ORIGINAL ARTICLE
SLEEP

SAS Care 1: sleep-disordered breathing in acute stroke and transient ischaemic attack – prevalence, evolution and association with functional outcome at 3 months, a prospective observational polysomnography study

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ABSTRACT Sleep-disordered breathing (SDB) is frequent in patients with acute stroke. Little is known, however about the evolution of SDB after stroke. Most of our knowledge stems from smaller cohort studies applying limited cardiopulmonary sleep recordings or from cross-sectional data collected in different populations.

This study aims to determine prevalence, type and intra-individual evolution of SDB based on full-night polysomnography (PSG) in acute stroke and 3 months thereafter. Furthermore, we aimed to identify predictors of SDB in the acute and chronic phase and to evaluate associations between SDB and functional outcome at 3 months (M3).

A total of 166 patients with acute cerebrovascular events were evaluated by full PSG at baseline and 105 again at M3. The baseline prevalence of SDB (apnoea–hypopnoea index (AHI)>5·h^{-1}) was 80.5% and 25.4% of the patients had severe SDB (AHI>30·h^{-1}). Obstructive sleep apnoea was more prevalent than central sleep apnoea (83.8% *versus* 13%). Mean±SD AHI was 21.4±17.6·h^{-1}and decreased significantly at M3 (18±16.4·h^{-1}; p=0.018). At M3, 91% of all patients with baseline SDB still had an AHI>5·h^{-1} and in 68.1% the predominant type of SDB remained unchanged (78.9% in obstructive sleep apnoea and 44.4% in central sleep apnoea). The only predictors of SDB at baseline were higher age and body mass index and in the chronic phase additionally baseline AHI. Baseline AHI was associated with functional outcome (modified Rankin score >3) at M3.

The high prevalence of SDB in acute stroke, its persistence after 3 months, and the association with functional outcome supports the recommendation for a rapid SDB screening in stroke patients.

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The high prevalence of SDB in acute stroke, its persistence after 3 months and its association with functional outcome support the recommendation for rapid SDB screening in stroke patients https://bit.ly/3bFWqV7

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Introduction

Over the last decades, an increase in the prevalence of sleep-disordered breathing (SDB) has been reported [1–3]. SDB has been recognised as an important, severity-dependent and potentially modifiable risk factor for all-cause and particularly cardiovascular and cerebrovascular morbidity and mortality [4–8]. In particular, obstructive sleep apnoea (OSA) has been identified as an independent risk factor for acute cerebrovascular events [7–15]. The prevalence of SDB in stroke patients averages at 70% and approximately 30% present with severe SDB (apnoea–hypopnoea index (AHI) >30·h^{-1}) [16]. OSA, the most frequently reported SDB pattern in the context of stroke, and central sleep apnoea (CSA) including Cheyne–Stokes respiration (CSR) are both observed more frequently in patients with incident stroke compared to the general population [10, 12, 17–29]. Accumulating evidence suggest the existence of a bidirectional association between SDB and cerebrovascular diseases; on the one hand, SDB is a severity-dependent risk factor for stroke and for stroke-related unfavourable short-term and long-term outcomes and on the other hand, stroke itself is a cause of new onset SDB or an aggravating factor of pre-existing SDB [6, 30, 34].

Although recent systematic reviews and meta-analyses confirm the high prevalence of SDB in stroke patients and the association with an unfavourable outcome, the comparability of included studies is limited due to a lack of homogeneity in the eligibility criteria for participants, the type of sleep study, and the timing of baseline and follow-up evaluation [13, 16, 31, 32, 35]. Most of our current knowledge on SDB in acute stroke and its evolution following acute stroke derives from cohort studies assessing nocturnal respiration based on different types of portable cardiorespiratory sleep recording equipment [10, 12, 17, 19–24, 36–41]. So far, only a few, smaller studies applied the diagnostic gold standard, full-night polysomnography (PSG) [9, 10, 12, 17, 18, 42, 43] and only two were longitudinal studies with full PSG at follow-up in small cohorts [12, 17].

