Continuous Positive Airway Pressure Treatment in Sleep Apnea Prevents New Vascular Events After Ischemic Stroke*

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Study objectives: A study was made of the role of continuous positive airway pressure (CPAP) treatment in the prevention of new vascular events following ischemic stroke or transient ischemic attack.

Design: Prospective study.

Patients and interventions: Demographic data, vascular risk factors, clinical manifestations associated to sleep apnea-hypopnea syndrome, and neurologic parameters were recorded in a group of patients presenting with acute ischemic stroke at least 2 months previously. A polygraphic study was carried out 2 months after the acute episode in all patients, with the prescription of CPAP in the event of an apnea-hypopnea index (AHI) > 20. Two groups were defined: patients who could tolerate CPAP (group 1), and patients who could not tolerate CPAP after 1 month of initial adaptation (group 2). Patients with an AHI < 20 were excluded. The incidence of new vascular events was evaluated throughout follow-up (18 months) in all patients, with an analysis of the role of CPAP in protecting the patients against such events.

Results: Ninety-five patients were studied. Fifty-one patients (53.7%; mean age, 72.7 ± 9.4 years) presented with an AHI > 20, and 15 patients (29.4%) tolerated CPAP. The incidence of new vascular events was greater in group 2 (6.7%) vs group 1 (36.1%; long-rank, p = 0.03). Intolerance of CPAP increased the probability of a new vascular event fivefold (odds ratio, 5.09) adjusted for other vascular risk factors and neurologic indexes.

Conclusions: We concluded that CPAP treatment during 18 months in patients with an AHI > 20 afforded significant protection against new vascular events after ischemic stroke.

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Key words: continuous positive airway pressure; ischemic stroke; sleep apnea; vascular events

Abbreviations: AF = atrial fibrillation; AHI = apnea-hypopnea index; AHT = arterial hypertension; BMI = body mass index; CI = confidence interval; CPAP = continuous positive airway pressure; SAHS = sleep apnea-hypopnea syndrome; TIA = transient ischemic attack; VE = vascular event; VRF = vascular risk factor

The relation between sleep apnea-hypopnea syndrome (SAHS) and certain vascular disorders has gained consistency in recent years.1,2 The most widely accepted hypothesis is that SAHS could constitute an independent vascular risk factor (VRF) for the development of cardiac ischemia,3 cardiac arrhythmias,4 congestive heart failure,5 cerebrovascular diseases,6–8 and particularly systemic arterial hypertension (AHT).9 In this sense, some authors have reported a drop in BP,10 a decrease in fibrinogen concentration,11 or stabilization of sympathetic tone12 in patients with SAHS during treatment with continuous positive airway pressure (CPAP), although the studies conducted to date in this sense have been contradictory.

The relation between SAHS and ischemic stroke has been the subject of much debate in recent years. Different studies6,8 have shown an excessive prevalence of obstructive respiratory events weeks after ischemic stroke or transient ischemic attack (TIA) following stabilization of the neurologic process.
However, an excess of SAHS-related symptoms prior to stroke has been reported. These observations suggest that independently of the existence of a transient worsening of SAHS as a result of stroke in its acute phase, many cases of obstructive SAHS could precede the latter and act as a risk factor for new cardiovascular or cerebrovascular events, although no conclusive demonstration of a causal relation has been forthcoming to date.

Stroke is known to be the first cause of incapacitation in the Western world. The risk of a recurrent vascular event (VE) in patients who have had ischemic stroke is estimated to be 13 to 14% during the first year and increases 6% each year thereafter. If the hypothesis identifying SAHS as a risk factor for ischemic stroke were correct, then long-term CPAP treatment of stroke patients could help reduce the number of new VEs and the associated morbidity/mortality. However, this possibility has not been evaluated to date (and to our knowledge). The present study analyzes the role of CPAP treatment during 18 months in the prevention of new cerebrovascular or cardiovascular events in individuals who have had ischemic stroke or TIA.

**Materials and Methods**

**Patients**

The present study selected all patients with ischemic stroke or TIA admitted to our center during the year 2002, and who had passed the acute phase of the neurologic event and were in a stable phase. Patients previously treated with CPAP were excluded. Patients with a new VE prior to conduction of the sleep diagnostic study were temporarily excluded until 2 months after the last cerebral vascular event. The study protocol was approved by the local ethics committee, and all patients gave informed consent to inclusion in the study.

