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Short- and long-term continuous positive airway pressure usage in the post-stroke population with obstructive sleep apnea

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Abstract

Purpose Obstructive sleep apnea (OSA) is highly prevalent in post-stroke patients and observational evidence suggests that untreated it is a harbinger of poorer outcomes in this population. Clinical trials on the impact of treatment of OSA with continuous positive airway pressure (CPAP) have countered difficulties with patient engagement and adherence to CPAP therapy. Real-world data on continuous positive airway pressure initiation and usage in the post-stroke population with obstructive sleep apnea is limited.

Methods We performed a clinical retrospective study between January 2006 and June 2015 to describe the short- and long-term CPAP usage in the post-stroke population with OSA, and to assess which patient, disease, and treatment-related factors were associated with CPAP purchase, initiation, and usage in this population.

Results Of 191 post-stroke patients' recommended CPAP therapy, post-prescription usage at 3, 6, 12, 24, and 60 months was 58%, 53%, 48%, 45%, and 39% respectively. OSA severity-related factors, such as AHI or degree of nocturnal hypoxemia, were not significantly associated with CPAP usage. Predictors of CPAP usage at all time points were younger age, male sex, never smokers, and no history of hypertension. There were some differences in predictors of CPAP usage between early and later time periods.

Conclusions We demonstrated that the long-term usage of CPAP therapy is possible with most of the attrition occurring in the first 3 months. Upfront healthcare resource allocation to CPAP initiation and usage in this population may improve longer-term usage.

Keywords Obstructive sleep apnea · Continuous positive airway pressure · Stroke · Adherence · Usage

Abbreviations

AHI	apnea-hypopnea index
BMI	body mass index
CCI	Charlson comorbidity index
CPAP	continuous positive airway pressure
ESS	Epworth sleepiness scale
IQR	interquartile range
ODI	oxygen desaturation index
OSA	obstructive sleep apnea
PSG	polysomnogram
SpO_2	peripheral arterial oxygen saturation
UHN	University Health Network

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Introduction

Continuous positive airway pressure (CPAP) is currently the gold standard treatment for obstructive sleep apnea (OSA). The adaptation to CPAP therapy for OSA treatment requires understanding of the disease and significant ongoing commitment by the patient. Regrettably, alternative therapies for the treatment of OSA are not or as effective as CPAP or readily accessible in some jurisdictions [1]. Untreated OSA is associated with significant healthcare costs [2] and adverse health

consequences to the individual, including an increased risk of incident stroke [3]. Conversely, the treatment of OSA with CPAP positively impacts quality of life and reduces daytime sleepiness [4].

Despite the acknowledged treatment benefits of CPAP therapy, the acceptance and adherence to CPAP therapy in the general population is often suboptimal and over the last 15 years has remained static at approximately 34.1% [5]. For the patient who has endured a stroke, this may be an even greater challenge. The physical and cognitive limitations and the associated psychological distress post-stroke provide additional hurdles that must be overcome by patients following a stroke. The prolonged lifespan of humans in the western world and improved medical treatment and care has positively impacted survival post-stroke [6]. With this increased lifespan comes an increase in comorbidities including OSA. This post-stroke population has a high incidence of OSA at approximately 70% [7–9]. Furthermore, untreated OSA in the post-stroke population may predispose to an increased risk of recurrent stroke [10] and poorer functional and neurocognitive outcomes [11]. In small studies, CPAP has been shown to improve both functional and neurocognitive outcomes in this population [9] although, the efficacy of CPAP in improving cardio or cerebrovascular outcomes is as of yet unproven [10].

Observational and randomized trials in the stroke population evaluating CPAP efficacy and effectiveness have been hindered by low acceptance and adherence, particularly when CPAP is not started in the acute or subacute phase of stroke [7, 9, 12]. The inference from these studies is that successful and sustained treatment of OSA in the general post-stroke population is likely to be fraught with difficulty and have a low probability of successful implementation and uptake. While randomized controlled trials have shown higher than expected short-term adherence, this may not reflect real-world outcomes [12]. There is very little data on the acceptance of, and long-term adherence to CPAP in the real-world stroke population [9, 13].

In light of the results from previous studies, we hypothesized that CPAP usage would occur in less than 40% of the post-stroke population. Furthermore, we hypothesized that individuals with more severe OSA, less stroke-related deficits, and family support were more likely to accept and use CPAP. Therefore, the research objectives of our study were (1) to describe the short (< 6 months) and long-term (≥ 6 months) CPAP usage and (2) to evaluate which patient, disease, and treatment-related factors were associated with PAP purchase, initiation, and usage in the post-stroke population with OSA.

Methods

Study setting and population

This was a single-center retrospective clinical chart review study, which included post-stroke adults referred to large urban academic center, the University Health Network (UHN) Sleep Disorders Clinic (Toronto, Canada) from January 1, 2006, to June 30, 2015. Eligible patients were (1) ≥ 18 years of age, (2) had an ischemic or hemorrhagic stroke confirmed by a neurologist with consistent findings on computerized tomography or magnetic resonance imaging [14], and (3) had OSA diagnosed post-stroke on an overnight sleep study with an apnea-hypopnea index (AHI) ≥ 5 events/h with symptoms or ≥ 15 events/h without symptoms [15]. Exclusion criteria included (1) previously diagnosed OSA on therapy,

