Relationship of Sleep Apnea to Functional Capacity and Length of Hospitalization Following Stroke

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Study Objectives: Recent evidence indicates that sleep apnea is common in patients with stroke. We hypothesized that the presence of sleep apnea among stroke patients would be associated with a greater degree of functional disability and longer hospitalization following stroke.
Design: Prospective study.
Setting and Patients: Sixty-one stroke patients admitted to a stroke rehabilitation unit.
Interventions: N/A.
Measurements and Results: Sleep studies were performed on all patients, and sleep apnea was defined as an apnea-hypopnea index of 10 or more per hour of sleep. Patients underwent functional assessments, including the Functional Independence Measure. Sleep apnea was found in 72% of patients; 60% had predominantly obstructive sleep apnea, while 12% had predominantly central sleep apnea. Although the severity of stroke was similar in the 2 groups, compared to patients without sleep apnea, those with sleep apnea had lower functional capacity [Functional Independence Measure score (mean ± SEM) 80.2 ± 3.6 versus 94.7 ± 4.3, $p<0.05$ at admission, and 101.5 ± 2.8 versus 112.9 ± 2.7, $p<0.05$ at discharge] and spent significantly more days in rehabilitation (45.5 ± 2.3 versus 32.1 ± 2.7 days, $p<0.005$). In addition, multiple regression analysis showed that obstructive sleep apnea was significantly and independently related to functional impairment and length of hospitalization.
Conclusions: Sleep apnea is very common among stroke patients undergoing rehabilitation, and its presence is associated with worse functional impairment and a longer period of hospitalization and rehabilitation. These data suggest that sleep apnea may be contributing to functional impairment and prolonged hospitalization following stroke.
Key Words: stroke, sleep apnea, rehabilitation
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INTRODUCTION

STROKE IS A COMMON DEBILITATING CONDITION IN THE OLDER POPULATION AND ACCOUNTS FOR A LARGE NUMBER OF ACUTE HOSPITAL ADMISSIONS. Chronic functional and mental disabilities are frequent sequelae of stroke, preventing patients with stroke from returning home. Because of this, patients with stroke are often hospitalized for prolonged periods in acute care hospitals and frequently must undergo further lengthy hospitalization in a stroke rehabilitation unit (SRU). Thus not only is stroke a serious medical condition, it is also a great financial burden on patients, their families, and the healthcare system. Unfortunately, medically, there is often very little that can be done to reverse the underlying condition. Thus, it would be important to identify any coexisting, potentially reversible, condition that could impair recovery from stroke. One such condition could be sleep apnea (SA).

Recent evidence indicates that SA, especially obstructive sleep apnea (OSA), is very common among patients with stroke.[1-6] A large population-based study also showed that OSA may be an independent risk factor for stroke.[7] Moreover, there is evidence to suggest that the presence of SA in patients with stroke is associated with poor functional status and high mortality.[1,2] OSA could predispose to stroke through its association with hypertension and increased platelet aggregability and blood coagulability.[8-14] Once a stroke has occurred, SA could aggravate functional and mental disabilities because of recurrent nocturnal hypoxia and fragmentation of sleep, as well as reduced cardiac output and cerebral perfusion.[15-18] If so, SA in patients with stroke may contribute to prolonged poststroke hospitalization and rehabilitation. However, there have been no studies examining the potential influence of SA on length of hospitalization following stroke. We therefore hypothesized that, in patients with stroke undergoing rehabilitation, the presence of SA will be associated with a greater degree of functional impairment and a consequent longer hospitalization than in patients with stroke but without SA.

METHODS

Subjects

We prospectively studied 61 consecutive patients following embolic, thromboembolic, or hemorrhagic strokes from their time of admission to the SRU of the Toronto Rehabilitation Institute from acute care units in other hospitals until their time of discharge from hospital. The diagnosis of completed stroke was confirmed by neurologists and was based on 1) a history of sudden onset of a neurologic deficit that lasted more than 24 hours, 2) presence of a neurologic deficit on physical examination, and 3) brain lesion compatible with the neurologic deficit on computerized tomography or magnetic resonance imaging of the brain. Patients with previously diagnosed SA or any form of dementia were excluded from the study. Upon admission to the SRU, demographic characteristics, stroke risk factors (history of hypertension, coronary artery disease, heart failure, atrial fibrillation, diabetes mellitus, hypercholesterolemia, and cigarette smoking), height, weight, and body mass index were determined. The study protocol was approved by the Ethics Committee for Human Experimentation at the University of Toronto. Written informed

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consent prior to entry into the study was obtained from all patients or their families.