**Assessment of Stroke and VRFs**

The diagnosis and location of ischemic stroke or TIA were determined by a neurologist, based on evaluation of the existing neurologic defects and of brain CT scans conducted in the first few hours after patient admission to the emergency department, and again several days later. Stroke subtypes were classified according to the Oxfordshire classification. Functional disability and neurologic impairment at hospital admission were evaluated using widely used neurologic scales: the Barthel index, which assesses daily activity on a scale of 0 to 100 (a score of 100 corresponding to full patient autonomy) and the Canadian scale, quantifying stroke-related symptoms severity from 0 to 10 (decreasing scores indicating greater severity).

Data were collected in all patients on the existence of the following VRFs: internal carotid stenosis (stenosis affecting > 50% of the vascular lumen) assessed by continuous Doppler flowmetry, transcranial Doppler flowmetry, and with magnetic resonance angiographic confirmation where appropriate; body mass index (BMI); current smoking (> 10 cigarettes per day); AHT (defined according to World Health Organization criteria or by the current use of antihypertensive drugs); the number of antihypertensive drugs taken; hypercholesterolemia (> 250 mg/dL in peripheral blood); diabetes mellitus; atrial fibrillation (AF); ischemic heart disease; and fibrinogen concentration in peripheral blood. Poorly controlled AHT was defined by persistent pressure values in excess of the normal values according to World Health Organization criteria despite correct adhesion to the prescribed antihypertensive medication. Following stroke, all patients received usual antiplalet treatment. In the presence of AF, treatment was moreover started with oral anticoagulants to maintain the international normalized ratio between 1.5 and 2.5 in the absence of contraindications, and an echocardiographic study was made to assess the presence of atrial thrombi. Both the neurologic and cardiological studies were based on the same standardized protocol in all patients. This protocol included VRF assessment based on the clinical history, the medication used by the patient, and certain complementary studies such as ECG, carotid Doppler ultrasound, and blood tests. The presence of any laboratory test alteration indicative of VRF was subsequently confirmed by repeated testing under the same conditions.

**Clinical Sleep Assessment**

Obstructive sleep apnea syndrome-related clinical manifestations prior to ischemic stroke were recorded as follows: A patient was considered to have significant snoring disorder if snoring occurred every or almost every night. Significant witnessed apneic episodes were those occurring every or almost every night, or repeatedly in the same night, and diurnal hypersomnia was assessed by the validated Spanish version of the Epworth sleepiness scale. Demographic and anthropometric variables (age, sex, BMI, and neck perimeter) were also recorded.

**Polygraphic Studies**

All patients finally included in the study underwent a respiratory polygraphic evaluation in the stable phase of stroke (2 months after the acute episode). CPAP treatment was offered to patients with an apnea-hypopnea index (AHI) > 20. The empirical initial CPAP pressure was calculated for each patient using a published prediction formula based on anthropometric parameters and on the AHI. In no patient did we prescribe an initial pressure of > 8 cm H2O in an attempt to improve compliance. This pressure was maintained for 1 month of initial adaptation until automatic CPAP titration was carried out. During this time, contact was maintained with the patient to provide instructions on the treatment and to try to solve any possible problems and side effects (particularly leakage problems), ensuring an appropriate nonleaking mask, since this problem in very common in such patients.

Both diagnostic and autotitration polygraphic studies were carried out using a portable system (Autoset Portable Plus II; ResMed; Sydney, Australia) as described elsewhere. Low-leak 55th percentile pressure was used. In the event of a severe leak (> 0.4 L/s), the titration was considered nonvalid and the test was repeated. Only those tests in which the patient claimed to have slept at least 3 h, and where at least 4 h of recording were available, were considered valid. We have defined apnea and hypopnea previously. All data were calculated as a function of total recording time. All tests were performed in our hospital, in rooms conveniently prepared to the effect by trained personnel.