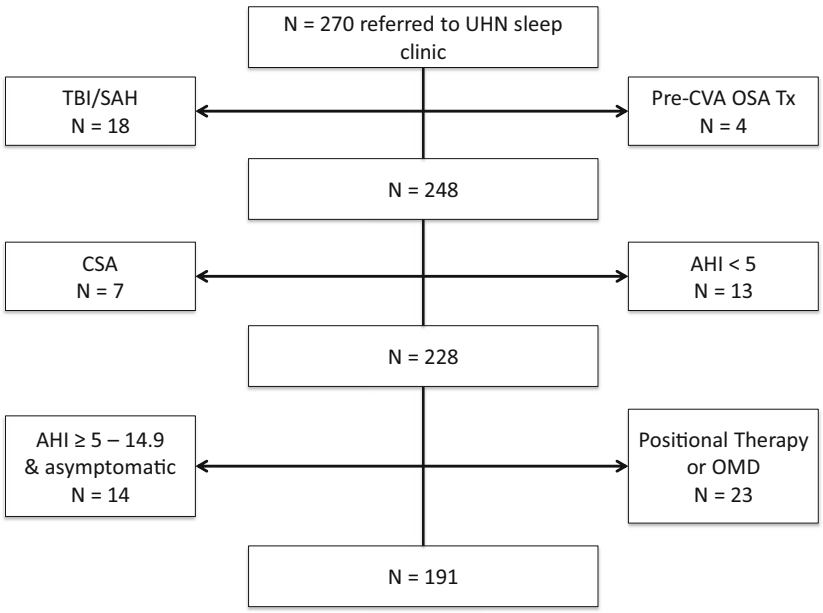

Fig. 1 Flow diagram. This diagram illustrates the flow of patients and the indications for exclusion of referred stroke patients from the analyzed cohort. TBI, traumatic brain injury; SAH, subarachnoid hemorrhage; Pre-CVA OSA Tx, on treatment for obstructive sleep apnea prior to the index stroke; CSA, central sleep apnea; AHI, apnea-hypopnea index; OMD, oral mandibular device

Table 1 Characteristics of post-stroke cohort with obstructive sleep apnea ($n = 191$)

	Total sample $N = 191$	At least 3 months on CPAP		P value	At least 6 months on CPAP		P value
		Yes ($n = 111$)	No ($n = 80$)		Yes ($n = 101$)	No ($n = 90$)	
Demographics							
Sex, male, *n* (%)	138 (72.3)	88 (79.3)	50 (62.5)	0.02*	80 (79.2)	58 (64.4)	0.03*
Age, median (IQR)	64.0 (56.5–73.0)	63.0 (55.0–70.0)	66.0 (60.0–77.0)	0.01*	63.0 (55.0–70.0)	66.0 (60.0–76.8)	0.01*
BMI, median (IQR)	27.5 (24.4–32.1)	28.0 (24.8–32.1)	26.5 (24.1–32.4)	0.18	28.0 (24.7–31.5)	27.0 (24.1–32.7)	0.59
Ever smoke, *n* (%)	66 (34.6)	33 (29.7)	33 (41.25)	0.13	27 (26.7)	39 (43.3)	0.02*
Family support, Yes v No (%)#	142 (74.3)	83 (74.8)	59 (73.8)	1.00	77 (76.2)	65 (72.2)	0.64
ESS, median (IQR)	6.0 (4.0–9.3)	5.0 (4.0–9.0)	7.0 (4.0–10.8)	0.13	5.0 (4.0–9.0)	6.5 (4.0–10.8)	0.06
Comorbidities, *n* (%)							
HTN	163 (85.3)	89 (80.2)	74 (92.5)	0.03*	80 (79.2)	83 (92.2)	0.02*
Diabetes	65 (34.0)	35 (31.5)	30 (37.5)	0.48	31 (30.7)	34 (37.8)	0.38
IHD	33 (17.3)	20 (18.0)	13 (16.2)	0.90	17 (16.8)	16 (17.8)	1.00
Cardiac arrhythmias	35 (18.3)	21 (18.9)	14 (17.5)	0.95	21 (20.8)	14 (15.6)	0.46
Prior stroke	18 (9.5)	8 (7.2)	10 (12.7)	0.31	8 (7.9)	10 (11.2)	0.60
Psychiatric history	48 (25.1)	28 (25.2)	20 (25.0)	1.00	23 (22.8)	25 (27.8)	0.53
No. comorbidities ≥ 3, Yes	51 (26.7)	26 (23.4)	25 (31.3)	0.30	23 (22.8)	28 (31.1)	0.26
CCI, age-adjusted, median (IQR)	4 (3–6)	4 (3–6)	5 (4–6)	0.36	4 (3–6)	5 (4–6)	0.35
PSG characteristics, median (IQR)							
TST, hours	5.1 (4.0–5.9)	5.3 (4.1–6.1)	4.8 (3.9–5.6)	0.15	5.3 (4.0–6.2)	4.8 (4.0–5.6)	0.17
SE, %	73.2 (58.6–82.0)	75.4 (58.9–84.2)	69.6 (58.5–79.4)	0.15	75.6 (59.2–84.4)	70.5 (58.2–79.4)	0.11
Sleep latency, minutes	10.8 (5.4–24.9)	9.0 (4.8–32.1)	11.1 (5.9–23.0)	0.89	8.4 (4.8–28.2)	11.4 (6.0–24.0)	0.37
AHI, events/h	31.8 (21.0–45.2)	31.7 (21.8–46.6)	32.9 (19.6–42.8)	0.85	33.0 (21.7–46.9)	31.8 (19.6–42.6)	0.59
OAHI, events/h	24.9 (15.2–38.0)	25.2 (16.9–38.3)	24.9 (14.7–37.5)	0.75	24.6 (17.1–38.6)	25.4 (13.7–37.3)	0.62
CAHI, events/h	1.20 (0.20–3.80)	1.25 (0.30–4.40)	1.00 (0.20–2.85)	0.36	1.3 (0.3–4.6)	1.0 (0.2–2.8)	0.22
ArI, events/h	27.3 (18.4–40.1)	26.6 (17.6–39.0)	28.4 (20.0–40.7)	0.24	26.7 (17.9–39.7)	27.8 (19.7–40.6)	0.42
Stage N1, %	9.7 (4.6–16.5)	8.9 (5.2–15.9)	10.5 (3.3–19.0)	0.62	9.5 (5.5–16.0)	9.7 (3.2–17.4)	0.88
Stage N2, %	60.7 (52.8–68.7)	63.4 (55.1–70.4)	57.5 (48.0–65.0)	<0.01*	63.4 (55.3–69.9)	57.7 (49.3–67.3)	0.02*
Stage N3, %	12.0 (3.3–18.6)	10.9 (3.9–16.8)	12.4 (1.7–22.1)	0.35	10.9 (4.3–16.8)	12.3 (1.3–22.2)	0.47
Stage REM, %	16.0 (9.6–21.2)	16.3 (9.8–21.2)	15.8 (9.6–21.2)	0.85	16.3 (10.4–21.1)	15.8 (9.6–21.9)	0.91
Min SpO_2, %	82 (73–87)	81.5 (74.0–86.0)	82.7 (71.0–88.3)	0.92	82.0 (73.6–86.0)	82.2 (73.0–88.0)	0.84
Mean SpO_2, %	94.9 (93.4–96.2)	94.6 (93.3–95.9)	95.0 (93.7–96.6)	0.11	94.8 (93.4–95.9)	94.9 (93.5–96.6)	0.40