Assessments

The primary outcome of our study was the length of hospitalization in the SRU following acute stroke, and the secondary outcome was the Functional Independence Measure (FIM) score at discharge from the SRU. Upon admission to the SRU, all patients underwent a series of tests administered by a research assistant to determine their level of functional and mental disability. The severity of the stroke according to the degree of neurologic impairment from brain damage was assessed by the Canadian Neurological Scale (CNS).[19] This test includes 8 items that measure level of consciousness, orientation, speech, motor function, and facial weakness. Functional capacity was evaluated by the FIM.[20] The FIM includes 18 items that measure self-care, sphincter control, mobility, locomotion, communication, and social cognition. Because the FIM is used as an important determinant of fitness to be discharged, it was also administered at the time of discharge from the SRU. Cognitive function was assessed by the Mini-Mental State Examination (MMSE)[21] and the Stroke Unit Mental Status Examination (SUMSE).[22] The SUMSE is a test that was specifically developed to assess cognitive impairment of stroke patients undergoing an active rehabilitation program. The test evaluates orientation, concentration, judgment and reasoning, immediate and remote memory, praxis, and language. For all the above tests, lower scores indicate greater impairment. In addition, subjective sleepiness was analyzed by means of the Epworth Sleepiness Scale (ESS).[23] Scores greater than 10 are indicative of excessive daytime sleepiness.

Sleep Studies

Overnight polysomnography was performed using standard techniques for scoring sleep stages and arousals as described for our laboratory.[24-26] The time from stroke onset until polysomnography was (mean ± SEM) 44.6 ± 3.1 days. Thoracoabdominal movements and tidal volume (V_T) were monitored by a calibrated respiratory inductance plethysmograph (Respitrace; Ambulatory Monitoring Inc., White Plains, NY).[27] Oxygen saturation (SaO_2) was continuously monitored by a pulse oximeter (Oxyshuttle; Sensormedics Corp., Anaheim, CA) and recorded on a computerized sleep scoring system (Sandman, Nellcor Puritan Bennett Ltd., Ottawa, ON.). The lowest SaO_2 was recorded, and the mean SaO_2 during sleep was quantified as the average of the highest and lowest SaO_2 for each 30-second epoch averaged over the entire sleep period. The O_2 desaturation index (DI) was calculated by dividing the number of desaturations with an SaO_2 less than 90% by total sleep time. The electrocardiogram was recorded using a single precordial lead. Apneas were defined as an absence of V_T for at least 10 seconds and were classified as obstructive if there was paradoxical rib cage and abdominal motion, and as central if there was no rib cage or abdominal motion. Hypopneas were defined as a 50% or greater reduction in V_T for at least 10 seconds. Hypopneas were classified as obstructive if there was paradoxical or phase-shifted motion of the rib cage and abdominal channels, and as central if there was no paradoxical or phase-shifted movement of the rib cage and abdomen. The frequency of apneas and hypopneas per hour of sleep was expressed as the apnea-hypopnea index (AHI). Patients having an AHI less than 10 per hour of sleep were classified as non-sleep apnea (non-SA), while those with an AHI of 10 or greater per hour of sleep were classified as having SA. OSA was diagnosed when at least 50% of the respiratory events were of the obstructive type, while central SA (CSA) was diagnosed when more than 50% of respiratory events were of the central type.

Statistical Analysis

All data are expressed as mean values ± SEM. Statistical analysis was performed using SigmaStat 2.03 (SPSS Inc., Chicago, IL). To compare values between the groups, we used the unpaired *t*-test or the Mann-Whitney rank sum test for continuous variables, as appropriate. The chi-square test or the Fisher's exact test was used for nominal variables. In addition, to determine whether there were significant relationships between the dependent outcome variables—FIM at discharge or length of hospitalization—and the independent variables—age, gender, type and location of stroke, CNS, obstructive and central AHI, DI, frequency of arousals from sleep, and MMSE—simple linear regression analyses were examined. Variables were then entered into a multiple linear regression model with length of hospitalization and FIM at discharge as the dependent variables if the p value was less than 0.05. Values of p greater than 0.05 were considered statistically significant.