The main aim of the present study was to determine the prevalence, type and evolution of SDB based on attended full-night PSG recordings in the acute phase of stroke and 3 months thereafter. In addition, we aimed to evaluate associations between type and severity of SDB and sleep-related stroke onset, stroke severity, cardiovascular risk factors, early neurologic worsening, and functional outcome at 3 months.

Methods

Patients

This analysis is based on data from the prospective multinational multicentre SAS-CARE trial (Sleep-Disordered Breathing in Transient Ischemic Attack (TIA)/Ischemic Stroke and Continuous Positive Airway Pressure (CPAP) Treatment Efficacy). Details regarding the design of the SAS-CARE study have been described previously [44]. Briefly, the SAS-CARE cohort includes patients aged between 35 and 75 years with newly diagnosed acute TIA and ischaemic stroke, admitted to a stroke unit within 2 days from onset of symptoms in one of the participating centres (Bern, Switzerland; Lugano, Switzerland; Münster, Germany; Milano, Italy). Exclusion criteria were: 1) unstable clinical condition (cardiorespiratory or life-threatening medical conditions); 2) current CPAP treatment or other SDB treatment during the last 3 months before stroke; 3) nonischaemic events (intracerebral/subarachnoid haemorrhage); 4) coma/stupor; and 5) any condition that may interfere with the acceptance of CPAP treatment. The study (ClinicalTrials.gov identifier: NCT01097967) was approved by the local ethics committees and conducted in accordance with the principles of good clinical practice and local laws. Written informed consent was obtained from all patients prior to participation.

For the evaluation of SDB prevalence at baseline (BL), we considered all patients who underwent nocturnal PSG within the first 7 days after the acute cerebrovascular event. For the analysis of the evolution of SDB and stroke outcome, we included all patients, who received full PSG recordings at BL and after 3 months (M3). Beside PSG parameters, the following information was considered from the SAS-CARE database: demographics (age, sex), body mass index (BMI), medical history (dyslipidaemia,

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diabetes, hypertension, previous cerebrovascular events, smoking and snoring), location and aetiology of stroke based on the criteria of the TOAST study [45], stroke/TIA severity according to the National Institutes of Health stroke scale (NIHSS) [46] at admission, after 24 h and after 3 months. Functional outcome of stroke/TIA was assessed using the modified Rankin (mRankin) scale 3 months following stroke/TIA.

Nocturnal polysomnographic studies

All PSG recordings from Lugano and Bern were registered with EMBLA® Titanium devices (EMBLA, Amsterdam, the Netherlands); the PSG recordings from Milano and Münster were exported in European data format and scored using the same software (EMBLA® Remlogic; EMBLA, Amsterdam, the Netherlands). PSG recordings included six channels of electroencephalogram, submental electromyogram, electro-oculogram, nasal airflow, abdominal and thoracic efforts, pulse-oximetry, bilateral tibialis anterior electromyogram, ECG, position.

All PSG were scored centrally according to the AASM 2012 international criteria [47, 48] by the same investigator (SM), who was blinded to clinical data and timing of the sleep study (BL or M3).

Definition of SDB

Presence of SDB was defined as an AHI >5·h^{-1} of sleep. SDB was classified as mild, moderate and severe if AHI was 5 to <15·h^{-1}, 15 to <30·h^{-1} and ⩾30·h^{-1}, respectively. SDB was considered as OSA if >50% of the respiratory events were of obstructive origin, whereas in the case of >50% of central respiratory events, SDB was classified as central.

Statistical analysis

This exploratory analysis included enrolled patients with a stroke or TIA diagnosis and an evaluable PSG at baseline or M3 and primarily made use of descriptive statistics and univariate statistical tests (unless otherwise specified: Fisher's exact test for categorical variables, Kruskal–Wallis test or Wilcoxon rank-sum test for continuous variables and Wilcoxon signed-rank test for paired continuous data). Multiple regression methods (analysis of covariance, logistic regression) were used to study baseline variables and outcomes associated with SDB.

No imputation of missing values was performed. All statistical tests were two sided and conducted at the 5% significance level, without adjustment for multiplicity.