*p values less than 0.05; #self-reported family support: Yes or No

CPAP, continuous positive airway pressure; *n*, number; *CCI*, Charlson Comorbidity Index, *IQR*, interquartile range; *BMI*, body mass index; *ESS*, Epworth sleepiness scale; *HTN*, hypertension; *IHD*, ischemic heart disease; *No. comorbidities*, number of medical comorbidities; *PSG*, polysomnogram; *TST*, total sleep time; *SE*, sleep efficiency; *AHI*, apnea-hypopnea index; *OAHI*, obstructive apnea-hypopnea index; *CAHI*, central apnea-hypopnea index; *Arl*, arousal index; *Min*, minimum; *SpO_2*, peripheral arterial oxygen saturation

(2) traumatic brain injury, subarachnoid hemorrhage, or ruptured brain aneurysm, and (3) patients in whom either positional therapy or an oral mandibular device was recommended by a sleep physician. Ischemic stroke type was classified according to the TOAST classification [16]. Eligible participants were retrospectively followed from their diagnostic sleep study until lost to follow-up or until June 30th, 2017, whichever occurred first. Patients who failed to attend the sleep clinic during the follow-up period were retained for the purposes of analysis and were deemed non-adherent to PAP.

The study was approved by the Research Ethics Board of the UHN (REB-15-9511-BE).

Polysomnography

Subjects underwent overnight polysomnography (PSG) at the UHN sleep laboratory using standard techniques. PSG signals were recorded on a computerized sleep scoring system (Sandman, Natus Medical Inc., Middleton, USA). Thoraco-abdominal movements and tidal volume were measured by

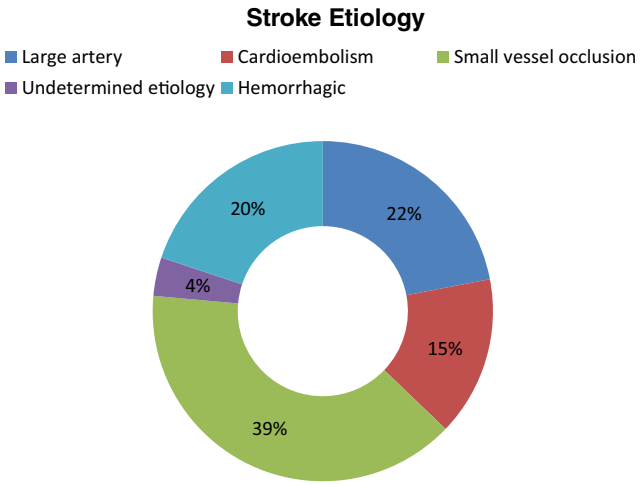

Fig. 2 Stroke etiology. This figure illustrates the etiology of the stroke in the subjects according to the TOAST classification

respiratory inductance plethysmography. Airflow was measured by nasal pressure cannulae and peripheral arterial oxyhemoglobin saturation (SpO_2) by finger oximetry. Apneas were scored as ≥ 90% reduction of tidal volume for ≥ 10 s. Hypopneas were scored as ≥ 30% reduction in tidal volume from baseline for ≥ 10 s on nasal pressure signal with an arousal and/or oxygen desaturation ≥ 3% [17]. Apneas and hypopneas were scored as central or obstructive in the absence or presence of out-of-phase thoraco-abdominal motion. The severity of OSA was determined by the number of apneas and hypopneas per hour of sleep. For the purposes of the study, OSA was defined as AHI ≥ 5 events/h of sleep and ≥ 50% of the events obstructive as was standard clinical practice during the study period. The Epworth sleepiness scale (ESS), a subjective measure of sleepiness, was completed on the night of the PSG [18].

Outcomes: CPAP purchase, initiation, and usage

The primary outcome was usage of CPAP at 3 months (yes/no) post-prescription. Our secondary outcomes were CPAP purchase, initiation, and usage since prescription at 6, 12, 24, and 36 months as yes/no variables. Details on CPAP purchase, initiation, and usage were extracted from the clinical charts of eligible participants. Patients that initiate CPAP were defined by those with proof of purchase of CPAP through home CPAP receipt and initiation provided either through patient self-report or CPAP downloads in clinic follow-up. Adherence to CPAP, if available, was determined by documented patient-reported (hours of use, % of time) or objective CPAP data at 3 months since prescription, and at the last available follow-up period.

Factors associated with CPAP purchase, initiation, and usage

The following groups of pre-specified factors potentially related to CPAP purchase, initiation, and usage were considered: (i) patient-related, (ii) stroke-related, (iii) OSA-related, and (iv) CPAP treatment-related. Patient-related variables included sex, age, English speaking, smoking status, the presence or absence of family support, comorbidities at baseline (e.g., hypertension, hyperlipidemia, type 2 diabetes, cardiovascular disease, physician-diagnosed psychiatric disease, a history of other stroke(s) more than 1 year before the PSG and the age-adjusted Charlson Comorbidity Index (CCI) [19]). The CCI is a method to measure burden of disease and has been validated for its ability to determine mortality in stroke [20]. Stroke-related factors included stroke type, neurological impairment as measured by stroke deficits including paresis, aphasia, and stroke, and time from stroke to OSA diagnosis. OSA-related factors included severity of sleep-disordered breathing measured by AHI and oxygen desaturation, sleep quality (i.e., sleep efficiency, total sleep time, arousal index), and the ESS score. CPAP-related factors included CPAP machine model, CPAP interface, CPAP pressure, in-laboratory versus auto-CPAP titration.

