also exhibited a wide variation. Nine of 47 patients (19%) had >100 desaturation events on the night of recording. The number of desaturation events per hour of recording time (DI) was 9.5±9.6. The distribution of DI values for all patients is shown in Fig 2. Fifteen of 47 patients (32%) had >10 events per hour, and 6 of 47 (13%) had >20 events per hour.

Mean admission BI was 32.7±13.3 and mean discharge BI 58.3±20.6. At 3 months after stroke onset, mean BI for 44 surviving patients was 70.7±22.8; at 12 months BI was 73.9±23.6 for 42 surviving patients (Table 2).

Correlation between functional status, as measured by BI, and oximetry measures of SDB was assessed with the use of Pearson correlation coefficients (Table 3). Although admission BI was correlated with time at <90% Sao_2, there was no correlation between admission BI and any other oximetry variable. In contrast, there was a strong correlation between multiple measures of SDB (including mean Sao_2, time at <90% Sao_2, total number

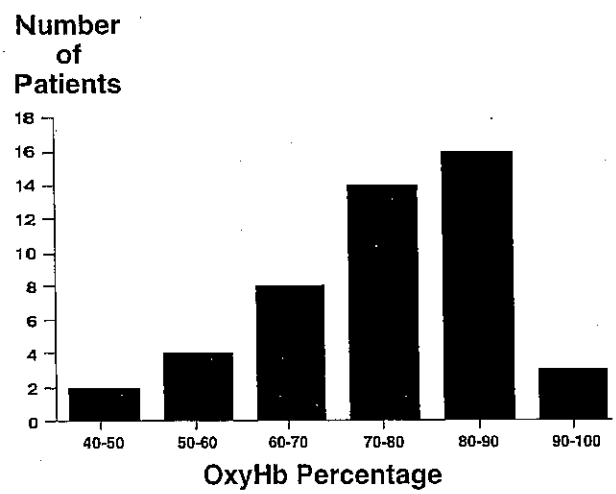

FIG 1. Distribution of lowest OxyHb values for each patient during overnight oximetry.

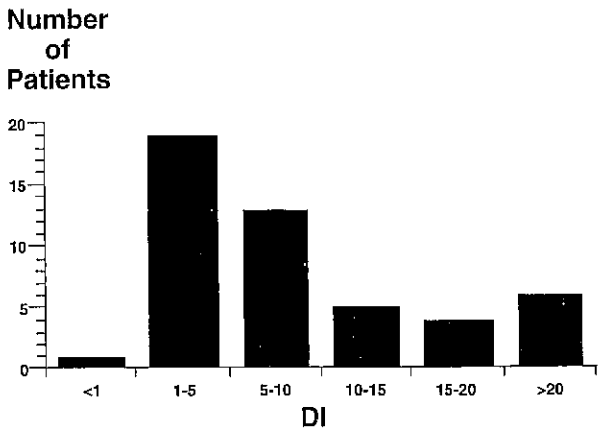

FIG 2. Distribution of DI calculated for each patient during overnight oximetry. DI is defined as the number of desaturation events (>4% OxyHb desaturation) per hour of recording time.

of desaturation events, and DI) and BI at discharge and improvement in BI between admission and discharge. The strong correlation between measures of SDB and BI persisted at 3 and 12 months after stroke. The variable that correlated least with functional status was the lowest recorded Sao_2.

At discharge, 35 of 47 patients (74%) had returned home or lived with relatives. At 3 months after stroke, 34 of 44 surviving patients (77%) were living at home, and at 12 months 33 of 42 patients (79%) were at home (Table 2). There was a correlation (Pearson coefficient) with return home at discharge and four oximetry measures: number of desaturation events (r=.29, P=.05), mean Sao_2 (r=−.35, P=.02), time spent at <90% Sao_2 (r=.42, P=.003), and DI (r=.29, P=.05). By 3 months, mean Sao_2 (r=−.31, P=.03) and percentage of time spent at <90% Sao_2 (r=.42, P=.003) correlated with ability to live at home, but by 12 months after stroke, no oximetry measure obtained after stroke correlated with continued residence at home.