Results

Sleep-disordered breathing in the acute stroke phase (baseline)

A total of 166 evaluable patients received full PSG within the first 7 days following the acute cerebrovascular event. Most patients had stroke (88.1%) and were males (72.3%) mostly with mild-to-moderate stroke/TIA (mean admission NIHSS 4.3±5.1 (range 0–25)). The mean age was 61.4±9.4 years; awakening stroke or TIA occurred in 26% of patients. For details on demographics and stroke/TIA characteristics see table 1 and figure 1 (distribution of NIHSS).

SDB (AHI >5·h^{-1}) was present in 136 patients (80.5%); and was mild, moderate and severe in 30.2%, 24.9% and 25.4%, respectively. OSA was the most common type of SDB (n=114; 83.8%). Predominant CSA was found in 13% of patients. Of interest, the distribution of SDB severity classified by AHI was different in OSA and CSA patients. SDB was categorised as severe in 26.3% in OSA patients *versus* 59.1% in CSA patients (p=0.009). Accordingly, CSA patients showed a higher AHI (35.6±18 *versus* 24.3±18.2; p=0.02) and ODI (25.3±18.9 *versus* 15.9±15.4; p=0.01) compared to OSA patients. CSR was observed in 36 patients (21.3%). Patients with any kind of SDB were older and weighed more than those with normal respiration and showed a higher percentage of light sleep and arousal index. Interestingly, no differences were found between OSA and CSA patients for all the variables, except for total AHI and AHI during nonrapid eye movement sleep, being highest in CSA patients.

Predictors of SDB in the acute phase of stroke

The only significant predictors of an increased AHI in the acute phase were age and BMI (table 2). No associations were found between baseline AHI and severity of stroke (baseline NIHSS, figure 2a), time spent in the supine position, and wake-up stroke, respectively. Furthermore, we did not find any significant associations between type of SDB and stroke/TIA location (infratentorial stroke *versus* supratentorial stroke). Patients with CSR were older than those without (65.6±7.3 *versus* 60.2±9.6, respectively, p=0.002).

TABLE 1 Demographics, National Institutes of Health stroke scale (NIHSS) and stroke characteristics

	PSG at baseline (n=166)	PSG at both time points (n=105)
Male	72%	79%
BMI kg·m^{-2}	27.7±4.9	27.0±4.9
Stroke	88.1%	92.4%
Known arterial hypertension	55.6%	58.1%
Current smoker	33.6%	29.5%
Current atrial fibrillation	11.2%	9.5%
Diabetes mellitus	14.7%	12.4%
NIHSS at admission	4.3±5.1	4.0±4.5
NIHSS after 24 h	2.9±3.9	2.3±2.9
NIHSS at discharge	1.7±3.1	1.2±2.0
Stroke location		
LACI	13%	13%
TACI	13%	6%
PACI	44%	52%
POCI	24%	25%
No information	6	4
Infratentorial	23.7%	17.1%

Data are presented as mean±SD unless otherwise stated. PSG: polysomnography; BMI: body mass index; LACI: lacunar infarction; TACI: total anterior circulation infarction; PACI: partial anterior circulation infarction; POCI: posterior circulation infarction.