Statistical analysis

Primary analyses

Descriptive statistics were used to characterize our population of interest, and to address our first objective—to describe CPAP usage in the post-stroke population with OSA. Differences between CPAP users and non-users was assessed in continuous data using *t* test or Mann-Whitney tests as appropriate for parametric or nonparametric parameters, respectively, and in categorical data using chi-squared tests. A *p*

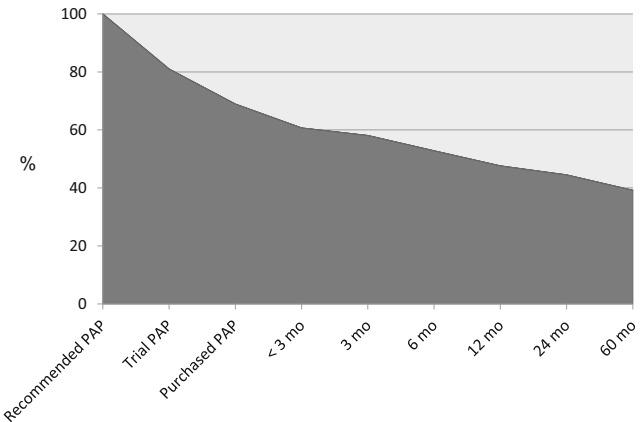

Fig. 3 Continuous positive airway pressure attrition over time in stroke subjects. This graph illustrates the percentage of stroke subjects ($n = 191$, 100%) who were initially recommended CPAP and the subsequent usage of therapy up to 60 months in those who were alive at each time point

Table 2 Subjective and objective continuous positive airway pressure usage at 3 months and at the last available follow-up*

	CPAP hours/night	CPAP % total nights used
CPAP usage at 3 months, median (IQR)		
Combined subjective and objective	6 (4.0–7.0)	90 (60–100)
Objective ($N = 40$)	5.3 (2.4–7.0)	94 (57–100)
Subjective ($N = 61$)	6.0 (4.0–7.0)	90 (71–100)
CPAP usage at the last available follow-up, median (IQR)		
Combined subjective and objective	5.1 (1.5–7.0)	90 (8–98)
Objective ($N = 29$)	5.4 (3.0–7.3)	98 (60–100)
Subjective ($N = 49$)	5.2 (1.5–6.5)	90 (5–95)

*The last available clinic visit is that prior to the censor date (June 30th, 2017) or the last documented clinic attendance by the patient

CPAP, continuous positive airway pressure; *IQR*, interquartile range

value < 0.05 was considered statistically significant for a two-sided test.

The combination of self-report and objective data on CPAP use presented at different time points and loss of follow-up for some individuals prevented us from incorporating time in our main analytic approach. As such, multivariate logistic regression models were used to assess our second objective—the ability of hypothesized variables to predict CPAP purchase, acceptance, and usage at 3 and 6 months and later time points. To simplify and unify presentation across different outcome of interest, we included in the statistical model variables selected using stepwise regression for at least one outcome. We used R^2 and C-statistic to evaluate the performance of our statistical models.

Secondary analyses

Cox regression analyses were used to evaluate an association between potential predictors and CPAP initiation. In this analysis, participants were followed from their diagnostic sleep study to the end of June 2017, or the date of CPAP initiation or all-cause death, whichever occurred first.

Analyses were conducted using R 2.15.1 [21].

Results

Cohort description

Among 270 patients initially identified as having a post-stroke referral to the sleep clinic, 79 patients did not meet our inclusion criteria and therefore, were excluded from the analysis (Fig. 1). The included 191 individuals were predominantly male with a median age of 64 years, overweight and not subjectively sleepy as assessed by the Epworth sleepiness score (Table 1). The majority of the subjects (85.3%) had comorbid hypertension at time of stroke and 26.7% had at least 3 or more medical comorbidities (Table 1). The stroke etiology was predominantly ischemic in nature (76.5%, Fig. 2). A median time from the stroke to assessment with a PSG was 93 days (IQR 48–206 days). The median time from the initial clinic visit to the end of follow-up was 76 months (IQR 51–98 months).

Objective 1: description of CPAP purchase, initiation, and usage in post-stroke cohort with OSA

Of the 191 patients included in this analysis and recommended CPAP by their sleep physician, 81% of patients had either an in-laboratory trial or a home trial of auto-CPAP for up to 1 month's duration. Of those who underwent a trial of CPAP therapy, 84.5% purchased a fixed CPAP device with heated humidification. Despite purchasing a home CPAP device, 11% ($n = 15$) of this group used it for less than 3 months. Therefore, continued CPAP usage at 3, 6, 12, 24, and 60 months was 58%, 53%, 47.6%, 44.5%, and 39% respectively (Fig. 3). The median (IQR) of CPAP pressure was 8 (7–10) cm H_2O for all users and a full-face interface was used in a small percentage of subjects (17%). The available patient-reported and CPAP-downloaded adherence at 3 months and at the final sleep clinic follow-up visit were similar (Table 2). Of the patients who were adherent at 6 and 12 months, 84% and 92% respectively, had reported longer-term adherence.

Table 3 Factors associated with continuous positive airway pressure purchase, initiation, and usage in the post-stroke cohort with obstructive sleep apnea