**Follow-up**

Following the diagnostic study and posterior CPAP titration, two groups were established and subjected to follow-up for 18
months: patients with an AHI ≥ 20 who could tolerate CPAP treatment (group 1), and patients with an AHI ≥ 20 who could not tolerate CPAP treatment (group 2). Patients with an AHI < 20 were excluded from the study. Follow-up of the groups started the day after CPAP titration. Adequate CPAP tolerance was considered when the system counter indicated that the patient was using the device for at least 4 h at night during at least 70% of the follow-up nights.\textsuperscript{20} Data were collected on the appearance of new VEs in the course of the study. All patients were instructed to report to our center in the event of any suspected new VEs during the follow-up period. A new VE was defined as the documented appearance of a new cerebrovascular or ischemic coronary event. The neurologic event was diagnosed by a neurologist based on the clinical picture, the imaging studies, or the consequences of the event. Coronary events (angina or acute myocardial infarction) in turn were evaluated by a cardiologist based on the clinical manifestations, the ECG changes, and laboratory test findings. At the end of follow-up, the number of hypertensive patients was recorded, along with the number of antihypertensive drugs used, hypertension control, the neurologist based on the clinical manifestations, the ECG changes, and laboratory test findings. At the end of follow-up, the number of hypertensive patients was recorded, along with the number of antihypertensive drugs used, hypertension control, the neurologist based on the clinical manifestations, the ECG changes, and laboratory test findings. At the end of follow-up, the number of hypertensive patients was recorded, along with the number of antihypertensive drugs used, hypertension control, the neurologist based on the clinical manifestations, the ECG changes, and laboratory test findings. At the end of follow-up, the number of hypertensive patients was recorded, along with the number of antihypertensive drugs used, hypertension control, the neurologist based on the clinical manifestations, the ECG changes, and laboratory test findings. At the end of follow-up, the number of hypertensive patients was recorded, along with the number of antihypertensive drugs used, hypertension control, the neurologist based on the clinical manifestations, the ECG changes, and laboratory test findings. At the end of follow-up, the number of hypertensive patients was recorded, along with the number of antihypertensive drugs used, hypertension control, the neurologist based on the clinical manifestations, the ECG changes, and laboratory test findings. At the end of follow-up, the number of hypertensive patients was recorded, along with the number of antihypertensive drugs used, hypertension control, the neurologist based on the clinical manifestations, the ECG changes, and laboratory test findings. At the end of follow-up, the number of hypertensive patients was recorded, along with the number of antihypertensive drugs used, hypertension control, the neurologist based on the clinical manifestations, the ECG changes, and laboratory test findings. At the end of follow-up, the number of hypertensive patients was recorded, along with the number of antihypertensive drugs used, hypertension control, the neurologist based on the clinical manifestations, the ECG changes, and laboratory test findings. At the end of follow-up, the number of hypertensive patients was recorded, along with the number of antihypertensive drugs used, hypertension control, the neurologist based on the clinical manifestations, the ECG changes, and laboratory test findings. At the end of follow-up, the number of hypertensive patients was recorded, along with the number of antihypertensive drugs used, hypertension control, the neurologist based on the clinical manifestations, the ECG changes, and laboratory test findings. All data relating to new VEs and their evolution parameters (Barthel index and Canadian scale), and any variation in the VRF, including BMI or new unregistered VEs (patients describing clinical manifestations compatible with a new VE during follow-up, but who failed to report to our center). All data relating to new VEs and their course, and to new VRFs during follow-up were obtained based on the same standardized protocol used in the baseline period.

**Statistical Analysis**

All data were tabulated as means ± SD for quantitative variables, and as absolute values (percentage) in the case of qualitative variables. Normality of the variables was determined using the Kolmogorov-Smirnov test. Comparison of values recorded at baseline (between groups) and at the end of the study (within groups) was based on the Student’s t test for unpaired or paired means respectively, or the χ² test in the case of dichotomic variables. The analysis of the role of CPAP treatment in the prevention of new VEs was based on the construction of Kaplan-Meier survival curves for each group studied. The curves were compared by the log-rank test adjusted for the presence of AF. Identification of the factors related to the appearance of a new VE was based on Cox proportional hazard model (forwards stepwise multivariate analysis). We initially included in the equation all the independent variables relating to the general characteristics of the patients, the polygraphic parameters, characteristics of stroke and its clinical repercussions, cardiovascular risk factors, and tolerance of CPAP treatment. Lastly, a calculation was made of the number needed to treated with CPAP in order to avoid a new VE.\textsuperscript{30} Statistical significance was accepted at p < 0.05.