Three of 47 patients died within 3 months after stroke, and a total of 5 patients were dead at the 12-month follow-up (Table 2). Death by 1 year correlated with mean Sao_2 (r=.37, P=.01) and percentage of time spent at <90% Sao_2 (r=−.41, P=.004).

PSG was performed on 19 patients. AHI, defined as the average number of episodes of apneas and hypopneas per hour of recording, was calculated for all patients. All but 1 patient studied with PSG had an AHI

TABLE 2. Outcome Measures for All Patients

BI	
Admission BI (n=47)	32.7±13.3
Discharge BI (n=47)	58.3±20.6
Change in BI (admission to discharge) (n=47)	25.6±12.6
BI at 3 mo (n=44)	70.7±22.8
BI at 12 mo (n=42)	73.9±23.6
Ability to live at home	
At discharge (n=47)	35 (74.5%)
At 3 mo (n=44)	34 (77.3%)
At 12 mo (n=42)	33 (78.6%)
Death (any cause)	
Total at 3 mo	3 (6.4%)
Total at 12 mo	5 (10.6%)

BI values are mean±SD.

TABLE 3. Correlation of Oximetry and BI (All Patients)

	Mean Sao_2	Time at <90% Sao_2	No. of Desaturation Events	Lowest Sao_2	DI
Admission BI (n=47)	r=.28	r=−.31	r=−.18	r=.18	r=−.18
	P=.06	P=.03*	P=.23	P=.22	P=.24
Discharge BI (n=47)	r=.45	r=−.55	r=−.36	r=.29	r=−.35
	P=.002*	P=.0001*	P=.01*	P=.05*	P=.02*
Change in BI (admission to discharge) (n=47)	r=.43	r=−.57	r=−.40	r=.28	r=−.38
	P=.002*	P=.0001*	P=.006*	P=.05*	P=.008*
BI at 3 mo (n=44)	r=.48	r=−.51	r=−.39	r=.10	r=−.37
	P=.0009*	P=.0004*	P=.009*	P=.50	P=.01*
BI at 12 mo (n=42)	r=.44	r=−.38	r=−.36	r=.15	r=−.35
	P=.003*	P=.02*	P=.02*	P=.34	P=.02*

*$P \leq .05$.

>10, 13 of 19 (68%) had an AHI >20, and 10 of 19 (53%) had an AHI >30. The AHI obtained during PSG was greater than the DI obtained for the same patients during oximetry in 18 of 19 cases. The mean AHI during PSG was 35.6±23.2 compared with a mean DI of 17.7±10.5 for the same group of patients during oximetry. Desaturation events on PSG were largely obstructive in nature, but the proportion of obstructive versus central events differed between hemispheric and brain stem stroke. Of total apneas, 93.3% were obstructive in the 16 patients with hemispheric stroke, but for the 3 patients with brain stem stroke, only 58.3% of the apneas were obstructive, the rest being central. All 3 patients with brain stem stroke had severe SDB, with a mean AHI on PSG of 57.0.

Functional status in those patients with the strongest oximetry evidence for SDB (DI group) is shown in Fig 3. Admission BI for the 15 patients constituting the DI group was 30.3±16.7, discharge BI was 49.3±24.1, BI at 3 months was 56.4±23.6, and BI at 12 months was 61.2±23.0. BI values measured at the same times for the other patients were 33.8±11.5, 62.5±17.6, 77.3±19.5, and 79.7±21.9, respectively. Although there was no statistically significant difference between the admission BI for the DI group and that for the other patients, by discharge, the mean BI for the DI group was significantly lower than that for the other patients (P<.04, Student's t test). Patients in the DI group continued to function worse at 3 and 12 months after stroke compared with the other patients (P<.004 and P<.02, respectively).

