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Sleep apnea in patients with transient ischemic attack and stroke:

A prospective study of 59 patients

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Article abstract—Although sleep apnea (SA) appears to be a cardiovascular risk factor, little is known about its frequency in patients with transient ischemic attack (TIA) and stroke. We prospectively studied 59 subjects (26 women and 33 men; mean age, 62 years) with stroke (n = 36) or TIA (n = 23) with the use of a standard protocol that included assessment of snoring and daytime sleepiness (Epworth Sleepiness Score [ESS]), a validated SA score (Sleep Disorders Questionnaire [SDQ-SA]), and a severity of stroke score (Scandinavian Stroke Scale [SSS]). SA was considered clinically probable (P-SA) when habitual snoring was associated with an ESS of >10 or when SDQ-SA score was ≥32 in women and ≥36 in men. Polysomnography (PSG) was obtained in 36 subjects (group 1) a mean of 12 days after TIA or stroke. In 23 subjects (group 2), PSG was not available (n = 11), refused (n = 10), or inadequate (n = 2). Clinical and PSG data were compared with those obtained in 19 age- and gender-matched control subjects. Groups 1 and 2 were similar in mean age (61 versus 64 years), type of event (36% versus 44% TIA), reported habitual snoring (58% versus 52%), and P-SA (58% versus 50%). PSG showed SA (Apnea-Hypopnea Index [AHI], ≥10) in 25 of 36 subjects (69%). The proportion of subjects with SA was similar in the TIA and stroke groups (69% versus 70%) and was well above the frequency found in our control group (15%). An AHI of ≥20 and a minimal oxygen saturation of <85% were each found in 20 of 36 subjects (55%). Gender and age did not correlate with severity of SA. Subjects with habitual snoring, P-SA, or severe stroke (SSS of <30) had a significantly higher AHI ($p < 0.05$). The sensitivity of P-SA for SA was 64%, and the specificity was 67%. We conclude that SA has a high frequency in patients in the acute phase of TIA and stroke and SA cannot be predicted reliably on clinical grounds alone but is more likely in patients with habitual snoring, abnormal SDQ-SA, or severe stroke.

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Several observations suggest a link between snoring that is always, or almost always, present (habitual snoring); sleep apnea (SA); and cardiovascular diseases. First, patients with snoring or SA often have cardiovascular risk factors such as obesity, hypertension, smoking, or alcohol abuse.[1-3] Second, SA is present in as many as 35 to 50% of patients with disorders such as stable congestive heart failure (CHF),[4] myocardial infarction,[5] and coronary heart disease (CHD).[6] Third, the high morbidity and mortality of patients with SA appear to be related to vascular diseases.[5,7-9] Fourth, treatment of SA can improve cardiac function and blood pressure control[10,11] and reduce overall cardiovascular morbidity and mortality.[7-9]

There also is evidence of an association between stroke and habitual snoring. In a cohort study of 4,388 male patients, the relative risk for stroke and ischemic heart disease combined in those with habitual snoring was 2.4 and remained essentially unchanged after adjustment for age, body mass index (BMI), hypertension, smoking, and alcohol consumption.[12] In a case-control study, habitual snoring was an independent risk factor for stroke, with an odds ratio of 2.1:1. The relative risk increased to 8.0 in those with snoring combined with a history of nocturnal apneic events, obesity, and excessive daytime sleepiness.[13] Furthermore, snoring is associated with stroke occurring during sleep or within the first 30 minutes after awakening[14] and may have a negative effect on recovery from stroke.[15] Also, a few reports suggest that the frequency of SA may be high in both infratentorial[16] and supratentorial[17] strokes. Finally, in a recent study, SA was present in 8 of 10 patients admitted to a rehabilitation unit within the first year after hemispheric stroke.[18]

Despite these epidemiologic data, the existence of a link between snoring and cerebrovascular diseases remains a matter of controversy,[14,19] and the frequency of SA in patients with acute cerebrovascular diseases is essentially unknown.

We therefore conducted a prospective study to determine the frequency of habitual snoring and symptoms suggestive of SA and polysomnographically documented SA in patients with transient ischemic attack (TIA) and stroke.