	Purchased CPAP Machine $N = 131$	Initiation of CPAP treatment (acceptance) $N = 116$	Using CPAP for at least 3 months $N = 111$	Using CPAP for at least 6 months $N = 101$
	Odds ratios (95% confidence interval), *p* value			
Obstructive sleep apnea-related variables*				
SE, %: 82 vs. 59 (IQR)	1.09 (0.67–1.78), 0.72	1.06 (0.68–1.65), 0.81	1.14 (0.73–1.79), 0.56	1.13 (0.73–1.75), 0.59
AHI: 45 vs 21	1.06 (0.63–1.78), 0.83	1.05 (0.66–1.66), 0.85	1.04 (0.65–1.64), 0.88	1.19 (0.75–1.89), 0.46
OAHI: 38 vs 15	1.16 (0.68–2.00), 0.59	1.19 (0.73–1.95), 0.49	1.08 (0.67–1.75), 0.75	1.24 (0.76–2.03), 0.39
SAHI: 52 vs 22	1.19 (0.74–1.92), 0.48	1.33 (0.86–2.05), 0.20	1.28 (0.83–1.96), 0.27	1.44 (0.93–2.21), 0.10
ArI: 40 vs 18	1.10 (0.65–1.87), 0.72	0.86 (0.54–1.36), 0.52	0.78 (0.49–1.24), 0.30	0.93 (0.59–1.47), 0.75
MeanSpO_2: 93 vs 96	*1.79 (1.01–3.19), < 0.05*	1.32 (0.80–2.19), 0.28	1.41 (0.84–2.34), 0.19	1.20 (0.74–1.96), 0.45
MinSpO_2: 87 vs 73	0.90 (0.57–1.43), 0.67	1.38 (0.88–2.17), 0.16	1.42 (0.90–2.25), 0.13	1.27 (0.81–1.97), 0.29
ESS: 4 vs 9	0.90 (0.52–1.55), 0.71	1.33 (0.84–2.10), 0.22	1.53 (0.96–2.43), 0.07	*1.73 (1.08–2.76), 0.02*
Predictors selected to be included in the final statistical model				
Age: 57 vs 73	*4.71 (2.02–10.97), < 0.01*	*3.80 (1.80–8.02), < 0.01*	*4.29 (2.03–9.09), < 0.01*	*3.62 (1.75–7.48), < 0.01*
Sex: M vs F	*3.74 (1.62–8.63), < 0.01*	*2.41 (1.12–5.18), 0.02*	*2.64 (1.22–5.72), 0.01*	*2.32 (1.09–4.96), 0.03*
BMI: 32 vs 24	1.28 (0.79–2.09), 0.32	1.21 (0.77–1.90), 0.40	1.24 (0.79–1.95), 0.35	1.13 (0.73–1.75), 0.60
Ever smoke: No vs Yes	*4.14 (1.84–9.33), < 0.01*	*2.68 (1.31–5.49), < 0.01*	*2.55 (1.24–5.24), 0.01*	*3.24 (1.58–6.63), < 0.01*
No. deficits: 0 vs 1+	*3.36 (1.21–9.31), 0.02*	*2.56 (1.05–6.24), 0.04*	*3.34 (1.35–8.28), < 0.01*	1.80 (0.76–4.25), 0.18
Cardiac Hx: Yes vs No	1.76 (0.77–4.07), 0.18	1.18 (0.56–2.48), 0.67	1.38 (0.65–2.93), 0.40	1.62 (0.75–3.49), 0.28
Hypertension: No vs Yes	2.49 (0.80–7.74), 0.12	2.72 (0.97–7.62), 0.06	*3.18 (1.13–8.94), 0.03*	*3.14 (1.16–8.48), 0.02*
CCI: 6 vs 3	*4.12 (1.39–12.25), 0.01*	*3.66 (1.38–9.73), < 0.01*	*4.37 (1.62–11.73), < 0.01*	*3.13 (1.23–7.97), 0.02*
Family support: Yes vs No	*2.52 (1.09–5.85), 0.03*	1.54 (0.71–3.34), 0.27	1.36 (0.62–2.97), 0.45	1.62 (0.75–3.49), 0.22
Model fit (without OSA-related variables)	$R^2 = 0.30$, C = 0.79	$R^2 = 0.22$, C = 0.74	$R^2 = 0.25$, C = 0.76	$R^2 = 0.23$, C = 0.74
Model fit (with OSA-related variables)	+ mean SaO_2 $R^2 = 0.32$, C = 0.80, $p < 0.05$			+ ESS $R^2 = 0.28$, C = 0.76, $p = 0.02$

Estimates are presented as odds ratios and 95% confidence interval: An odds ratio greater than one indicates higher odds of the outcome occurring; an odds ratio of less than one indicates lower odds of the outcome occurring. Statistically significant values are in italics

*The effect of each obstructive sleep apnea-related variable was tested separately in the statistical model controlling for sex, age, body mass index, smoking status, history of prior hypertension, and cardiovascular disease, No. deficits, CCI and family support

SE, sleep efficiency; *vs*, versus; *IQR*, interquartile range; *AHI*, apnea-hypopnea index; *OAHI*, obstructive apnea-hypopnea index; *SAHI*, supine apnea-hypopnea index; *Arl*, arousal index; *SpO_2*, peripheral arterial oxygen saturation; *ESS*, Epworth sleepiness scale; *BMI*, body mass index; *No. deficits*, number of stroke deficits; *CCI*: Charlson Comorbidity Index; *OSA*, obstructive sleep apnea

Objective 2: factors associated with CPAP purchase, initiation, and usage

Most PSG variables were poorly predictive of the purchase and usage of CPAP therapy (Tables 3 and 4, Fig. 4). Patients with a higher baseline ESS (9 vs 4) were less likely to use CPAP at 3 to 36 months and those with a higher mean oxygen saturation were less likely to purchase a CPAP. There was no difference in long-term adherence based on PAP pressure, interface or in-laboratory versus in a home Auto-CPAP trial.

Multivariable logistic regression showed that individuals of younger age, never smokers, and males with absence of hypertension were more likely to use CPAP both over the short (3 and 6 months) and the long term (up to 60 months). The presence of more medical comorbidities was a predictor of CPAP use up to 12 months. However, there was no relationship with the presence or absence of other medical comorbidities (e.g., diabetes, cardiovascular disease, psychiatric disease). Family support predicted both the purchase of CPAP and the longer-term usage after 12 months. Stroke-related factors (time from stroke, stroke etiology or impairment, and prior strokes) were not associated with CPAP purchase or usage.

We confirmed predictors of CPAP initiation using Cox regression analysis (Table 5).