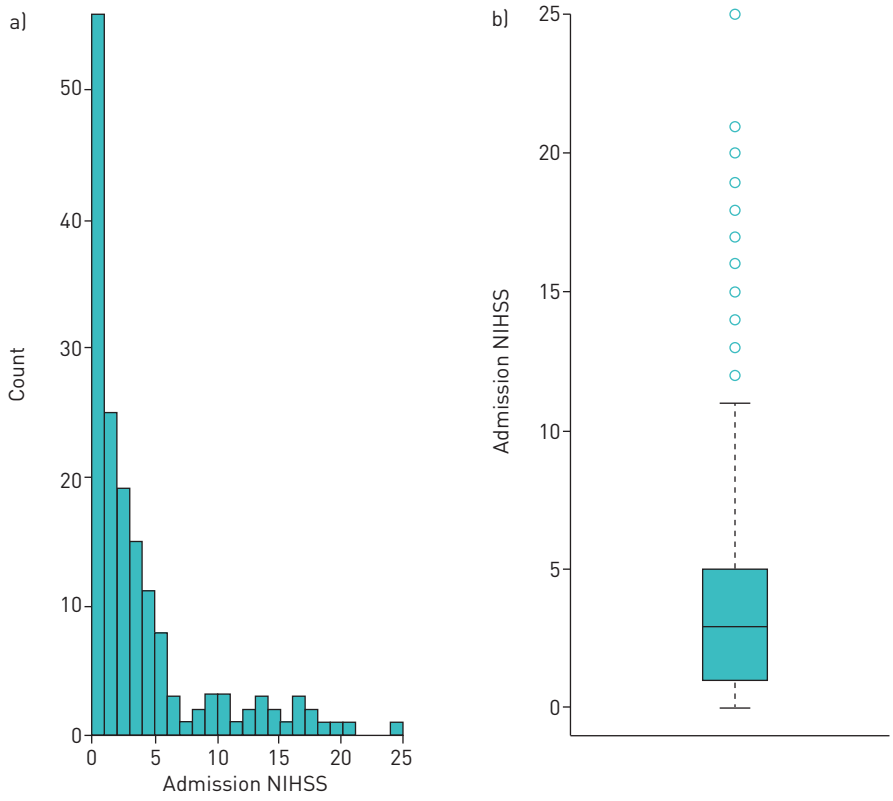


FIGURE 1 Distribution of National Institutes of Health stroke scale (NIHSS) score at admission. a) Histogram; b) box and whisker plot.

TABLE 2 Factors associated with baseline apnoea–hypopnoea index (ANCOVA)

	Coefficient	Standard error	p-value
Intercept	−40.99	16.29	0.013
Age years	0.43	0.16	<0.001
Sex male	2.86	3.35	0.39
BMI kg·m^{-2}	0.99	0.34	0.005
Admission NIHSS	0.24	0.30	0.44
Arteriole hypertension	3.96	3.22	0.22
Smoking >10 years	4.94	3.15	0.12
Time spent in supine position %	0.08	0.05	0.11
Time in bed h	0.21	0.93	0.83
Atrial fibrillation	−1.32	4.92	0.79
Stroke on awakening or during sleep	0.69	3.19	0.83
diabetes mellitus	2.14	4.26	0.61
TOAST: large artery *versus* cardioembolic	−0.523	5.65	0.93
TOAST: other/unknown *versus* cardioembolic	−4.37	3.91	0.27

BMI: body mass index; NIHSS: National Institutes of Health stroke scale. Multiple R^2=0.27, p<0.001 (F statistic 3.727 on 13 and 131 degrees of freedom).

Evolution of SDB from acute stroke to 3 months follow-up

A total of 105 out of the 166 patients evaluable at baseline received follow-up PSG at M3. Demographics as well as stroke/TIA severity and characteristics were comparable with the whole cohort (table 1). Quality of sleep significantly improved at follow-up with an increase in total sleep time and sleep efficiency, less time spent awake after sleep onset, and an increase in S2 sleep. Major PSG parameters that may interfere with the occurrence of SDB, such as time spent in the supine position or the proportion of rapid eye movement (REM) sleep remained unchanged (table 3).

SDB (AHI >5·h^{-1}) was present in 85.6% of patients at baseline and in 82.7% at M3. Moderate and severe SDB were present in 23.1% and 27.9% at baseline and in 27.9% and 17.3% at M3, indicating an overall shift towards less severe SDB at M3 (figure 3). The mean AHI decreased from 21.4±17.6·h^{-1} to 18±16.4·h^{-1} at M3 (p=0.018). The decrease in AHI at M3 was numerically higher in patients which were initially classified as having predominantly central and mixed SDB compared to predominant OSA (9.1±19.9, 8.4±15.0 and 1.9±14.2 events·h^{-1}, respectively; p=0.40), although the number of obstructive apnoeas decreased significantly in the whole cohort, whereas the decrease in central apnoeas (central apnoea index (CAI)) was only by trend (table 3). However, when excluding the seven patients with TIA from analysis, a small but significant decrease was found also in CAI (2.8±7.7·h^{-1} *versus* 1.9±5.8·h^{-1}; p<0.05).

