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ORIGINAL ARTICLE

A Pilot Study on the Efficacy of Continuous Positive Airway Pressure on the Manifestations of Dysphagia in Patients with Obstructive Sleep Apnea

Fabio Azevedo Caparroz[1] · Milena de Almeida Torres Campanholo[1] · Danilo Anunciatto Sguillar[1] · Leonardo Haddad[1] · Sung Woo Park[1] · Lia Bittencourt[2] · Sergio Tufik[2] · Fernanda Louise Martinho Haddad[1]

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Abstract
There is evidence in the literature demonstrating that patients with obstructive sleep apnea (OSA) may present with dysphagia, but few studies have evaluated whether this complaint can be reversed with treatment of OSA. To assess whether findings of dysphagia in patients with OSA can be reversed with the use of continuous positive airway pressure (CPAP) devices. Seventy adult patients (age 18–70 years) with moderate or severe OSA were included in the study. All patients underwent fiberoptic endoscopic evaluation of swallowing (FEES) and completed the SWAL-QOL questionnaire on quality of life in dysphagia. Patients with visible abnormalities on FEES were treated with CPAP and reassessed after 3 months. The prevalence of dysphagia was 27.3% (18 patients). Premature spillage was the main finding. On comparison of groups with and without dysphagia, the SWAL-QOL score was significantly worse in the dysphagia group in domain 2 (eating duration and eating desire, $p = 0.015$), with no impact on overall score ($p = 0.107$). Of the 18 patients with dysphagia, 12 were started on CPAP; 11 exhibited satisfactory adherence and remained in the study. Abnormal FEES findings resolved in 81% ($n = 9/11$) of patients who started CPAP ($p = 0.004$), and dysphagia-specific quality of life also improved significantly (overall SWAL-QOL score, $p = 0.028$). In this sample of patients with OSA, the overall prevalence of dysphagia (as demonstrated by premature spillage on FEES) was 27.3%. Treatment of OSA with CPAP was able to reverse the endoscopic findings of swallowing dysfunction and to improve quality of life as measured by the SWAL-QOL.

Keywords Dysphagia · Deglutition · Deglutition disorders · Obstructive sleep apnea

Introduction

There is evidence in the literature that patients with obstructive sleep apnea (OSA) experience a high prevalence of signs and symptoms of dysphagia. A recent study showed that approximately 50% of patients with moderate or severe OSA had signs of oropharyngeal dysphagia [1]. It is possible that the neural lesions found in the soft palate and oropharynx secondary to respiratory trauma [2] could lead to dysfunctions in the deglutition process, since neural afferents could be compromised in these cases [3]. At the same time, it is believed that the natural history of OSA and the repetitive low-frequency vibratory trauma produced by snoring in individuals with the disease cause histopathological alterations consistent with peripheral nerve injury in the pharynx of these patients [4], which could impair the neuromuscular efficiency of the upper airway—the response to negative pressure during inspiration, preventing collapse—and the central integration between swallowing and breathing functions [5, 6].

In patients with OSA, abnormalities of deglutition may be symptomatic or asymptomatic, and consist mostly of premature spillage of the food bolus and residual food in the pharynx after the swallow is completed [1, 6, 7].

✉ Danilo Anunciatto Sguillar
danilo_sguillar@hotmail.com

[1] Departamento de Otorrinolaringologia e Cirurgia de Cabeça e Pescoço, Universidade Federal de São Paulo, São Paulo, Brazil

[2] Departamento de Psicobiologia, Universidade Federal de São Paulo, São Paulo, Brazil

OSA alone has a series of systemic negative impacts, increasing cardiovascular risk, worsening cognition, and impairing quality of life. It is also well known that dysphagia is a frequent symptom in the elderly, and has a substantial impact on morbidity and mortality. Thus, the association between OSA and dysphagia can have even more serious consequences as patients age. It is therefore important to identify these factors and ensure early treatment of these disorders.

Continuous positive airway pressure (CPAP) is currently the most effective treatment for OSA, abolishing obstructive respiratory events and snoring. Whether OSA treatment would be able to reverse the symptoms and signs of dysphagia in patients with this disorder is unknown, although anecdotal reports do suggest so [8].

Within this context, the present study sought to assess whether findings of dysphagia in patients with OSA can be reversed with the use of continuous positive airway pressure (CPAP) devices.

Methods

This study was conducted in two stages. The first stage consisted of a cross-sectional historical cohort study of patients seen at the Outpatient Sleep Disorders Clinic of the Department of Otorhinolaryngology and Head and Neck Surgery, Federal University of São Paulo (UNIFESP/EPM), from September 2014 to December 2017, in which patients with moderate or severe OSA were included. The second stage of the study consisted of prospective follow-up of patients diagnosed with dysphagia by fiberoptic endoscopic evaluation of swallowing (FESS). These patients were prescribed CPAP (considered standard treatment for moderate and severe OSA) and scheduled to undergo reassessment after 3 months. This project was approved by the institutional Research Ethics Committee/Plataforma Brasil with opinion number 195196 (report 35375814.0/0000.5505).