Table 4 Factors associated with long-term continuous positive airway pressure usage in the post-stroke cohort with obstructive sleep apnea as identified in logistic regression analyses

	CPAP use at 12 months $N = 91$	CPAP use at 24 months $N = 80$	CPAP use at 36 months $N = 77$	CPAP use at last follow-up $n = 67$
		OR (95% CI)		
Sleep apnea-related variables*				
SE, %: 82 vs. 59 (IQR)	1.19 (0.76–1.85)	1.17 (0.74–1.84)	1.19 (0.75–1.88)	1.29 (0.80–2.09)
AHI: 45 vs 21	1.07 (0.68–1.67)	1.19 (0.75–1.88)	1.15 (0.72–1.84)	1.05 (0.65–1.68)
OAHI: 38 vs 15	1.01 (0.63–1.61)	1.05 (0.65–1.69)	1.01 (0.62–1.63)	1.05 (0.64–1.70)
SAHI: 52 vs 22	1.35 (0.89–2.04)	1.42 (0.93–2.16)	1.34 (0.88–2.05)	1.11 (0.74–1.68)
ArI: 40 vs 18	0.82 (0.52–1.28)	0.81 (0.51–1.28)	0.82 (0.52–1.31)	0.64 (0.39–1.04)
MeanSaO_2: 96 vs 93	0.86 (0.54–1.38)	0.84 (0.52–1.35)	0.81 (0.50–1.31)	0.88 (0.54–1.44)
MinSaO_2: 87 vs 73	1.52 (0.96–2.41)	1.27 (0.80–2.01)	1.26 (0.79–2.00)	1.04 (0.65–1.65)
ESS: 9 vs 4	*0.62 (0.40–0.98)*	*0.56 (0.35–0.90)*	*0.58 (0.36–0.94)*	0.67 (0.42–1.07)
Variables included in the statistical model without sleep apnea-related variables				
Age: 57 vs 73	*2.99 (1.49–5.99)*	*2.63 (1.31–5.29)*	*3.12 (1.52–6.40)*	*2.85 (1.39–5.83)*
Sex: M vs F	*2.28 (1.07–4.86)*	1.97 (0.91–4.28)	2.00 (0.91–4.40)	*2.52 (1.09–5.83)*
BMI: 32 vs 24	1.07 (0.69–1.65)	1.06 (0.68–1.65)	1.01 (0.64–1.59)	1.20 (0.75–1.92)
Ever Smoke: No vs Yes	*2.92 (1.44–5.91)*	*3.28 (1.59–6.76)*	*3.42 (1.64–7.15)*	*2.42 (1.16–5.06)*
No. Deficits: 0 vs 1+	2.10 (0.90–4.90)	1.62 (0.69–3.78)	1.38 (0.58–3.25)	1.56 (0.65–3.73)
Cardiac Hx: Yes vs No	1.42 (0.69–2.93)	1.20 (0.58–2.51)	1.21 (0.57–2.54)	1.28 (0.60–2.74)
Hypertension: No vs Yes	*2.73 (1.07–6.92)*	*2.65 (1.05–6.66)*	*2.79 (1.10–7.06)*	*3.25 (1.29–8.17)*
CCI: 6 vs 3	*2.56 (1.04–6.30)*	1.92 (0.78–4.69)	1.98 (0.80–4.89)	2.13 (0.85–5.32)
Family Support: Yes vs No	1.92 (0.89–4.14)	*2.94 (1.31–6.60)*	*2.60 (1.16–5.86)*	*2.67 (1.13–6.29)*
Model fit (without OSA-related variables)	$R^2 = 0.21$ C = 0.74	$R^2 = 0.22$ C = 0.74	$R^2 = 0.23$ C = 0.75	$R^2 = 0.21$ C = 0.74

Estimates are presented as odds ratios and 95% confidence interval. Statistically significant values are in italics

SE, sleep efficiency; *vs*, versus; *OR,* odds ratio; *CI*, confidence interval; *IQR*, interquartile range; *AHI*, apnea-hypopnea index; *OAHI*, obstructive apnea-hypopnea index; *SAHI*, supine apnea-hypopnea index; *Arl*, arousal index; *SaO_2*, oxygen saturation; *ESS*, Epworth sleepiness scale; *BMI*, body mass index; *No. deficits*, number of stroke deficits; *CCI*, Charlson Comorbidity Index; *OSA*, obstructive sleep apnea

*The effect of each obstructive sleep apnea-related variable was tested separately in the statistical model controlling for sex, age, body mass index, smoking status, history of prior hypertension and cardiovascular disease, No. deficits, Charlson Comorbidity Index, and family support

Discussion

This is the first study to evaluate both the uptake and usage of CPAP in stroke patients in the clinical setting with newly diagnosed OSA over a median follow-up of approximately 6 years. Firstly, our study showed that of those recommended to commence CPAP, just over half were using at the 3- and 6-month time period. However, both subjective and objective CPAP adherence demonstrated stability from 3 months following commencement of CPAP to the final available follow-up. Secondly, OSA-related characteristics such as the severity of the disease, arousal index, or sleep architecture were poorly predictive of usage. Although, those patients with a lower mean nocturnal oxygen saturation were more likely to purchase CPAP. Thirdly, CPAP pressure, interface, or device type had no bearing on usage. Finally, while those with greater stroke-related deficits were less likely to use CPAP over the short term, we showed that those patients who were younger, male, never smokers and those with multiple comorbidities were more likely to use CPAP. Our study has a number of strengths as it takes a relatively large undifferentiated group of patients with stroke from a sleep clinic and assesses their uptake and usage of CPAP in the real-world setting. All stroke patients seen at the clinic and assessed were included and as such it accurately reflects the true uptake of CPAP and has limited recall bias. We believe that this more accurately reflects the reality of patient adherence in the general stroke community. The study also identifies a group of stroke patients with OSA (female subjects, smokers, older age,