Patients and methods. During a 16-week period (June through October 1995), all patients between the ages of 18

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and 80 years admitted to the University of Michigan Hospitals with acute TIA or ischemic stroke and not in coma or in an unstable medical condition were considered for inclusion in the study. The study protocol was approved by the institutional review board of the University of Michigan. Patients were examined with a protocol that included a questionnaire, clinical assessment, and standard investigations for cerebrovascular diseases. Among 59 eligible subjects, 38 (64%) gave informed consent for overnight polysomnography (PSG), 10 refused PSG, and 11 could not be studied in a timely manner.

Clinical stroke assessment. Patients were assessed clinically by one of the authors, usually within the first 2 days of hospitalization. Cardiovascular risk factors were recorded, including family history of stroke, hypertension (defined as blood pressure of >160/90 mm Hg on at least two occasions before the TIA or stroke), diabetes (defined as fasting glucose level of >6.0 mmol/L known to exist before the TIA or stroke), hypercholesterolemia (fasting blood cholesterol of >6.5 mmol/L), smoking (number of pack-years), and alcohol consumption (drinks per week). Any history of CHF or CHD was also noted. The maximal severity of stroke was estimated with the use of the Scandinavian Stroke Scale (SSS).[20] The total SSS score ranges from 0 (maximal deficit) to 58 (no deficit). A score of <30 represents severe stroke, and a score of >30 represents mild-to-moderate stroke.[21] BMI was calculated as weight (in kg)/height (in meters).[2]

Clinical sleep assessment. A detailed history of sleep-wake habits and symptoms preceding the TIA or stroke was obtained and included questions about snoring and daytime sleepiness. Snoring was considered to be habitual when it was reported to occur often or always. Daytime sleepiness was estimated with the Epworth Sleepiness Scale (ESS)[22] and considered excessive when the ESS score was >10. The Sleep Apnea subset of questions from the Sleep Disorders Questionnaire (SDQ-SA) was also administered; a SDQ-SA score of ≥36 for men and of ≥32 for women has a sensitivity and a specificity of about 80% for polysomnographically proven SA.[23] When a reliable history could not be obtained from a subject because of aphasia or confusion, information was obtained from relatives. SA was considered to be clinically probable (P-SA) when there was habitual snoring and an ESS score of >10 or when the SDQ-SA was ≥36 for men or ≥32 for women ("abnormal" SDQ-SA).

Stroke studies. Systematic investigations included standard blood tests; 12-lead ECG; chest X-ray; Doppler ultrasonography; and brain CT, brain MRI, or both. Brain images were reviewed with a standard protocol of evaluation by a neuroradiologist (D.Q.) who was blinded to the clinical context. Cerebral angiography, echocardiography, 24-hour Holter monitoring, and specific blood tests were performed in selected cases. Etiology of stroke was determined according to the criteria of the Trial of Acute Stroke Treatment study (TOAST) as large artery disease, small artery disease, cardioembolism, other, or undetermined.[24]

Polysomnography. PSG was performed between 10 PM and 6 AM at the patient's bed in either the intensive care unit or a ward (n = 33) or in the sleep laboratory (n = 5) with a 16-channel paper recording system at a paper speed of 10 mm/sec. We recorded eight EEG channels, two electro-oculogram (EOG) channels, one chin-EMG channel, nasooral flow (thermistor), chest and abdominal wall excursion, heart rate, oxygen saturation (Sao_2), and two tibialis anterior EMG channels. Only PSGs with ≥4 hours of sleep were considered adequate. Sleep stage scoring was done visually according to standard criteria.[25] An apnea was defined as >80% reduction in nasooral airflow lasting ≥10 seconds. A hypopnea was scored when there was an obvious reduction (usually >20%) in airflow and/or effort, lasting ≥10 seconds and accompanied by an arousal or a fall in Sao_2 of ≥4%. Central apneas were identified by the absence of respiratory effort during cessation of airflow. The numbers of apneas and of apneas plus hypopneas per hour of sleep were expressed as the Apnea Index (AI) and Apnea-Hypopnea Index (AHI), respectively. SA was considered present when the AHI was ≥10. We also noted the maximal and average durations of respiratory events, minimal Sao_2, and number of oxygen desaturations to <85%. The ratio of central apneas to all respiratory events was calculated. Cheyne-Stokes respiration (CSR) was defined as periodic breathing with central apneas or hypopneas alternating with hyperpnea in a crescendo-decrescendo pattern over ≥10% of sleep time.