Participation in the study was voluntary. Patients who agreed to take part signed an informed consent form, which had been previously approved by the institutional Research Ethics Committee.

Patients with moderate and marked OSA (2014 International Classification of Sleep Disorders definition), diagnosed by overnight polysomnography, of both genders, aged 18–70 years, were consecutively included. Patients with decompensated clinical and/or psychiatric illnesses, users of alcohol or other psychoactive substances, including sedative medications, and those with neuromuscular or neurodegenerative diseases were excluded. Patients with a history of stroke, patients who had undergone pharyngoplasty, and patients who had used CPAP therapy in the preceding 3 months were also excluded.

All patients underwent overnight polysomnography. An EMBLA® S7000 polygraph (Embla Systems, Inc., Broomfield, CO, USA) was used. The following

physiological variables were monitored simultaneously and continuously: four channels for the Electroencephalogram (EEG); two channels for the electrooculogram; four channels for the surface electromyogram (submentonian region, anterior tibialis muscle, masseter region, and seventh intercostal space); one channel for an electrocardiogram; airflow detection via two channels through a thermocouple (one channel) and nasal pressure (one channel); respiratory effort of the thorax (one channel) and of the abdomen (one channel) using inductance plethysmography; snoring (one channel) and body position (one channel); oxy-hemoglobin saturation (SpO_2); and pulse rate. Technicians visually scored all PSGs according to standardized criteria for investigating sleep. EEG arousals and leg movements were scored according to the criteria established by the AASM Manual for Scoring Sleep and Associated Events. Apneas were scored and classified following the recommended respiratory rules for adults suggested by the AASM Manual (drop in the peak signal excursion by $\geq$ 90% of pre-event baseline using an oronasal thermal sensor for at least 10 s). Hypopneas were scored according to the following criteria: peak signal excursions drop by $\geq$ 30% of pre-event baseline using nasal pressure for at least 10 s and in association with either $\geq$ 3% arterial oxygen desaturation or an arousal. Therefore, sleep staging, recording of arousals, and analysis of respiratory events were all performed in accordance with the American Academy of Sleep Medicine (AASM) criteria [9–11].

The selected patients then began the study protocol, which included a thorough history with a particular focus on sleep-disordered breathing, sleep questionnaires, a dysphagia-specific quality of life questionnaire (SWAL-QOL), and fiberoptic endoscopic evaluation of swallowing (FEES) to assess for dysphagia. After this evaluation, patients were divided into two groups: with and without dysphagia. Data of interest were compared between these groups. Patients diagnosed with dysphagia were prescribed CPAP treatment for OSA and scheduled to undergo reassessment, per the same protocol, after 3 months. The examiners who performed FEES were blinded to OSA severity, SWAL-QOL score, and CPAP use.

The SWAL-QOL questionnaire completed by patients consists of 44 items divided into 11 domains, originally related to food quality (swallowing as a burden, eating duration, eating desire, symptom frequency, food selection, communication, fear, mental health, social interaction, sleep, and fatigue). For the final Portuguese language

version [12], which has been validated elsewhere and was used in this study, items from the original domains 2 and 3 (eating duration/eating desire) were placed in the same domain. The possible answers are: all of the time (0 points), most of the time (25 points), some of the time (50 points), a little of the time (75 points), and none of the time (100 points). The score for each domain is obtained by the sum of the answers to the domain items divided by the number of questions in each domain. Each domain score can range from 0 (worst) to 100 (best). In this study, we used the Portuguese-language version of the SWAL-QOL, translated and validated by Portas [12].

FEES was performed using a 3.4-mm flexible fiberoptic nasopharyngoscope (Pentax®, Japan) [13]. The optic was introduced through the nasal cavity which offered the least resistance, without topical anesthetic, so as not to alter the sensitivity of the upper airway mucosa. Patients were seated comfortably so as to simulate a natural meal position, with the trunk slightly flexed forward.

On initial evaluation after introduction of the optic, we observed the anatomy of the entire upper aerodigestive tract, velopharyngeal closure (assessed by sustained vowel emission), presence or absence of salivary stasis, sensitivity of the larynx (glottic adduction to the touch/presence of scope, cough reflex), and mobility of the vocal folds.

On dynamic evaluation, under direct visualization, we observed swallowing of foods of four different consistencies (thin liquid, thick liquid, puree, and solid), all stained with aniline blue dye.