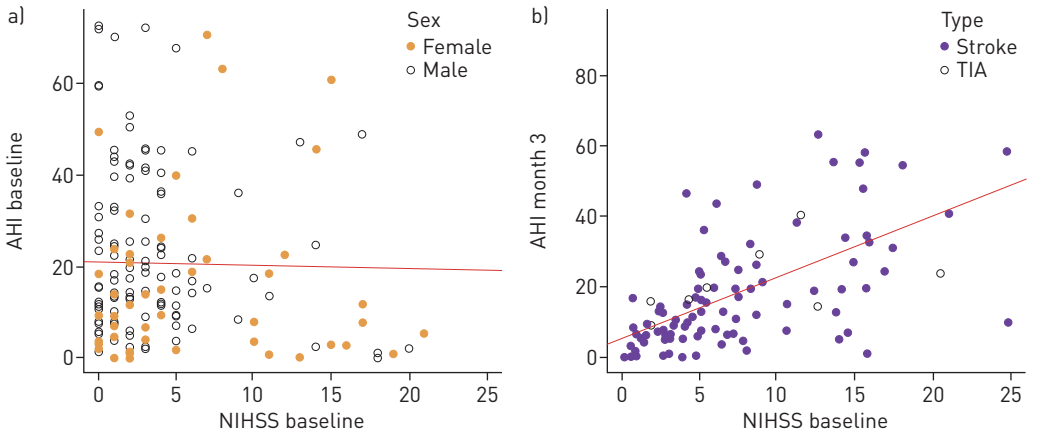


FIGURE 2 Correlations between a) baseline apnoea–hypopnoea index (AHI) and b) AHI at month 3, and admission National Institutes of Health stroke scale (NIHSS). Spearman rank correlation: a) ρ=−0.01, p=0.88; b) ρ=0.64, p<0.001.

TABLE 3 Polysomnography data of patients included in the sleep-disordered breathing evolution analysis (n=105)

PSG parameter	Baseline	3 months	p-value
TST h	5.3±1.5 (5.6)	5.8±1.4 (5.8)	<0.01
Sleep efficiency %	58.9±18.2 (60.1)	66.8±17 (69.6)	<0.001
WASO min	194±103.8 (167)	150.6±86.5 (124.5)	<0.001
WASO %	36.6±17.3 (35.3)	29.6±15.8 (26.7)	<0.001
N1 %	6.8±3.5 (6.2)	7.7±4.1 (7)	0.077
N2 %	27.9±9.8 (26.5)	31.9±10.9 (32.5)	<0.001
N3 %	16.3±7.3 (16.8)	18.1±7.5 (18.1)	0.14
REM %	12±5.7 (11.8)	12.8±5.6 (12.7)	0.24
REM % TST	18.4±6.7 (18.4)	17.6±6.5 (17.6)	0.23
Arousal index events per h	24±12.2 (21.4)	23.3±11.1 (21.8)	0.96
Supine position %	37.2±28.7 (31.1)	44.5±30.9 (44.3)	0.073
AHI events per h	21.4±17,6	18±16.3	0.01
AHI during REM events per h	21.9±19,4	23.4±20,5	NS
AHI during N1–N3 events per h	20,6±18,6	16.9±16.67	0.02
AHI in supine position events per h	32.7±28.5	27.5±24.5	NS
OAI events per h (n=104)	6.3±9.9	3.8±6.7	0.009
CAI events per h (n=104)	2.7±7.5	1.8±5.6	NS
MAI event per h (n=104)	1.5±4.4 (0.1)	0.6±1.7 (0)	0.04
Cheyne–Stokes breathing n (%)	26 (24.8)	18 (17.1)	NS
Mean S_{pO_2} %	92.5±1.9	92.7±2.3	NS
Lowest S_{pO_2} %	86±4.79	85.3±5.69	NS
S_{pO_2} <90% % TST	10.2±22.2	10.9±23.4	NS
ODI events per h	15.1±16	14±15.5	NS

Data are presented as mean±SD (median) or mean±SD, unless otherwise stated. TST: total sleep time; WASO: wake after sleep onset. N: non-REM sleep; REM: rapid eye movement sleep; AHI: apnoea–hypopnoea index; OAI: obstructive apnoea index; CAI: central apnoea index; MAI: mixed apnoea index; S_{pO_2}: oxygen saturation measured by pulse oximetry; ODI: oxygen desaturation index.