**RESULTS**

Of 139 consecutive patients admitted to our center with a diagnosis of ischemic stroke or TIA, 110 patients survived for at least 2 months after the ischemic cerebrovascular episode. Of these patients, two who had received CPAP treatment previously were excluded. Twelve patients either failed to give consent or did not report to the sleep study, and 3 patients could not be contacted following the acute episode. The diagnostic polygraphic study was carried out after 64 ± 11 days in 95 patients, except in 3 patients who had a new VE (repeat ischemic stroke in all cases) during this period of time. In these patients, polygraphy was performed 2 months later. A total of 51 patients (53.7%) with an AHI ≥ 20 were finally included. Mean age was 72.7 ± 9.4 years (range, 57 to 82 years; 63% men; BMI, 26.8 ± 4.4 kg/m²; neck circumference, 40.2 ± 6.3 cm). Of these, 39 patients (76.8%) had a chronic snoring habit; 15.7% ischemic heart disease; 35.3% hypercholesterolemia; and mean fibrinogen concentration, 335 ± 78 mg/dL. Twelve of the neurologic events (23.5%) consisted of TIs. The mean Barthel index was 76.9 ± 32.7, with a Canadian scale score of 8.17 ± 1.9. There were no significant differences in the baseline data between groups with respect to age, sex, VRF, or characteristics of stroke (Table 1). The mean duration of Autoset Portable Plus II registry was 6.3 ± 3.1 h (range, 3.5 to 9 h). The mean AHI was 37.4 ± 7.9, with an obstructive apnea index of 31.5 ± 10.8, a central apnea index of 2.1 ± 2.1, and a counting time with an oxygen saturation of < 90% of 10.2 ± 4.4%.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Group 1 (n = 15)</th>
<th>Group 2 (n = 36)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean age, yr (male gender, %)</td>
<td>73.1 (64)</td>
<td>72.3 (57)</td>
</tr>
<tr>
<td>AF</td>
<td>4 (26.7)</td>
<td>9 (25)</td>
</tr>
<tr>
<td>BMI</td>
<td>27.5 ± 4.8</td>
<td>26.2 ± 5.2</td>
</tr>
<tr>
<td>AHT</td>
<td>10 (67)</td>
<td>26 (72)</td>
</tr>
<tr>
<td>Anti-AHT drugs</td>
<td>0.84 ± 0.6</td>
<td>0.93 ± 0.7</td>
</tr>
<tr>
<td>TIA</td>
<td>4 (26.7)</td>
<td>8 (22.2)</td>
</tr>
<tr>
<td>LACI/POCI/TACI/PACI, %</td>
<td>40/13/27/20</td>
<td>33/14/20/33</td>
</tr>
<tr>
<td>Epworth sleepiness scale</td>
<td>8.7 ± 5.2</td>
<td>6.4 ± 4.1</td>
</tr>
<tr>
<td>Total AHI</td>
<td>40.8 ± 14.2</td>
<td>34 ± 12.5†</td>
</tr>
<tr>
<td>Obstructive apnea index</td>
<td>36 ± 8.5</td>
<td>27 ± 10.7†</td>
</tr>
<tr>
<td>CT90%, %</td>
<td>11.2 ± 4.7</td>
<td>8.9 ± 4.4†</td>
</tr>
<tr>
<td>Barthel index</td>
<td>89.3 ± 20.8</td>
<td>64.5 ± 35.7§</td>
</tr>
<tr>
<td>Canadian scale</td>
<td>8.5 ± 0.8</td>
<td>8.2 ± 2.2†</td>
</tr>
</tbody>
</table>

*Data are presented as mean ± SD or No. (%) unless otherwise indicated. LACI = lacunar syndromes; POCI = partial posterior circulation syndromes; TACI = total anterior circulation syndromes; PACI = partial anterior circulation syndromes; CT90% = time spent at arterial oxygen saturation < 90%.†p = 0.04.§p = 0.03.§p = 0.008.
CPAP treatment was offered to all patients. An empiric pressure of 6 to 8 cm H₂O according to the formula used for each patient was applied during a 1-month adaptation period, and attempts were made to resolve all problems derived from such treatment. Nevertheless, CPAP treatment was discontinued in 36 patients (70.6%) because of a lack of tolerance at the end of this period. In this month of adaptation to CPAP treatment, no recurrent VEs were recorded in any of the study groups.

Following the initial 1-month adaptation period (33 ± 7 days), automatic CPAP titration was carried out in those patients who tolerated CPAP treatment (n = 15; 29.4%). In this group, AHI decreased on the night of titration from 40.8 ± 14.2 to 3.8 ± 3.3. The mean pressure (95th percentile) was 8.2 ± 4.4 cm H₂O (range, 6 to 13 cm H₂O). The mean duration of CPAP use was 5.7 ± 2.1 h during the study.