RESULTS

One patient who could not sleep during polysomnography was excluded from this study. Of the remaining 60 patients, 36 (60% of total) were men and 24 (40%) were women. Their mean age was 66.4 ± 1.6 years. There were 48 patients with ischemic stroke (80% of total) and 12 patients with hemorrhagic stroke (20%). Forty-nine patients suffered from their first stroke, whereas 11 had had a previous stroke. Since we did not find any differences in the prevalence of SA or in functional capacity between patients with a first stroke or with a history of a previous stroke, we studied them together. The time from the onset of their most recent stroke until SRU admission averaged 21.9 ± 1.9 days. The average time spent in the SRU was 41.7 ± 2.0 days. The prevalence of SA was 72%. Eighty-eight percent of patients with SA were discharged home, and 12% of them went to a chronic care facility or nursing home, while 94% of those without SA were discharged home.

Characteristics of the subjects are shown in Table 1. There were 36 patients with predominantly OSA (60% of total) and only 7 patients with predominantly CSA (12%). There were no significant differences in age, body mass index, systolic or diastolic blood pressure, and ESS scores between the non-SA and SA groups. The severity of stroke, assessed by the CNS, did not differ significantly between patients with and without SA. In addition, although there was a tendency to a higher prevalence of hemorrhagic and infratentorial strokes in patients with SA than in those without SA, these differences were not statistically significant. There was a tendency for fewer risk factors for stroke among the patients with SA, but only heart failure was significantly less frequent ($p<0.05$) (Table 2).

Polysomnographic data are shown in Table 3. By design, the AHI was significantly higher in the SA than in the non-SA group. The DI was also higher in patients with SA than in those without SA. However, sleep structure did not differ significantly between the two groups.

Comparisons of functional and cognitive assessments between stroke patients with and without SA are shown in Table 4. Patients with SA had

Table 1—Characteristics of subjects

	Non-SA n = 17 (28%)	SA n = 43 (72%)
Age, yr	63.6 ± 4.0	67.5 ± 1.5
Male: Female, n	7 : 10	29 : 14
Body mass index, kg/m^2	27.7 ± 1.2	28.8 ± 1.0
Systolic blood pressure, mmHg	135 ± 3	132 ± 2
Diastolic blood pressure, mmHg	78 ± 2	76 ± 1
Epworth Sleepiness Scale	5.4 ± 0.5	5.5 ± 0.5
Canadian Neurological Scale	8.7 ± 0.3	8.0 ± 0.2
Stroke characteristics		
Type		
Ischemic, n (%)	15 (88%)	33 (77%)
Hemorrhagic, n (%)	2 (12%)	10 (23%)
Location		
Supratentorial, n (%)	15 (88%)	32 (75%)
Infratentorial, n (%)	1 (6%)	7 (16%)
Supra- & Infratentorial, n (%)	1 (6%)	4 (9%)

Abbreviations: SA, sleep apnea. Values are means ± SEM.
There were no significant differences between the groups for any of the variables.

worse functional capacity than did those without SA, as measured by the FIM, both at the times of admission to and discharge from the SRU. In addition, univariate analyses demonstrated significant correlations between FIM at discharge from the SRU and CNS (R = 0.575, p<0.001), obstructive AHI (R = 0.394, p = 0.002), and MMSE (R = 0.419, p = 0.001). However, there was no significant relationship between FIM at discharge and central AHI. In the multiple linear regression analysis, CNS, obstructive AHI, and MMSE were found to be significantly and independently correlated with FIM at discharge from the SRU (Table 5A). For every 10-unit increase in obstructive AHI, the FIM score decreased by 2.3, and for every 1-unit increase in CNS or MMSE, the FIM increased by 3.99 and 1.27, respectively. There were no significant relationships between FIM at discharge and age, gender, type and location of stroke, DI, or frequency of arousals from sleep.