Control group. The control subjects were 19 healthy volunteers (11 men and 8 women) with a mean ± SD age of 63 ± 9 years (range, 49 to 75 years) recruited through the University of Michigan Alcohol Research Center. Control subjects were free by history and clinical examination of diabetes, uncontrolled hypertension, heart disease, chronic obstructive pulmonary disease, TIA, stroke, dementia, parkinsonism, seizures, schizophrenia, and depression. All control subjects had limited alcohol consumption (Audit score of <8).[26] Subjects with a history of loud snoring accompanied by witnessed apneas were also excluded.

Statistical analysis. To compare values between groups (studied versus nonstudied, TIA versus stroke, patients versus control subjects), we used the chi-square test for nominal variables, the Mann-Whitney U test for ordinal variables, and the unpaired Student's t test for continuous variables. Statistical significance was set at $p < 0.05$.

Results. The group of 59 patients included 33 men and 26 women with a mean age of 62 years (range, 34 to 80 years). There were 23 subjects with TIA and 36 patients with stroke. More than 50% of all events occurred between 5:30 AM and noon. The mean SSS score for stroke patients was 39. Stroke involved the anterior (carotid) territory in 26 subjects and the posterior (vertebrobasilar) territory in 11 subjects. Seven subjects who showed old nonlacunar strokes on neuroimages were included in the stroke group.

The 36 subjects who had PSG and the 23 who did not have PSG had similar mean age, gender, type and topography of cerebrovascular events, BMI, cerebrovascular risk factors, sleep history, and stroke severity (table 1). Habitual snoring and abnormal SDQ-SA scores occurred in about 50% in both groups, whereas an ESS score of >10 was somewhat more common in studied subjects (35% versus 19%, p = NS). Subjects who were studied were more likely to have had cardioembolic stroke, probably in part because the longer hospitalization in such patients made it more likely that the sleep laboratory would be available for the sleep study.

PSG was performed in 38 subjects and considered to be adequate in 36 of the 38. Recordings occurred within the

first 10 days from TIA or stroke in 26 subjects (72%), with a mean time of 12 days (range, 1 to 71 days). Sleep apnea (AHI of ≥10) was found in 69% of patients (versus 15% in normal subjects) and was considered to be clinically significant (AHI of ≥20 or minimal Sao_2 of <85%) in 55% of subjects. Respiratory events consisted mainly of obstructive or mixed apneas. Central apneas constituted about 10% (range, 0 to 70%) of all respiratory events. In 4 subjects, however, the proportion of central apneas was ≥30%. Cheyne-Stokes breathing occurred in 37% of studied patients, usually in association with obstructive events, and was significantly associated with a positive history of CHF ($p < 0.05$). Only 1 subject, who had a TIA after cardiac transplantation, presented with prominent and isolated CSR. Two of the 3 patients who had CSR for >50% of the sleep time had severe stroke (SSS score of <30) and had no history or clinical signs of CHF. In a few patients, we observed the combination of CSR in non-REM sleep and obstructive SA in REM sleep. In these cases, the differentiation between CSR and periodic hypopneas related to obstructive events often was difficult.

Control subjects and subjects with TIA or stroke were similar in age, BMI, and gender but differed in the prevalence of habitual snoring (58% versus 12%), in AHI (27 versus 6), and in minimal Sao_2 (82% versus 89%; table 2A). Among subjects with TIA or stroke, AHI was similar in men and women, in subjects above and below the age of 50, and in subjects with and without excessive daytime sleepiness. The AHI was significantly higher in subjects with habitual snoring, with P-SA, and with an SSS score of <30 (table 2B).

Studied subjects with TIA (n = 13) and with stroke (n = 23) were similar in age, gender, BMI, most cerebrovascular risk factors, sleep history, and PSG findings (table 3). SA was found in 69% of subjects with TIA and in 70% of those with stroke, but there was a trend toward higher AHI and AI in those with stroke. The proportion of central apneas, prevalence of CSR, and average and maximal durations of apneas were similar in the two groups.

The criteria chosen for the definition of P-SA had a sensitivity of 64% for an AHI of ≥10 and 74% for an AHI of ≥20 and a specificity for any AHI of ≥10 of 67%. Of the 25 subjects with an AHI of ≥10, 32% reported no habitual snoring, 58% did not complain of hypersomnia (ESS score of ≤10), and 36% had a normal SDQ-SA score.