OSA was the dominating type of SDB with a prevalence of 79.8% (BL) and 80.2% (M3). CSA was detected in 17.3% at BL and in 16.3% at M3, respectively, whereas CSR occurred in 24.8% at BL and in 17.1% at M3 (p>0.05).

At follow-up, 91% of all patients with BL SDB still had disturbed respiration during sleep (AHI>5·h^{-1}) and in the majority of patients (68.1%) the predominant type of SDB remained unchanged (figure 4). This was particularly true for patients with OSA (78.9%) in comparison with CSA patients (44.4%; p<0.001). At M3, 66.7% of all non-SDB patients still presented undisturbed nocturnal respiration (AHI<5·h^{-1}).

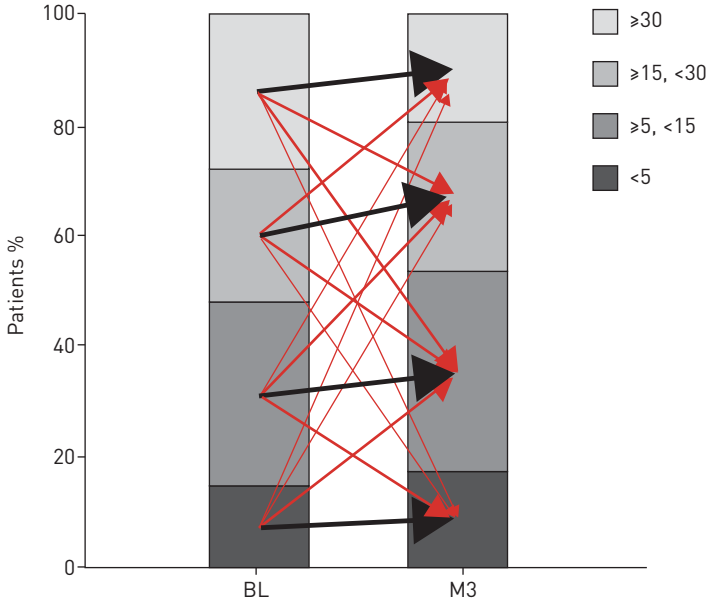
Predictors of AHI after 3 months and association of SDB with functional outcome

In a multiple regression model (similar to table 2), AHI at M3 was significantly associated only with baseline AHI (0.48·h^{-1}, p<0.001; see also figure 2b). A marginally significant association existed for age (0.31·y^{-1}, p=0.076), BMI (0.69 m^2·kg^{-1}, p=0.059).

We found no association between baseline AHI and NIHSS improvement after 24 h. In a multiple linear regression model, only admission NIHSS was significantly associated with mRankin score after 3 months. However, when looking at functional independence (M3 mRankin 0–2 *versus* 3–6), baseline AHI was the only at least marginally significant predictor of mRankin scores >3 at M3 (table 4).