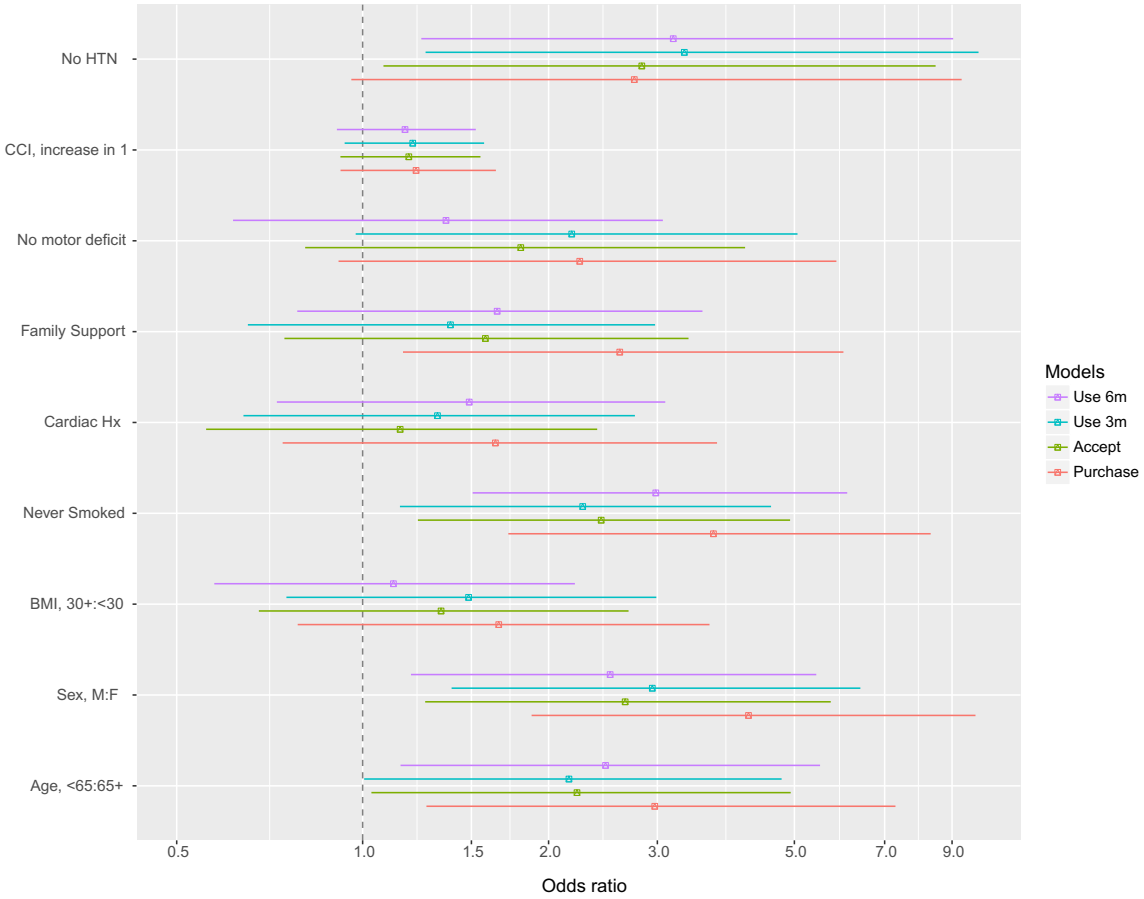

Fig. 4 Results from four multivariable logistic regression models presented as odds ratios with 95% confidence interval. Outcomes: CPAP purchase, initiation, and usage at 3 and 6 months. The vertical dotted line represents an odds ratio of 1 (no effect) and the open squares represent odds ratios and the horizontal lines represent 95% confidence interval. An odds ratio > 1indicates higher odds of CPAP purchase, initiation, and usage at 3 and 6 months. BMI, body mass index; HTN, hypertension, CCI, Charlson comorbidity index; Hx, history

more stroke-related deficits) who may require greater time and resource input to improve uptake of CPAP therapy.

OSA (AHI ≥ 10 events per hour) is highly prevalent in the post-stroke population occurring in approximately 68% of subjects [22]. Untreated OSA in this population is associated with poorer functional outcomes, longer hospitalization [23] greater stroke severity [11] and lower mood. Small studies of CPAP treatment in stroke have shown improvements in functional outcomes, reduction in sleepiness [9] and improvements in neurological recovery [13]. Therefore, treatment of OSA is highly desirable to potentially improve outcomes. The efficacy of CPAP treatment for OSA is contingent upon patient usage and adherence [24].

CPAP usage in the general population vs our study

In the general population, CPAP adherence is approximately 30–70% with no significant change in adherence rates over the last 15 years despite advances in technology and behavioral interventions [5, 25–27]. Most of these studies had relatively short follow-up, although more recent studies in the general population have evaluated CPAP usage at 60 months and documented rates of 50% [27], 81% [28], and 89% [29], respectively. Furthermore, a recent North American 36 months follow-up study by Russell et al. demonstrated comparable CPAP adherence rates of 42% [26] to our stroke population. Differences in socioeconomic factors [26] and physician recommendation biases for initiation of CPAP may play a role in the differences in adherence rates between studies. Additionally, there are notable differences between the stroke and general populations in these CPAP studies. Stroke patients with OSA report less sleepiness [30], are of older age and are more likely to have impaired motor and neurocognitive function and multiple comorbidities. Furthermore, contrary to the general population studies, a lower (4) not higher ESS (9) was a predictor of CPAP adherence in our stroke

Table 5 Factors associated with continuous positive airway pressure acceptance in the post-stroke cohort with obstructive sleep apnea as identified in cox regression analyses

	Initiation of CPAP treatment (acceptance) HR (95% CI), *p* value
Sleep apnea-related variables*	
SE, %: 82 vs. 59 (IQR)	1.21 (0.92–1.58), 0.17
AHI: 45 vs 21	0.97 (0.74–1.27), 0.80
OAHI: 38 vs 15	1.03 (0.79–1.36), 0.82
SAHI: 52 vs 22	1.04 (0.82–1.32), 0.73
ArI: 40 vs 18	0.86 (0.65–1.14), 0.28
MeanSaO_2: 96 vs 93	0.88 (0.66–1.17), 0.38
MinSaO_2: 87 vs 73	1.10 (0.84–1.44), 0.47
ESS: 9 vs 4	0.94 (0.72–1.23), 0.66
Variables included in the statistical model without sleep apnea-related variables	
Age: 57 vs 73	2.04 (1.40–2.97), < 0.01
Sex: M vs F	1.81 (1.12–2.93), 0.02
BMI: 32 vs 24	1.27 (0.98–1.64), 0.07
Ever Smoke: No vs Yes	1.85 (1.21–2.83), < 0.01
No. Deficits: 0 vs 1+	1.73 (1.07–2.79), 0.02
Cardiac Hx: Yes vs No	0.89 (0.57–1.40), 0.62
Hypertension: No vs Yes	1.92 (1.15–3.23), 0.01
CCI: 6 vs 3	2.32 (1.37–3.92), < 0.01
Family Support: Yes vs No	1.39 (0.88–2.19), 0.15
Model fit (without OSA-related variables)	R2 = 0.18