Table 1 Clinical characteristics of studied and not studied patients with TIA/stroke

	Studied (n = 36)	Not studied (n = 23)	p
General characteristic			
Age (yr)	61 ± 11	64 ± 11	NS*
TIA/stroke (%)	36/64	44/56	NS
Males/females (%)	61/39	48/52	NS
Body mass index	27.4 ± 5	26.4 ± 5	NS
Cerebrovascular risk factor			
Hypertension (%)	58	70	NS
Diabetes (%)	31	30	NS
Hypercholesterolemia (%)	54	50	NS
Smoking (pack-yr)	27 ± 25	21 ± 26	NS
Alcohol (drinks/wk)	4 ± 8	4 ± 6	NS
Coronary heart disease (%)	40	17	NS
Congestive heart failure (%)	11	27	NS
Sleep history			
Habitual snoring (%)†	58	52	NS
Epworth Sleepiness Score >10 (%)‡	34	19	NS
Abnormal SDQ-SA Score (%)§	54	52	NS
Clinical probable sleep apnea (%)¶	58	50	NS
Acute cerebrovascular event			
TIA/stroke (%)	36/63	43/57	NS
Anterior/posterior distribution (%)	74/26	69/31	NS
Scandinavian Stroke Scale#	37 ± 15	39 ± 23	NS
Etiology of TIA/stroke (%)			
Large vessel disease	33	24	NS
Cardioembolism	19	0	<0.05
Other/unknown cause	48	76	<0.05

Values are expressed as mean ± SD or as percentage of patients presenting with the finding.

* Statistically nonsignificant difference ($p > 0.05$).
† Often or always.
‡ Minimal score, 0 (no sleepiness); abnormal score, >10; maximal score, 24.[22]
§ In men, ≥36; in women, ≥32.[23]
¶ Habitual snoring + Epworth Sleepiness Score >10 or abnormal SDQ-SA score.
# Calculated only for patients with stroke: minimal score, 0; maximal score, 58 (no deficits).[20]

SDQ-SA = Sleep Disorders Questionnaire–Sleep Apnea.

Discussion. This is the first detailed, prospective study of the frequency of SA in a consecutive and unselected series of patients with TIA or ischemic stroke. The clinical characteristics of subjects were comparable to those in larger series of patients with cerebrovascular events.[27,28] Our data therefore appear to be representative of patients with TIA or stroke who are admitted for acute care.

The main finding of this study was the observation of SA consisting mainly of obstructive or mixed respiratory events in 70% of the patients with TIA or stroke and of significant SA (an AHI of ≥20, minimal Sao_2 of <85%, or both) in 55%. These proportions are similar to the 80% of significant SA recently reported in 10 patients with hemispheric stroke admitted to a rehabilitation unit.[18] Similarly, in a study presented in abstract form, Kapen et al[17] reported an AHI of ≥10 in 72% and an AHI of >20 in 53% of 47 patients with acute stroke. However, in the first study,[18]

***Table 2** Sleep apnea in 36 patients with TIA or stroke*

	A: Clinical and polysomnographic findings in patients with TIA and stroke vs normal control subjects		
	TIA/stroke (n = 36)	Normal control subjects (n = 19)	*p*
Clinical characteristic			
Age (yr)	60 ± 11	63 ± 9	NS*
Males/females (%)	61/39	58/42	NS
Body mass index	27.4 ± 5	25.5 ± 4	NS
Habitual snoring (%)	58	12	<0.05
Polysomnographic finding			
Apnea-Hypopnea Index	27 ± 29	6 ± 7	<0.05
≥10 (%)	69	16	<0.05
≥20 (%)	55	5	<0.05
Apnea Index	12 ± 25	3 ± 5	NS
Minimal Sao_2	82 ± 9	89 ± 4	<0.05
Minimal Sao_2 <85% (%)	55	10	<0.05

	B: Relation between severity of sleep apnea and gender, age, reported snoring, Epworth Sleepiness Score, clinical suspicion of sleep apnea, and severity of stroke in patients with TIA/stroke	
	Apnea-Hypopnea Index	*p*
Men vs women	29 ± 29 vs 34 ± 30	NS
Age, >50 yr vs <50 yr	27 ± 40 vs 27 ± 27	NS
Habitual vs nonhabitual snoring	35 ± 34 vs 15 ± 11	<0.05
Epworth Sleepiness Score, >10 vs ≤10†	33 ± 30 vs 24 ± 29	NS
Clinical probable sleep apnea, yes vs no‡	35 ± 35 vs 15 ± 11	<0.05
SSS, <30 vs >30§	64 ± 32 vs 22 ± 16	<0.05

Values are expressed as mean ± SD or as a percentage of patients presenting with the finding.