Discussion

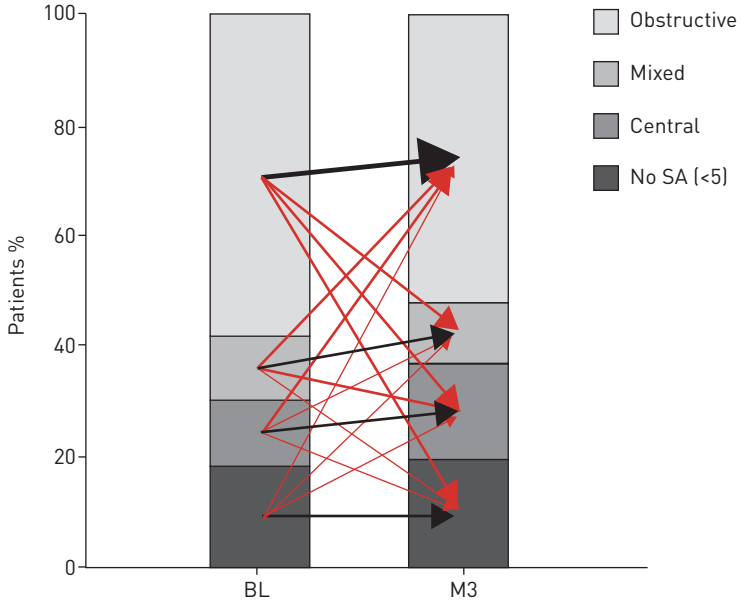
This is the largest prospective full PSG study so far evaluating the prevalence and the longitudinal evolution of SDB in acute stroke and TIA. Only a few studies applied full PSG during the acute phase of stroke in smaller cohorts of up to 93 patients [9, 12, 17, 18, 43, 49]. Thereof, only two studies observed longitudinally the intra-individual evolution of SDB with full PSG re-evaluation 1–6 months after the acute event in up to 33 patients [12, 17]. The largest cohorts so far evaluating the time course of SDB following stroke in 86 patients and 204 patients were published by Parra *et al.* [10] in 2000 and Huhtakangas *et al.* [50] in 2018, evaluating SDB by limited respiratory polygraphy. Our large prospective PSG study clearly confirms the high prevalence of SDB in stroke patients described in earlier cohorts and



		3-months follow-up				
		No-SDB	Mild	Moderate	Severe	Total
Acute phase	No-SDB	10 (66.7%)	4 (26.7%)	1 (6.7%)	0 (0%)	15 (14.4%)
	Mild (AHI 5–<15 per h)	5 (13.9%)	22 (61.1%)	8 (22.2%)	1 (2.8%)	36 (34.6%)
	Moderate (AHI 15–<30 per h)	2 (8.3%)	7 (29.2%)	13 (54.2%)	2 (8.3%)	24 (23.1%)
	Severe (AHI <30 per h)	1 (3.4%)	6 (20.7%)	7 (24.1%)	15 (51.7%)	29 (27.9%)
	Total	18 (17.3%)	39 (37.5)	29 (27.9%)	18 (17.3%)	104

FIGURE 3 Evolution of sleep-disordered breathing (SDB) severity from the acute to the chronic stroke phase (M3). Overall, 104 patients were evaluable. The size of the arrows reflects the number of patients moving between categories (black: same; red: different category). BL: baseline; M3: follow-up after 3 months.

recent meta-analyses [7, 10, 12, 16, 35, 50]. As in most previous studies, OSA is by far the most common type of SDB in this population. Despite the absence of pre-stroke data, the stability of the SDB type, particularly of OSA, and severity at 3 months after the acute event support the hypothesis of pre-existence of SDB and a potential association between SDB and stroke/TIA, as emphasised by others [10, 50]. This is further supported by the lack of an association between stroke severity and AHI, whereas known OSA risk factors (higher age and BMI) were the only predictors of AHI at baseline. As described before, we observed a significant overall decrease in AHI from baseline to the follow-up at 3 months of roughly 15%, which may suggest an aggravation of SDB caused by the acute cerebrovascular condition, or a high level of breathing instability accompanying the typical sleep disruption. A comparable reduction in AHI following stroke/TIA was described by Parra *et al.* [10] and our own group [12]. This decrease was more profound in patients presenting with initial CSA despite the fact that we did not observe a significant decline in CAI, but in obstructive apnoea index (OAI). When excluding TIA patients, we were able to confirm a significant decrease in CAI as described previously [10]. This finding, together with the fact that in the chronic stroke phase only 44% of all patients with initial CSA still exhibit CSA compared to 78.9% of OSA patients with consistent SDB type, supports the hypothesis that the CSA may have been predominantly caused by the acute stroke [40]. In contrast to Parra *et al.* [10], we also observed a significant decline in OAI. The use of full PSG, allowed to exclude changes in sleep architecture between the acute and the chronic phase, particularly the amount of REM sleep and the time actually spent sleeping as well as positional effects. This is of importance when evaluating the longitudinal evolution of SDB following stroke as it has been assumed that patients are more likely to sleep in supine position in the acute phase, particularly when they stay in a stroke unit, which may have contributed to the observed variations in respiratory events. Therefore, our results indicate that OSA may also worsen in the acute phase following a cerebrovascular event, as we observed a significant decrease in OAI, although the time spent in supine position and the amount of REM sleep remained unchanged.