**CPAP Effectiveness**

Figure 1 shows the Kaplan-Meier survival curves corresponding to the groups studied. Fourteen new VEs were identified in the course of the study (11 ischemic strokes, 1 episode of angina, and 2 acute myocardial infarctions). The incidence of new VE in groups 1 and 2 was 6.7% and 36%, respectively (p = 0.03). Of these events, one stroke (6.7%) in group 1 and seven strokes (19.4%) in group 2 occurred in patients with AF.

During the 18 months of follow-up, there were four deaths (three strokes and one death due to nonvascular causes), with no significant differences being recorded among the follow-up groups. Likewise, there were no significant changes in the course of the study in the number of VRFs or BMI, although there was a non-significant tendency toward a decrease in the number of hypertensive individuals (67% vs 58%, p = 0.32), the number of antihypertensive drugs prescribed (0.84 vs 0.6, p = 0.24), and the percentage of patients with poorly controlled AHT (45% vs 30%, p = 0.12) in the group who tolerated CPAP. The neurologic recovery indexes improved in the two groups 18 months after stroke, although significant differences were only reached in terms of the improvement recorded in group 2 (Table 2).

Table 3 shows the result of the Cox multivariate analysis with the factors that independently influenced the appearance of a new VE. The variable exhibiting the most notorious influence was the absence of adaptation to CPAP treatment, which increased the risk of a new VE more than fivefold. Based on these data, it was calculated that the appearance of a new VE could be avoided in one of every four patients (95% confidence interval [CI], 2 to 26 patients) adequately treated with CPAP.

**CPAP Tolerance**

Only 15 patients (29.4%) of the total study population were able to tolerate the treatment for the full
duration of follow-up, with a mean of 6.4 ± 2.2 h per night (range, 4 to 10 h per night). The patients who failed to tolerate CPAP presented with significantly less hypersomnia (Epworth scale, 6.4 ± 4 vs 8.7 ± 5.2, p = 0.04), a lower AHI (34 ± 12.5 vs 48.0 ± 14.2, p = 0.03), and greater dependency following stroke according to the recorded Barthel index (64.5 ± 35.7 vs 89.3 ± 20.8, p = 0.008).

**DISCUSSION**

According to our results, CPAP treatment protected against the appearance of a new VE after ischemic stroke or TIA in patients with an AHI ≥ 20 following stabilization of the neurologic process, without inducing changes in the neurologic recovery parameters, although tolerance of the treatment was low. Some studies have analyzed the course of patients with ischemic stroke in relation to the incidence of SAHS after stabilization of the ischemic event. Good et al. reported poorer neurologic recovery and an increase in mortality after 1 year of follow-up in patients who presented with greater nocturnal oxygen desaturation, while Dyken et al. found that 4 years after stroke, the patients who died (21%) presented with greater AHI.

Very few studies have evaluated the role of CPAP treatment after stabilization of the neurologic event in the recovery of patients with high AHI values. After patients had 4 weeks of CPAP treatment, Sandberg et al. found that patients with an AHI > 15 showed improvement in the depression parameters without cognitive or neurologic amelioration. Wessendorf et al. reported a better well-being index and normalization of the "deeper" nocturnal BP pattern. The results of our study support these conclusions in part, since CPAP treatment was not shown to improve the neurologic course in our patients. In the present series, our patients improved their neurologic indexes in the course of the study, although the group that tolerated CPAP failed to reach statistical significance in this sense. This cannot be attributed to a negative effect of CPAP on the neurologic course of these individuals but rather to the fewer patients conforming the group that tolerated CPAP treatment, and to statistical regression to the mean—since this patient group presented significantly less neurologic impairment at the start of follow-up than the other group. However, the long time elapsed between the acute neurologic event and the start of CPAP treatment (approximately 2 months in our study) may cause irreversible stroke-induced brain damage and thus adversely affect patient recovery. In this sense, some authors postulate that CPAP treatment in the acute phase of stroke could contribute to recovery of the still viable ischemic zones, thereby improving the posterior neurologic course, although to our knowledge, no studies have yet explored this possibility.