* Statistically nonsignificant difference ($p > 0.05$).
† Minimal score, 0 (no sleepiness); abnormal score, >10; maximal score, 24.[22]
‡ Habitual snoring + Epworth Sleepiness Score >10, or abnormal SDQ-SA score.
§ Mild to moderate stroke, SSS >30; severe stroke, SSS <30.[21]

SDQ-SA = Sleep Disorders Questionnaire–Sleep Apnea; SSS = Scandinavian Stroke Scale.

***Table 3** Comparison of clinical and polysomnographic findings in 14 patients with TIA and 23 patients with stroke*

	TIA (n = 13)	Stroke (n = 23)	*p*
General characteristic			
Age (yr)	64 ± 10	59 ± 12	NS*
Males/females (%)	54/46	65/35	NS
Body mass index	26.8 ± 3	27.6 ± 6	NS
Cerebrovascular risk factor			
Hypertension (%)	69	52	NS
Diabetes (%)	20	39	NS
Hypercholesterolemia (%)	50	56	NS
Smoking (pack-yr)	17 ± 18	32 ± 26	NS
Alcohol (drinks/wk)	8 ± 10	3 ± 6	NS
Coronary heart disease (%)	33	43	NS
Congestive heart failure (%)	8	13	NS
Sleep history			
Habitual snoring (%)†	54	61	NS
Epworth Sleepiness Scale >10 (%)‡	33	35	NS
Abnormal SDQ-SA score (%)§	50	43	NS
Clinical probable sleep apnea (%)¶	54	61	NS
Polysomnographic finding			
Apnea-Hypopnea Index	18.6 ± 10	31.7 ± 35	NS
≥10 (%)	69	70	NS
≥20 (%)	62	48	NS
Apnea Index	4.5 ± 5	16 ± 30	NS
Percent of central to total apneas	9 ± 12	9 ± 19	NS
Cheyne-Stokes respiration (%)#	33	39	NS
Duration of apneas (aver/max)	17 ± 5/ 41 ± 30	19 ± 8/ 40 ± 19	NS
Minimal Sao_2	79 ± 8	82 ± 9	NS
Minimal Sao_2 <85% (%)	69	48	NS

Values are expressed as mean ± SD or as percentage of patients presenting with the finding.

* Statistically nonsignificant difference ($p > 0.05$).
† Often or always.
‡ Minimal score, 0 (no sleepiness); abnormal score, >10; maximal score, 24.[22]
§ In men, ≥36; in women, ≥32.[23]
¶ Habitual snoring + Epworth Sleepiness Scale >10 or abnormal SDQ-SA score.
# For at least 10% of recording time.

SDQ-SA = Sleep Disorders Questionnaire–Sleep Apnea.

there was a bias toward selection of more severe stroke patients. In the second study, criteria of inclusion and clinical characteristics of the patients were not specified, and only men were studied. Approximately one-third of the patients described here had CSR, and this finding was the sole respiratory abnormality in only 1 patient. CSR tended to predominate in non-REM sleep and to improve in REM sleep, whereas obstructive events were more commonly seen or more severe in REM sleep, as reported previously.[29] On occasion, the distinction between the two breathing disturbances was difficult, and a combina-

tion of obstructive and nonobstructive respiratory problems was present. Our results suggest that the high frequency of CSR reported by others in patients with acute stroke studied with impedance pneumography but without monitoring of airflow and state of consciousness[30,31] could have been an overestimation because of the inability to recognize obstructive SA. Reports of improvement in both CSR and central apneas during treatment with continuous positive airway pressure further support this possibility and emphasize the complex interaction of peripheral and central influences in breathing control.[32,33]

The frequency of SA reported in this study is similar to that reported in patients with cardiac disease[4,34] but well above the 15% found in our age- and gender-matched control group and the 5 to 35% reported in the literature for healthy elderly subjects.[35,36] In our study the frequency of SA was similar in males and females. Although the number of respiratory events required for the diagnosis of SA remains controversial and our normal control subjects were to some extent screened for conditions associated with SA, the high percentage of our subjects with an AHI of $\geq$20 and with a minimum Sao_2 of <85% suggests that clinically significant SA is present in many patients with acute TIA and stroke.