There was no significant difference in the length of hospitalization in an acute care facility from the onset of stroke until admission to the SRU between the two groups (Table 4). However, stroke patients with SA spent 14 (40%) more days in the SRU (p<0.005) and 16 (30%) more total days in the hospital than did patients without SA (p<0.05) but still had significantly worse FIM scores at discharge. In addition, univariate analyses showed that the length of hospitalization in the SRU correlated significantly with CNS (R = 0.394, p = 0.002) and obstructive AHI (R = 0.374, p = 0.003). In the multiple linear regression analysis, CNS and obstructive AHI were found to be significantly and independently correlated with the length of hospitalization in the SRU (Table 5B). For every 10-unit increment in obstructive AHI, there was a 2.7-day increase in length of hospitalization in the SRU, and for every 1-unit increase in CNS, there was a 3.08-day decrease in length of hospitalization. There were no significant relationships between the length of hospitalization and age, gender, type and location of stroke, central AHI, DI, arousals from sleep, MMSE or SUMSE.

DISCUSSION

Our study has given rise to several novel and interesting observations. First, we found a very high (72%) prevalence of SA among this group of patients with stroke who were admitted to a rehabilitation unit. Second, compared to patients without SA, those with SA had a greater degree of functional impairment. Third, with respect to our primary outcome measure, we found that patients with SA had a 14-day (ie, 40%) longer hospitalization on average in the SRU than did those without SA. The length of hospitalization was significantly related to the severity of obstructive, but not central, SA, independent of the severity, type, and location of the stroke. These findings indicate that SA is very common among patients admitted to an SRU. They further suggest that SA contributed to the patients' functional incapacity and length of rehabilitation.

The findings of the present study are consistent with previous studies in showing a very high prevalence of SA in patients with stroke. Using the same AHI cut off at least10 per hour as in our study, others found that 43% to 95% of patients with stroke had SA.[1-6] Our data are also in agreement with previous studies in showing a marked predominance of OSA over CSA and in showing no relationship between the presence and type of SA and stroke type or location.[4,6]

Although we were unable to determine whether SA preceded or followed stroke, evidence from previous studies suggests that, in most patients, SA more likely precedes the onset of stroke. For example, in a large epidemiologic study, OSA was found to be an independent risk factor for stroke.[7] Bassetti et al[3] found an equal prevalence of SA in patients following transient ischemic attack and completed stroke. Since transient ischemic attack is not associated with permanent brain damage, and often precedes stroke, these data also suggest that, in many cases, SA precedes the onset of stroke. In another study, Parra et al[6] observed that, in patients with stroke, the number of central apneas decreased from immediately after stroke onset to 3 months later, while the number of

Table 2—Stroke risk factors

	Non-SA (n = 17)	SA (n = 43)
Hypertension, n (%)	13 (77%)	31 (72%)
Coronary artery disease, n (%)	5 (29%)	10 (23%)
Heart failure, n (%)	6 (35%)	3 (7%)*
Atrial fibrillation, n (%)	5 (29%)	6 (14%)
Diabetes mellitus, n (%)	10 (59%)	14 (33%)
Hypercholesterolemia, n (%)	3 (18%)	10 (23%)
Smoking, n (%)	4 (24%)	6 (14%)

* p<0.05

Table 3—Baseline polysomnographic data

	Non-SA (n = 17)	SA (n = 43)
AHI, #/hr sleep	4.3 ± 0.7	27.3 ± 2.9‡
obstructive AHI, #/hr	2.8 ± 0.6	19.1 ± 2.4‡
central AHI, #/hr	1.5 ± 0.4	7.4 ± 2.2
Mean SaO_2, %	94.8 ± 1.0	94.1 ± 0.4
Lowest SaO_2, %	87.2 ± 1.5	76.9 ± 1.7‡
DI, no/hr sleep	1.2 ± 0.3	9.2 ± 2.0‡
TST, min	317.2 ± 17.0	342.1 ± 10.9
Sleep latency, min	19.2 ± 3.1	26.9 ± 5.3
Sleep efficiency, %	73.8 ± 3.1	72.4 ± 2.1
Stage I sleep, % of TST	6.4 ± 0.7	7.7 ± 0.9
Stage II sleep, % of TST	59.9 ± 2.0	62.5 ± 1.6
SWS, % of TST	15.8 ± 2.2	12.2 ± 1.3
REM sleep, % of TST	17.9 ± 1.4	17.6 ± 1.2
Arousals, #/hr sleep	16.9 ± 2.9	20.7 ± 1.7
PLM, #/hr sleep	20.6 ± 9.3	13.6 ± 4.7

SA, sleep apnea; AHI, apnea-hypopnea index; SaO_2, oxyhemoglobin saturation; DI, desaturation index; TST, total sleep time; SWS, slow wave sleep; REM, rapid eye movement; PLM, periodic leg movements.
Values are means ± SEM. ‡ p<0.001.