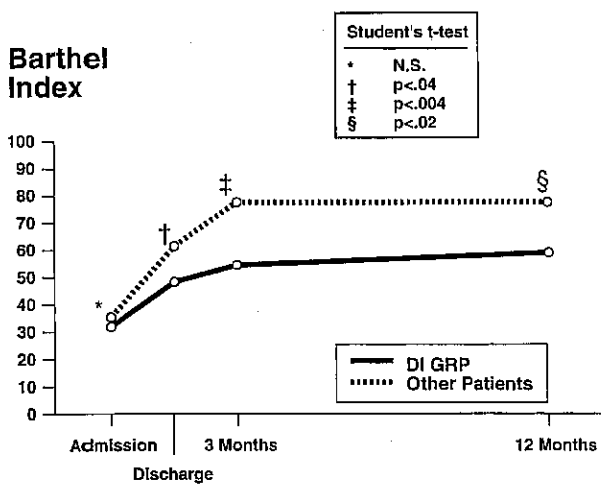

FIG 3. BI scores at admission for rehabilitation, discharge, and 3- and 12-month follow-ups for patients with DI >10 (DI GRP) compared with other patients.

Brain stem location of stroke correlated with DI calculated during oximetry (r=.49, P=.0005) and number of desaturation events (r=.50, P=.0004) but not with any other oximetry measures. Because all 3 patients with brain stem stroke had PSG that demonstrated significant SDB, the data were also analyzed after these patients were excluded (Table 4). Although the correlation between oximetry measures and lower BI is not as great for patients with hemispheric stroke alone, the association is still statistically significant for mean Sao_2 and percentage of time spent at <90% Sao_2. BI at all time intervals after stroke was not significantly different between patients with brain stem and hemispheric strokes.

For hemispheric strokes, there was no correlation between oximetry measures and location of stroke (cortical, subcortical, or combined cortical and subcortical). No association was found between oximetry measures and degree of neurological deficit (pure motor hemiparesis versus hemiparesis plus hemisensory loss versus hemiparesis plus hemisensory loss plus homonymous hemianopsia). The Mini-Mental State score correlated with the lowest Sao_2 (r=.40, P=.04) and percentage of time with Sao_2 <90% (r=−.44, P=.02). No association was found between oximetry variables and presence of dysarthria or aphasia.

Oximetry values did not correlate with sex, age, or body mass index. A history of stroke correlated with mean Sao_2 (r=.32, P=.03) but no other oximetry variable. No correlation was found between oximetry values and history of any other preexisting medical condition, including prior myocardial infarction and pulmonary disease.

An adequate history of prior sleep habits, usually from the spouse, was obtained in all but 3 patients. Of these, 23 of 44 (52%) were said to usually or always snore, and 10 of 44 (23%) had apneic episodes during sleep. However, a clear history of inappropriate daytime sleepiness was obtained in only 5 patients. By Pearson correlations, a prior history of snoring correlated with the lowest recorded Sao_2 (r=−.45, P=.001) and assignment to the DI group (r=−.35, P=.02). A history of previous apneic spells correlated with DI (r=−.45, P=.002) and the number of desaturation events (r=−.46, P=.002). A history of previous inappropriate

TABLE 4. Correlation of Oximetry With BI (Hemispheric Strokes Only)

	Mean Sao_2	Time at <90% Sao_2	No. of Desaturation Events	Lowest Sao_2	DI
Admission BI (n=44)	r=.28	r=−.29	r=−.08	r=.13	r=−.10
	P=.06	P=.06	P=.62	P=.39	P=.52
Discharge BI (n=44)	r=.44	r=−.53	r=−.24	r=.26	r=−.24
	P=.003*	P=.0003*	P=.12	P=.09	P=.11
Change in BI (admission to discharge) (n=44)	r=.41	r=−.54	r=−.30	r=.27	r=−.29
	P=.005*	P=.0002*	P=.05*	P=.07	P=.06
BI at 3 mo (n=41)	r=.48	r=−.48	r=−.30	r=.05	r=−.30
	P=.001*	P=.002*	P=.06	P=.76	P=.06
BI at 12 mo (n=39)	r=.43	r=−.31	r=−.26	r=.13	r=−.26
	P=.006*	P=.06	P=.11	P=.44	P=.11

*$P \leq .05$.

daytime sleepiness did not correlate with any oximetry variable.