Estimates were expressed as hazard ratios and 95% confidence interval. A hazard ratio greater than 1 indicates higher hazard of the outcome occurring; a hazard ratio of less than 1 indicates lower hazard of the outcome occurring

*The effect of each obstructive sleep apnea-related variable was tested separately in the statistical model controlling for sex, age, body mass index, smoking status, history of prior hypertension and cardiovascular disease, No. deficits, CCI, and family support

population suggesting that the ESS has no utility for the prediction of OSA in the post-stroke population [31].

CPAP usage and older age

The relationship between older age and the uptake and usage of CPAP is uncertain [32]. In a small retrospective study of sleep clinic patients' older than 65 years, 64% were compliant with therapy [33], demonstrating greater usage than our stroke population. Adherence in this study was greatest in those who initiated CPAP at a younger age and would be consistent with our finding of younger age as a significant predictor of CPAP usage. However, the study by Lopez-Padilla et al. [34] of 155 subjects greater than 80 years prescribed CPAP therapy for moderate to severe OSA, a quarter of whom had a reported stroke, also had superior adherence (60% at 53 months) compared to our study. Differences in CPAP usage between this study and ours may be due to the severity of OSA. Our study included those with mild, moderate, and severe OSA, although those subjects with an AHI < 20 events per hour were a relatively small percentage (11%). Overall our uptake and usage of CPAP compared to the general population is lower but not too dissimilar.

CPAP usage in stroke cohorts

In the stroke population, many of the early studies highlighted difficulties with CPAP initiation and usage in this group. Most studies were of short duration and the majority had dismal adherence rates ranging from 27 to 50% [6, 9] with occasional exceptions where patients had close monitoring and supervision [9, 35]. A recent meta-analysis of 10 randomized controlled trials in stroke subjects calculated an overall CPAP usage of 4.53 h per night, albeit with considerable heterogeneity and short-term follow-up for the majority of the studies included. In this meta-analysis, it was noted that there was reduced usage in those with delayed (10–28 days) compared to early (within 7 days) initiation of CPAP [12]. In contrast, our study evaluated a more delayed start to CPAP (usually

within 3 months) and we did not find that time from stroke to CPAP initiation was a significant predictor in our cohort. In this meta-analysis, it was also noted that many of the studies did not consider early dropouts and may have biased towards an overestimation of adherence. The results from the longer 5 year follow-up studies also demonstrate highly varying adherence rates ranging from 29% in the study by Martinez-Garcia and colleagues [36] to 80% adherence rate in that by Parra et al. [24]. Our cohort adherence rates are in the midrange of both of these studies. The inclusion of subjects with an AHI < 20 events per hour in both our study and the study by Martinez-Garcia et al. may in part account for the lower CPAP adherence compared to the study by Parra et al. Although, the majority of studies have demonstrated that adherence to CPAP was unrelated to age, obesity, and sex our study demonstrated that younger age and male sex was an important predictor of CPAP usage in this population. Interestingly, while those with greater stroke deficits and hypertension had poorer adherence, patients with multiple comorbidities had significantly greater initiation and early usage of CPAP. However, obtained results on hypertension may be affected by a relatively small number of individuals without hypertension ($n = 28$) in our cohort. The results also confirm that stroke-related deficits impede PAP application at night adversely impacting usage. Lastly, the presence of family support while augmenting CPAP purchase also had an influence in terms of longer-term adherence to therapy after 12 months. Our study highlights that most of those with CPAP adherence at 3 months continued to have longer-term usage. This pattern has also been demonstrated in the general population [25] highlighting the need to target poor users early with interventions to improve usage and adherence.

This study had several limitations including the retrospective nature of the study which limited the ability to effectively identify factors that impacted patient adherence, the relatively small sample size in subgroups which limited the statistical power to detect many traditional factors associated with CPAP adherence, the single-center location of the study and the absence of a measure of objective function of these stroke patients. Another significant limitation was the absence of objective adherence data in the majority of patients. This was related to both the available device therapy at the time and to the then government provincial funding of CPAP therapy within Ontario, Canada, which did not cover the additional cost of the smart card technology. In those in whom data was available the average adherence did not demonstrate a downward trend over the study period as was demonstrated in the SAVE study [37]. It is acknowledged that self-reported usage overestimates actual CPAP use by approximately 1 h per night when compared to objective data [5, 38]. The delay in obtaining treatment post-stroke for OSA may have impacted CPAP adherence negatively and may not be representative of the management of stroke populations with OSA in other jurisdictions. Furthermore, we did not assess for specific CPAP- related factors that would impact patient use. A recent study by Fung et al. identified operational factors with CPAP equipment as a limitation to usage in a subset of older subjects [39]. Socioeconomic status which has been shown to reduce CPAP usage was not assessed in this group, nor were personality traits [40]. Lastly, we did not have the PSG calculated oxygen desaturation index (ODI) and therefore, were unable to assess whether or not this was an important factor in CPAP adherence as was demonstrated in some other studies [27].

In summary, this study demonstrates that in the real-world setting, long-term usage of CPAP therapy is feasible and occurs in 40–50% of post-stroke patients. Future prospective studies should be performed to specifically address the factors that impede CPAP uptake and adherence.

Compliance with ethical standards

The work was performed at the University Health Network, Toronto, Canada.

All authors have seen and approved the manuscript.

Conflict of interest The authors declare that they have no conflicts of interest.

Ethical approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards.

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