Table 4—Comparison of functional and cognitive tests, and length of hospitalization

	Non-SA (n = 17)	SA (n = 43)
FIM (range 18-126)		
at admission	94.7 ± 4.3	80.2 ± 3.6*
at discharge	112.9 ± 2.7	101.5 ± 2.8*
MMSE (range 0-30)	24.9 ± 0.8	23.3 ± 0.6
SUMSE (range 0-65)	51.6 ± 2.3	50.1 ± 1.3
Time in acute care facility, days	20.3 ± 2.7	22.5 ± 2.4
Length of hospitalization in the SRU, days	32.1 ± 2.7	45.5 ± 2.3†
Total length of hospitalization, days	51.4 ± 3.8	67.0 ± 3.6*

SA, sleep apnea; FIM, Functional Independence Measure; MMSE, Mini-Mental State Exam; SUMSE, Stroke Unit Mental Status Exam; SRU, stroke rehabilitation unit
Values are means ± SEM. * p<0.05. † p<0.005.

Table 5—Multivariate regression analyses

A. FIM at discharge from hospital

Variables	Coefficient	Standard error	p value
Canadian Neurological Scale	3.992	1.202	0.002
Obstructive AHI	-0.232	0.104	0.030
MMSE	1.274	0.430	0.005

Full model: multiple R = 0.665, multiple R^2 = 0.443
FIM, Functional Independence Measure AHI, apnea-hypopnea index; MMSE, Mini-Mental State Exam

B. Length of hospitalization in the SRU

Variables	Coefficient	Standard error	p value
Canadian Neurological Scale	-3.079	1.361	0.028
Obstructive AHI	0.265	0.128	0.043

Full model: multiple R = 0.464, multiple R^2 = 0.215
SRU, stroke rehabilitation unit; FIM, Functional Independence Measure; AHI, apnea-hypopnea index; MMSE, Mini-Mental State Exam

obstructive apneas did not change. These findings suggest that acute stroke predisposes to CSA but that, in many cases, OSA probably precedes the onset of stroke. OSA would predispose to stroke because of its association with hypertension, increased platelet aggregability, and elevated plasma fibrinogen and whole blood viscosity.[8-14] In this regard, there was a tendency for fewer risk factors for stroke among the patients with SA than in those without SA (Table 2). These observations provide further circumstantial evidence that SA itself may be a risk factor for stroke.

The most interesting and novel findings of our study concern the relationships between SA, functional capacity, and length of hospitalization. We found that on admission to and discharge from the SRU, patients with SA had significantly worse functional capacity, as measured by the FIM, than did patients without SA. Furthermore, the severity of obstructive (ie, obstructive AHI) but not central SA was significantly related to FIM scores at time of discharge from the SRU. A striking finding of our study was that patients with SA had a 14-day (40%) longer hospital stay in the SRU than did patients without SA. As expected, the severity of stroke (ie, CNS score) contributed to prolonged hospitalization. However, the severity of OSA also contributed significantly to the length of hospitalization, independent of stroke severity (Table 5B). At discharge from the SRU, FIM scores remained lower in patients with SA than in those without SA, despite their longer period of rehabilitation (Table 4). Patients with and without SA were very well matched for severity of stroke (as determined by the CNS), age, and body mass index (Table 1). Finally, although there was a tendency for a higher proportion of hemorrhagic and infratentorial strokes in the group with SA, when other factors were taken into account, the type and location of stroke did not have a significant independent impact on outcomes. Thus differences in functional capacity and length of hospitalization between patients with and without SA could not be attributed to these potentially confounding influences.

The above observations suggest that SA plays a role in impairing recovery from stroke. There are several reasons why SA might have adverse effects on neurologic function following stroke. Apneas and hypopneas cause recurrent cerebral hypoxia that can lead to the elaboration of neuroinhibitory peptides and increased apoptosis of cerebral neurons, both of which could further compromise cerebral function.[15,28] Although we did not find any significant relationship between nocturnal DI and functional impairment, this does not rule out apnea-related hypoxia as a factor in impairing functional capacity.