Nursing observations of snoring and apnea during oximetry testing showed less correlation with oximetry results than history from a sleep partner. Nursing staff observed snoring in 25 of 47 patients (53%) during the night of oximetry testing. Nursing observations of snoring did correlate with the number of desaturation events (r=.41, P=.004) and DI (r=.40, P=.005). Apneic episodes were observed by nurses in 10 of 47 patients (21%) but did not correlate with oximetry measures. There was no correlation between oximetry results and weekly reports by nurses or therapists regarding excessive daytime sleepiness, which occurred in only 5 patients.

Discussion

This study confirms our clinical observation that snoring and SDB accompanied by OxyHb desaturation are common in patients undergoing stroke rehabilitation. Several studies have documented a higher prevalence of prior snoring in stroke patients compared with control subjects and have attributed an increased stroke risk to the snoring.[19-23] However, only a few other studies used objective measures of SDB in patients with recent stroke,[29-31] with results similar to ours. In a study of 47 patients published in abstract form,[29] 72% had an AHI of ≥10 events per hour, 53% had an AHI >20, and 30% had an AHI >40. In another study (published in abstract form), PSG evaluations performed in 20 consecutive patients with acute stroke revealed that 14 (70%) had significant sleep apnea.[30] Mohsenin and Valor[31] performed PSG on 10 stroke patients admitted to a rehabilitation unit and also on 10 control subjects matched for age, body mass index, presence of hypertension, and smoking history. The AHI in stroke patients was 52±10 events per hour compared with 3±1 events per hour in the control group. The majority of the events were obstructive apneas associated with Sao_2 desaturation and arousal.

Our study extends previous work by demonstrating that SDB after stroke is associated with higher mortality at 1 year and poor functional outcome in survivors. Only one measure of nighttime OxyHb desaturation was associated with functional abilities on admission to rehabilitation. In contrast, several oximetry variables correlated strongly with lower BI scores at the time of discharge from rehabilitation, and this association remained strong at 3- and 12-month follow-ups. The association between measures of SDB and worse functional outcome was even more dramatic for a subgroup of the study population who had >10 episodes of OxyHb desaturation per hour of recording (DI group). BI on admission for the DI group was no different than that of the other patients, but by discharge, BI was significantly lower for the DI group, a difference that remained significant at 3 and 12 months. Several oximetry measures were also associated with inability to return home after discharge from the rehabilitation program. However, no measure was associated with ability to live at home at 12 months, which is consistent with the frequent observation that ability to live at home depends on many factors, including social support systems.

Vascular mortality is higher in patients with untreated OSA,[35,36] which was the most common type of SDB among those patients who received PSG in our study. Spriggs et al[20] discovered a definite relationship between mortality at 6 months after stroke and severity of snoring. In our study we found a correlation between death at 1 year and two oximetry measures, which is consistent with earlier studies.

Numerous comparisons were made in this study, and there is potential for observing values of $P<.05$ by chance alone. However, given the number of oximetry measures that correlated with outcome measures with values of $P<.05$, this seems unlikely.

The reason for the association between OxyHb desaturation and worse functional outcome is unclear. Since outcome after stroke is multifactorial, we considered the possibility that SDB was a marker for other variables. However, we found no association between oximetry measures and sex, age, location of hemispheric stroke, cognitive impairment, the degree of neurological impairment, history of chronic pulmonary disease, or history of any other preexisting medical condition. We did find an association between one oximetry measure and history of prior stroke, but this is insufficient to establish a causative relationship. It is possible that the small number of patients studied may have been insufficient to demonstrate an association with another important outcome variable or that variables were overlooked. Although oximetry abnormalities were correlated with brain stem stroke, the association between SDB and worse functional outcome was still significant for hemispheric stroke patients.