For the thin liquid consistency, patients were simply offered a bolus of filtered water at room temperature. For the thick liquid and puree consistencies, an agar-based food thickener (*EspessaMais* Clean®—MoreCare, N&S Nutritional®) was added to filtered water at room temperature. The following formulas were used to achieve standard consistencies: Thick liquid: 4.5 g thickener/100 mL water; puree: 9.0 g thickener/100 mL water.

At least three complete swallows of each consistency were assessed, for a minimum total of 12 swallows. For each non-solid consistency (thin liquid, thick liquid, and puree), 5- and 10-mL boluses were administered to the patient. For the solid consistency, patients were offered 2.5 × 2.5-cm pieces of saltine crackers, which had also been dyed blue. All examinations were videotaped for later analysis. Each FEES session was monitored by two specialist laryngologists, who were blinded to severity of OSA, as well as to SWAL-QOL scores.

The following parameters were classified as present or absent: (1) premature spillage—was defined as the bolus falling over the base of the tongue or lower this level before whiteout; (2) velopharyngeal dysfunction—the soft palate does not completely occlude the nasopharynx during swallowing, allowing food to leak into the nasal passages; (3) laryngeal penetration—food enters the laryngeal rim, but does not cross the glottis; (4) Tracheal aspiration—food crosses the glottis; and (5) presence of residue (some food residue remains in the pharynx even after three complete attempts at deglutition). Premature spillage was considered to be present when it occurred in at least 2 of the 12 swallows, whereas the other parameters were classified as present after a single occurrence.

The observed abnormalities were reviewed and confirmed on reassessment in order to obtain a critical evaluation of the examination, seeking to ensure that positive findings actually corresponded to a diagnosis of dysphagia.

In patients diagnosed with dysphagia, whose findings were evaluated by two independent laryngologists per study protocol, the indication of CPAP for treatment of OSA was maintained, and reassessment after 3 months of CPAP therapy (following the same protocol used for initial assessment) was scheduled.

The CPAP device recommended and initially used was set to automatic pressure mode unless contraindicated. A nasal mask was used. After at least 7 days of use, the first reading of the memory card was performed, and device pressure was set at the 95th percentile. Patient follow-up was done by reading the memory card once every 2 weeks or once monthly as needed. During these visits, guidance on use of the device was provided and adjustments were made by a physician specialized in non-invasive ventilation for management of sleep-disordered breathing. Patients who used the device adequately and satisfactorily for at least 4 h per night on 70% of nights, with no mask leakage or acceptable leakage (< 25 L/min), satisfactory sleep quality, and resolution of snoring and subjective daytime sleepiness were classified as having good adherence and remained in the study. The parameter of 4 h per night on 70% of nights was used since it is well established that this is the number of hours needed to eliminate cardiovascular risk in patients with OSA. Nevertheless, most of the patients in our study have used CPAP for at least 6 h per night.

For exploratory data analysis, continuous variables were expressed as means, medians, standard deviations, and ranges, while categorical variables were expressed as absolute and relative frequencies. The normality of distribution of continuous variables was analyzed by skewness, kurtosis, and the Kolmogorov–Smirnov test. SWAL-QOL domain scores were described as means and standard deviations, as conventionally done in the literature.

Comparisons between two independent groups (patients with and without dysphagia) were performed by Student's *t* test or the Mann–Whitney test for continuous or ordinal variables, respectively, and by Pearson's Chi-square or Fisher's exact test, as appropriate, for categorical variables.

Comparisons between two related groups (before and after CPAP) were performed by the Chi-square, McNemar, or Wilcoxon signed-rank tests, for categorical or ordinal variables, respectively.

Statistical power was calculated in G*POWER 3.1 (Universität Dusseldorf) [14].

All statistical analyses were carried out in IBM SPSS Statistics for Windows, Version 24.0 (Armonk, NY: IBM Corp). All tests were two-tailed, and p-values < 0.05 were considered significant.

Results

Seventy patients were included in this study. The mean age was 48.9 years, and 49 (70%) were male. The mean (SD) BMI was 31.8 (± 4.6) kg/m^2. The median AHI was 42.4 events/h (16.3–164.0), and the median RDI was 45.1 (18.9–117.0). The mean (SD) neck circumference was 41.7 (± 4.4) cm. Table 1 describes the characteristics of the 70 included patients.