The second main finding of our study was the lack of significant differences in the frequency of SA between the TIA and stroke groups. This finding could reflect a selection bias in that we cannot rule out the possibility that findings may differ in younger patients with TIA and a low cardiovascular risk profile who are investigated as outpatients. Alternatively, sample sizes may be inadequate to detect a real difference between subjects with TIA and stroke. Nevertheless, the high frequency of SA in the subjects with TIA suggests that in most cases SA probably is not a consequence of acute brain ischemic injury. On the other hand, acute stroke may aggravate or even cause SA in some patients. In fact, we observed a trend toward higher AHI and AI values in patients with stroke. Also, severe strokes were associated with more severe SA and with more prominent forms of CSR, although the overall frequencies of the latter breathing disturbance and of central apneas were similar in the stroke and TIA groups. The limited number of patients in this study does not allow conclusive analysis of patient subgroups. Patient characteristics as well as topography and etiology of stroke possibly influence the frequency, severity, and type of sleep breathing disturbances. In another study, for example, an AHI of $\geq$10 occurred in only 3 of 12 young patients (mean age, 43 years) with small thalamic infarctions of presumed cardioembolic etiology.[37]

The third main finding of the study was that clinical prediction of the presence of SA in patients with TIA or stroke may be possible in fewer than two-thirds of cases. Habitual snoring was reported by 58% of our patients with TIA or stroke. In a recent analysis of 164 consecutive patients hospitalized acutely because of acute cerebrovascular events, a similar percentage of 48% was reported.[38] In our study, the sensitivity for the diagnosis of SA was similar when the report of habitual snoring was combined with the complaint of excessive daytime sleepiness (as assessed with the ESS) or when using the SDQ-SA score and lower than what has been found in other selections of patients.[23] Also, there were no significant differences in the frequency of SA according to gender and age above or below 50. Studies in larger groups of patients are needed to identify the best predictors of SA in patients with TIA or stroke.

Our study supports the hypothesis of a strong association between SA and cerebrovascular disease but does not prove that SA is an independent risk factor for stroke. Earlier studies, however, have suggested that snoring may represent an independent risk factor for stroke even after adjustment for other associated cardiovascular risk factors.[12,13,38]

Several lines of evidence suggest that SA may lead to increased risk of cerebrovascular events. First, SA is associated with hypertension and ischemic heart disease.[3,10,11,39-41] Second, hypoxia and sympathetic activation after apneas may accelerate atherogenesis.[42] Third, hypotension, cardiac ischemia,[5,43,44] bradyarrhythmias[45,46] associated with apneas,[47,48] decreased fibrinolytic activity during wakefulness and sleep,[49,50] and high catecholamine levels and tachyarrhythmias related to sudden arousals may contribute to hemodynamic, thrombotic, and cardioembolic strokes. Fourth, altered cerebral blood flow,[40,51,52] fluctuations in intracranial pressure,[51] and impaired cerebrovascular autoregulation[52] may aggravate stroke after its onset.

Although the data presented here should be interpreted with caution and considered preliminary, our observations suggest that clinical assessment for the presence of SA should be part of the evaluation of patients who present with acute cerebral ischemia and that sleep studies should be obtained at least in such patients when SA is suspected. Based on our study and on data available from observational studies, we believe that SA should be considered a probable risk factor for stroke.[53,54]

Further studies are needed to identify more precisely the degree of association between SA and cerebrovascular diseases in different subgroups of patients, to determine the best predictors and diagnostic approach for SA, to assess its impact on the prognosis of TIA and stroke, and to determine whether treatment of coexisting SA alters the short- and long-term courses of cerebrovascular disease.

Addendum. After this manuscript was submitted for publication, two studies were published on SA in stroke patients with findings similar to ours. In a series of 19 patients with ischemic stroke admitted to a rehabilitation ward 1 to 10 weeks from the acute event, 95% of the patients had an AHI of >10 (Good et al, Stroke 1996;27:252–257). In a prospective study of 24 consecutive patients assessed 2 to 5

weeks after ischemic or hemorrhagic stroke, an AHI of >10 was found in 77% of 13 men and 64% of 11 women (Dyken et al, Stroke 1996;27:401–407). In none of the two studies were TIA patients studied.

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