One possible explanation for the association of measures of SDB and poor outcome is that patients with

SDB develop subtle deficits of attention and concentration that impair their abilities to perform activities of daily living and to acquire new skills. We could not demonstrate overt excessive daytime sleepiness in our study population, but the measure used was rather imprecise and consisted of questioning nursing and therapy staff weekly regarding whether any inappropriate daytime sleepiness had been observed. We did not perform more detailed testing of attention and concentration. We did find a weak association with two oximetry readings and Mini-Mental State score. However, we cannot conclude that there was a causal relationship. Another possibility is that changes in arterial oxygen and carbon dioxide saturations that accompany SDB result in local alternations in cerebral blood flow, which in turn inhibit the process of neuronal reorganization. There is ample evidence of decreased cerebral blood flow in patients with OSA during wakefulness as well as sleep.[8,37-39] Impaired cerebral autoregulation may also accompany OSA.[13] Recurrent hypoxemia associated with SDB might be detrimental to cerebral recovery after stroke. At this time, the reason for the association of SDB and worse functional outcome remains speculative. Treatment trials of patients exhibiting SDB after stroke would help determine whether SDB is truly an independent cause of worse outcome and might shed light on the underlying mechanism of this effect.

Computerized overnight oximetry was used to screen for SDB in this study because it is a convenient, noninvasive procedure that can be performed easily on elderly stroke patients on a rehabilitation ward. It is a reliable measure of Sao_2 when Sao_2 is >70%.[40] A limitation of oximetry is that it fails to measure respiratory events that cause sleep fragmentation but not OxyHb desaturation.[41] Therefore, PSG remains the standard for the diagnosis of SDB.[1,41] However, oximetry alone can be of significant diagnostic value, with sensitivity and specificity values dependent on the type of data analysis used.[41-44] Studies comparing the two procedures indicate that oximetry is sensitive for detecting more severe cases of SDB but not mild cases.[41,45] For those with a high pretest probability of disease, finding ≥15 desaturations of ≥4% per hour of monitoring is sufficient to confirm the diagnosis of sleep apnea with a specificity of 98%.[41]

The lack of objective information regarding the duration of sleep is regarded by some as a major limitation of oximetry in screening patients for sleep apnea syndrome. However, Douglas and coworkers[45] found that the AHI is abnormal in most patients in whom sleep apnea is diagnosed, whether the index is calculated per hour of sleep or per hour in bed. Farney et al[46] have shown that a reasonable estimate of the duration of sleep can be made and rapid eye movement and non–rapid eye movement sleep differentiated based on analysis of the oximetry tracing alone.

Only 19 of our patients had PSG. Although this was performed based on predetermined criteria, it was not performed in 5 patients because of refusal or unavailability of the sleep laboratory. Since our primary goal was to identify a simple way to screen stroke patients for SDB, PSG was done primarily to validate the use of oximetry to identify SDB. Nevertheless, the results are instructive. Although there is no absolute PSG cutoff point for the diagnosis of SDB,[1] we chose AHI >10 as a marker for SDB. All but one patient studied with PSG had an AHI >10, 13 of 19 (68%) had an AHI >20, and 10 of 19 (53%) had an AHI >30. These figures are similar to those reported by Kapen and coworkers.[29] We also calculated a DI based on the number of OxyHb desaturation events >4% from baseline per hour of oximetry recording for all patients. We presume that most or all of these desaturation events were due to apneas or hypopneas. Other evidence to explain the desaturations (advanced heart/lung disease, morbid obesity) were not present, and oximetry recordings were edited to eliminate artifacts (eg, due to probe dislodgment) that might mimic desaturations due to SDB. The DI during oximetry was lower than the AHI obtained during PSG for 18 of the 19 patients who received both procedures. The mean AHI obtained during PSG for the 19 patients was a striking 35.6 events per hour compared with 17.7 events per hour for the same patients during oximetry. This discrepancy is expected, since apneas and hypopneas with either a >4% desaturation in Sao_2 or a sleep arousal were scored on PSG, but only Sao_2 desaturations were measured on oximetry. Nevertheless, this difference suggests that oximetry underestimated the severity of SDB in our patient population and that the scope of the problem is much greater than our oximetry data indicate.