Apneas also lead to cerebral hypoperfusion secondary to reduced cardiac output.[16-18] Reduced cerebral perfusion could predispose to cerebral ischemia and cerebrovascular accidents in subjects with flow-limiting lesions of the carotid or vertebral circulations. These adverse effects of SA might extend brain damage, impair recovery from stroke, or both. Interestingly, the inverse relationship between functional capacity and AHI held only for obstructive events but not for central ones. One possible explanation for this is that central events were too few to have a significant influence relative to more frequent obstructive events. Another is that reductions in cerebral blood flow are more pronounced during obstructive events than during central ones.[18] A third possibility is that central events resulted from the stroke itself and therefore did not contribute independently to functional impairment beyond that due to the stroke.[6]

Sleep disruption and excessive daytime sleepiness could also play a role in impairing functional capacity in patients following stroke.[29,30] However, in our study, the degree of sleep disruption did not differ significantly between patients with and without SA due to frequent periodic leg movements and arousals in both groups. Moreover, among patients with SA, sleep latencies were normal (mean, 27 minutes) and did not differ from those in patients without SA. This latter finding is in keeping with the lack of subjective daytime sleepiness among patients with SA as judged by normal ESS scores that did not differ from those in patients without SA (Tables 1 and 3). Large epidemiologic studies have demonstrated that the majority of individuals with SA identified on polysomnography do not complain of excessive daytime sleepiness.[31] Thus it is possible that our patients generally had relatively asymptomatic SA that eluded clinical detection prior to participation in this study.

SA is often associated with neurocognitive and performance deficits, which could aggravate cognitive impairment following a stroke.[32-34] Contrary to our expectations, however, we found no significant differences in cognitive ability between patients with and without SA (Table 4). These findings imply either that the presence of SA had little influence on cognitive impairment or that the tests used were too insensitive to detect subtle differences in cognition between the groups. Since it is difficult for stroke patients with mental handicaps to perform complicated neurocognitive tests, we applied relatively simple and clinically applicable tests that are the standard means of assessing cognitive ability in patients following stroke. For example, although the MMSE has been best validated as a test of cognition in diffuse brain diseases where it detects rather gross impairment, it is widely used to assess gross cognitive function in patients with stroke. However, since it is not designed to detect subtler degrees of cognitive impairment, our findings in this regard must be interpreted with caution. Nevertheless, the reason why SA was associated with worse functional but not cognitive capacity in our study remains unclear. Further research will be required to address this issue.

Patients who participated in our study may not have been representative of all stroke patients. First, they had to survive their stroke. Second, to be admitted to the SRU, they had to have sufficient disability to prevent them from immediately returning to the community. However, they could not be so disabled that they did not have potential for rehabilitation. Thus, our patients were in the mid range of stroke severity and disability. Discharge criteria from the rehabilitation program are that patients are able to return and live in the community or that their progress has reached a plateau and they, therefore, require long-term care. Since the great majority of patients in our study were discharged home from the SRU, social factors appear not to have played a major role in the length of hospitalization. There are obviously many factors that influence the length of hospitalization following a stroke, such as co-existing medical conditions that may require treatment. Our data suggest that SA may be one such condition.

In North America, stroke is the third leading cause of death and the leading cause of long-term disability.[35] It often requires prolonged hospitalization that imposes a huge financial burden on the healthcare system. For example, in the United States, there are over 3 million stroke survivors and the cost of acute and long-term care is estimated to be $30 billion per year.[35] The situation is similar in Canada.[36,37] The present findings may be relevant to this issue. We demonstrated that the 72% of patients with stroke who had SA had a much longer hospitalization than did those without SA, even after controlling for severity of stroke and other potentially confounding variables. These data suggest that SA may be having an adverse impact on the social and financial burden associated with stroke disability and rehabilitation. Further studies involving larger numbers of patients will be required to examine this possibility.

In summary, the present study demonstrates that SA, especially OSA, is very common in patients admitted for stroke rehabilitation and is associated with longer hospitalization and worse functional capacity. From the clinical standpoint, whether SA developed before or after the onset of stroke is not critically important: in either case, the possibility remains that treatment of SA might improve functional capacity and reduce length of hospitalization. Accordingly, our data provide a strong rationale to conduct well-designed randomized clinical trials to determine whether treatment of SA could improve functional outcomes and reduce the length and cost of rehabilitation in patients following a stroke.

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