Acute phase		3-months follow-up			
		No-SDB	OSA	CSA	Total
	No-SDB#	10 (66.7%)	4 (26.7%)	1 (6.7%)	15 (14.4%)
	OSA¶	7 (9.9%)	56 (78.9%)	8 (11.3%)	71 (68.3%)
	CSA¶	1 (5.6%)	9 (50%)	8 (44.4%)	18 (17.3%)
	Total	18 (17.3%)	69 (66.3%)	17 (16.3%)	104

FIGURE 4 Evolution of sleep-disordered breathing (SDB) type from baseline to 3 months (M3), individual changes in predominant type of SDB. Overall, 104 patients were evaluable. The size of the arrows reflects the number of patients moving between categories (black: same; red: different category). BL: baseline; M3: follow-up after 3 months; SA: sleep apnoea; OSA: obstructive sleep apnoea; CSA: central sleep apnoea. #: apnoea–hypopnoea index (AHI) <5·h^{-1}; ¶: AHI >5·h^{-1}.

The only predictors of SDB in acute stroke/TIA were higher age and BMI. We found no associations between AHI and severity of stroke (baseline NIHSS) and drop of NIHSS within 24 h, which is in line with other studies. In addition, as in other studies, we identified AHI as a potential predictor of higher disability (mRankin ⩾3) in chronic stroke [31].

There are some limitations that need to be mentioned. 1) baseline PSGs were performed within 1 week after the acute event, not during the night directly following stroke onset or always on the same night after stroke. Therefore, it might be possible that hyperacute effects of stroke on nocturnal respiration could have

TABLE 4 Predictors of unfavourable neurological outcome (modified Rankin score >3) in logistic regression model (dichotomised modified Rankin 0–2 *versus* 3–6)

	OR	95% CI	p-value
Age years	0.900	0.770–1.040	0.11
Sex male	0.377	0.038–3.718	0.53
BMI kg·m^{-2}	0.904	0.719–1.138	0.24
Admission NIHSS	1.188	0.940–1.500	0.23
Hypertension	0.238	0.011–5.383	0.22
Atrial fibrillation	0.000	0.000–∞	1.00
Stroke on awakening or during sleep (*versus* no or unknown)	15.699	0.726–339	0.10
Infratentorial stroke (*versus* other or unknown)	0.216	0.006–7.566	0.40
Cheyne–Stokes respiration	0	0–∞	0.99
Baseline AHI	1.107	1.010–1.214	0.052

BMI: body mass index; NIHSS: National Institute of Health stroke scale; AHI: apnoea–hypopnoea index.

been missed. 2) Duration of follow-up was 3 months, not allowing any conclusions on longer-term outcomes to be drawn (*i.e.* after 12 months). 3) Despite the prospective nature of our study there were some missing data; imputation was not applied. 4) The majority of our patients had mild stroke which may be an explanation for the modest overall effects observed in our study. 5) The analysis was exploratory and some of the presented findings are based on data-driven evaluations of selected aspects. Furthermore, the power for the detection of predictors of specific effects, such as stroke outcomes, was most likely very limited. 6) Echocardiographic data (*i.e.* ejection fraction), were not collected systematically, preventing an analysis of the impact of stroke/TIA on central respiratory effects, as concomitant congestive heart failure may have been present is some patients.

In conclusion, our results confirm the high prevalence of SDB both in the first week and at 3 months after acute stroke/TIA and supports the assumption of a causal association between pre-existing SDB, in particular OSA, and stroke. Furthermore, we found a high rate of persistence of SDB type and severity, particularly in OSA, and a possible association between AHI and functional outcome after 3 months. Considering the increasing evidence of a favourable effect of CPAP in stroke patients with SDB [6], results support the recommendation for a rapid screening for SDB in this clinical setting.

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