The most common form of SDB in this study appears to have been OSA. In the patients undergoing overnight PSG, the vast majority of apneas and hypopneas recorded were obstructive in nature. Although a history of snoring does not always predict OSA documented by objective studies, a history of heavy habitual snoring provided by a sleep partner does appear to be a valid marker for OSA.[17] There is suggestive evidence that SDB, particularly OSA, preceded stroke in our patients. Fifty-two percent of our patients in whom an adequate sleep history could be obtained were said to usually or always snore before the stroke, and in almost one quarter a history of apneic episodes during sleep was obtained from the bed partner. Since no age- or sex-matched control subjects were available, this information must be interpreted cautiously. However, our results are comparable to those in the study of Spriggs and coworkers,[20] who obtained a history of habitual snoring in 57.7% of 400 stroke victims compared with 30.1% of age- and sex-matched control subjects. In a case-controlled study in which 133 patients with ischemic stroke were compared with control subjects matched for age and sex, Neau et al[22] obtained a history of habitual snoring in 23.3% of stroke patients and only 8.3% of control subjects. In the present study, a prior history of habitual snoring and nocturnal apneic episodes correlated with evidence of OxyHb desaturation during oximetry, also suggesting that OSA was a preexisting problem.

Although we believe that SDB in most patients in our study preceded the stroke, it seems possible that the stroke caused SDB in some patients. CSA is well described in association with brain stem stroke.[17,28] Brain stem stroke was clearly associated with oximetry measures of SDB in our study. Although only three patients had brain stem stroke, all had significant SDB by both oximetry and PSG measures. All had mixed apnea patterns, but the relative proportion of central apneas was higher in these three patients than in 15 of the 16 patients with hemispheric stroke who had PSG. This

observation supports the hypothesis that central regulation of respiration is selectively impaired in patients with brain stem stroke.

Patients with hemispheric strokes might also develop SDB after the stroke. CSR may occur after stroke,[8] resulting in a clinical presentation virtually indistinguishable from "traditional" sleep apnea syndromes, including major OxyHb desaturation and sleep fragmentation.[9,47] In addition, dysphagia due to impairment of pharyngeal mobility occurs in a high percentage of unilateral hemispheric strokes and is often unsuspected clinically.[24-27] Since the primary mechanism of OSA is functional obstruction of the oral airway, it seems possible that the risk of developing OSA might be higher in stroke patients with pharyngeal muscle dysfunction.[31]

No age-matched control patients were included in this study, and since the prevalence of SDB increases with age, the results should be interpreted cautiously. However, the frequency of oximetry abnormalities consistent with SDB is quite dramatic, and the association with increased mortality at 1 year and poor long-term functional outcome in survivors warrants further investigation. Although the increased mortality in our patients is consistent with earlier studies, the association with worse functional outcome has not been reported previously. Whether SDB is truly an independent variable associated with worse functional abilities cannot be answered at present.

Obviously, a major question resulting from this study is whether treatment trials using continuous positive airway pressure for stroke patients with SDB might prevent the worse outcome seen in SDB patients. This study did not address treatment, although we did counsel patients regarding weight loss and use of sedative drugs and notified primary care physicians about the results. Only one patient was subsequently treated with nasal continuous positive airway pressure. This patient refused further treatment after a short time. Our own anecdotal experience suggests that continuous positive airway pressure is not well tolerated by elderly patients with recent stroke. In this sense, they are very different than patients presenting with excessive daytime sleepiness or other overt clinical manifestations of SDB. Despite ample evidence of the deleterious effects of SDB, our observation has been that most stroke patients, their physicians, and their families do not consider SDB a problem significant enough to warrant what they perceive as the nuisance of treatment. However, the results of this study strongly indicate that SDB associated with stroke should be studied further and that trials of treatment for SDB associated with stroke should be undertaken.

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