We believe that the present study is the first to prospectively analyze the role of long-term CPAP treatment in protecting stroke patients from new VEs. Our main finding was that CPAP treatment during 18 months significantly reduced the risk of recurrent VEs adjusted for the presence of AF (ie, patients in whom the risk of a cardioembolic origin of stroke is greater), other VRFs, and neurologic indexes such as the Barthel index or the Canadian scale. Thus, intolerance of CPAP treatment was associated with a more than fivefold increase in the risk of new coronary or cerebrovascular events. Based on these results, we calculated that in order to
avoid a VE, four patients would have to be treated with CPAP (95% CI, 2 to 26). It is known that during the first 5 years after stroke, vascular recurrence is the principal cause of death in these patients, and that half of those who survive suffer incapacitation and are dependent on others for their daily life activities. This implies a very important deterioration in quality of life, as well as great health-care costs. On further taking into account the high prevalence of both ischemic stroke and SAHS in the general population, and the important percentage of stroke patients with a high incidence of sleep-disordered breathing (> 50% in our series showed an AHI ≥ 20), it can be seen that the number of events that could be avoided with CPAP treatment would be important indeed, with all the positive consequences in terms of morbidity and mortality this implies. We consider that in our patients, the protective role of CPAP treatment against recurrent VEs is of a multifactor origin. Improved AHT control may play a preponderant role, since AHT is known to be the most important risk factor for stroke. In this sense, we observed a nonsignificant tendency toward improved AHT control, with a decrease after 18 months of CPAP treatment in the percentage of hypertensive patients, the number of antihypertensive drugs required, and the number of patients with poor hypertension control. However, neither were all patients with AHT controlled by CPAP (30% in our series), nor is it to be expected that all the effects of long-term CPAP therapy are attributable to improved AHT control. It is possible that after 18 months of treatment, some other known effects of CPAP become manifest, such as a decrease in the risk of arteriosclerosis or in platelet aggregation.

The cutoff point selected for treating our patients with CPAP (AHI > 20) is based on the high incidence of SAHS observed in more elderly individuals such as those in our series (mean age, 72.7 years). In Spain, Duran et al25 published a prevalence of AHI > 20 of > 40% among the general population aged 71 to 100 years. Interestingly, in our series, the incidence of new VEs among the patients excluded from the study and in whom CPAP was not indicated (AHI < 20, n = 44) was significantly lower than the value calculated for the group with an AHI > 20 who did not tolerate CPAP (n = 36; 19.4% vs 36.8%, p = 0.04), other risk factors being equal. This implies a possible association between untreated SAHS and an increase in the posterior incidence of new VEs. However, within the group excluded from the study in which CPAP was not indicated, the patients with a new VE showed a nonsignificant tendency for more respiratory events (AHI, 13 vs 7; p = 0.12); we therefore consider that the choice of a lower cutoff point for prescribing CPAP may have even further improved the capacity of the latter to protect against new VEs.

The present study has a series of limitations that require comment. A first limitation refers to the system used for the sleep study. While it is true that the Autoset Portable Plus II system has important limitations in the identification of central events, no high incidence of such events is to be expected following stabilization of the acute neurologic event. The Autoset Portable Plus II system has been validated by different authors,25–27 and the best results have been obtained using high cutoff values in the definition of SAHS, with high prevalence of SAHS in the study population. Both characteristics are found in our series of patients (> 50% of the patients presented with an AHI ≥ 20 after stabilization of the neurologic process).

Another limitation of our study refers to the previously mentioned low percentage of patients who tolerated and continued CPAP treatment (n = 15; 29%) during 18 months, thus causing the calculations to lose statistical power. Nevertheless, and taking into account the difficulties reported by all authors in treating these patients with CPAP due to the inherent nature of stroke, we consider that the number of patients obtained has been considerable. In this sense, Hui et al23 were only able to keep 4 of 34 ischemic stroke patients on long-term domiciliary CPAP. In turn, Sandberg et al24 and Wessendorf et al25 were able to achieve short-term (approximately 4 weeks) CPAP tolerances of 50% and 70%, respectively. The posterior domiciliary follow-up figures are not known, although they were likely to have been much lower. In agreement with other series,24,31 the patients who were unable to adapt to CPAP had less hypersomnia, fewer respiratory events, and greater neurologic repercussions of stroke.

Lastly, mention should be made of the fact that we did not use CPAP placebo (sham CPAP), since this option is not available in our center. This obliged us to use as control group those patients who did not tolerate CPAP, adjusting the results for those variables found to be statistically different at the start of the study among the different treatment groups, particularly other VRF and neurologic recovery parameters.

It can be concluded that in our series of patients with ischemic stroke or TIA, the risk of a new VE was greater than expected in those individuals with SAHS not treated with CPAP after 18 months of follow-up. While CPAP compliance was low, the treatment could prove effective in avoiding recurrent VEs. Further studies are needed to confirm these
results and explore a formula for improving adhesion to CPAP therapy among ischemic stroke patients.

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