Table 1 Baseline characteristics of the 70 included patients

Variable	$N = 70$
Age (years)	48.9 ± 11.2
Sex, *n* (%)	
Male	49 (70)
Female	21 (30)
Smoking, *n* (%)	
Yes	9 (14.1)
No	55 (85.9)
Neck circumference (cm)	41.7 ± 4.4
BMI (kg/m^2)	31.8 ± 4.6
IAH (events/h)	42.4 (16.3; 164.0)
RDI (events/h)	45.1 (18.9; 117.0)
Arousal index (events/h)	26.3 (0.0; 153.3)
Epworth sleepiness score	15 (3; 24)
N1 (%)	16.3 (0.9; 83.4)
N2 (%)	45.1 (2.3; 79.8)
N3 (%)	15.7 (0.0; 31.9)
REM (%)	15.9 ± 6.5
Sleep efficiency (%)	83.0 (49; 97.3)
O2 saturation nadir (%)	74.0 (50; 88)
Saturation below 90% (% of time)	15.5 (0.1; 76.1)

Continuous variables described as median ± SD or median (range); categorical variables described as *n* (%)

cm centimeters, *BMI* body mass index, *IAH* apnea–hypopnea index, *RDI* respiratory disturbance index, *N1* % time in stage I of sleep, *N2* % time in stage II of sleep, *N3* % time in stage III of sleep, *REM* % time in REM (rapid eye-movement) sleep

According to FEES, dysphagia was present in 27.3% of the sample. All abnormalities corresponded to premature spillage; there were no cases of penetration and/or aspiration. Of these patients, 12 could be followed up after 3 months of CPAP use. One patient was unable to adapt satisfactorily to CPAP therapy. Of the 11 patients reassessed after 3 months, 9 exhibited improvement of dysphagia findings on FEES (Table 4).

Table 2 provides a comparative analysis of sociodemographic and clinical characteristics, stratified by the presence or absence of dysphagia. There was a trend toward significant overrepresentation of female patients in the dysphagia group ($p = 0.069$).

Table 3 provides a comparative analysis of quality of life, stratified by the presence or absence of dysphagia. The only SWAL-QOL domain significantly worse in the dysphagia group was domain 2 (eating desire/eating duration), with $p = 0.015$.

Table 4 provides a comparative analysis of dysphagia-specific quality of life before and after CPAP. Of the 18 patients with dysphagia in the sample, 12 could be followed up after 3 months of CPAP use. One patient was unable to adapt satisfactorily to CPAP therapy. Significant improvements in dysphagia were observed after 3 months of CPAP ($p = 0.004$). Specifically, significant changes after CPAP were observed for the overall SWAL-QOL score ($p = 0.028$) and for domains 5 (communication) and 10 (fatigue), with $p = 0.026$ and $p = 0.046$, respectively.

Finally, Table 5 shows the characteristics of the 11 patients with endoscopic signs of dysphagia before and after CPAP use.

Discussion

In this sample, the impact of CPAP on dysphagia symptoms and characteristics in OSA patients have been studied for the first time. When SWAL-QOL scores in patients with and without dysphagia were compared, we observed worse quality of life score in only one domain, with no significant impact on total score, suggesting that the manifestations of dysphagia have little additional negative impact on the quality of life of patients with OSA. On the other hand, treatment of OSA with CPAP improved not only FEES findings in 81% of patients (9/11), suggesting possible reversibility of dysphagia in this setting, but also improved quality of life, partly by improving possible symptoms resulting from OSA (observed in the fatigue domain) and possibly related to dysphagia (observed in the communication domain).

The prevalence of dysphagia in the studied group was lower than that reported in the literature (27.3% vs. 50–64%). This was one of the limiting factors in our study,

Table 2 Comparative analysis of sociodemographic and clinical characteristics, stratified by the presence or absence of dysphagia

Variable	Dysphagia present $N = 18$	Dysphagia absent $N = 48$	p value
Age (years)	49.5 ± 11.3	49.3 ± 11.5	0.953
Sex, *n* (%)			
Male	10 (20.8)	38 (79.2)	0.069
Female	8 (44.4)	10 (55.6)	
Smoking, *n* (%)			
Yes	3 (37.5)	5 (62.5)	0.429
No	13 (25)	39 (75)	
Neck circumference (cm)	40.9 ± 4.2	42.1 ± 4.5	0.369
BMI (kg/m^2)	31.7 ± 4.9	31.9 ± 4.7	0.881
AHI	36.4	44.6	0.558
RDI	40.0	45.1	0.955
Arousal index	26.7	25.3	0.832
N1 (%)	20.9	15.6	0.205
N2 (%)	41.0	45.7	0.247
N3 (%)	18.6	15.7	0.495
REM (%)	15.9 ± 7.2	16.0 ± 6.3	0.960
Sleep efficiency (%)	82.7	79.7	0.437
O_2 nadir	74.0	74.0	0.420
Saturation below 90% (% of time)	14.4	16.7	0.603

Continuous variables described as median ± SD or median (range); categorical variables described as *n* (%)

cm centimeters, *BMI* body mass index, *IAH* apnea–hypopnea index, *RDI* respiratory disturbance index, *N1* % time in stage I of sleep, *N2* % time in stage II of sleep, *N3* % time in stage III of sleep, *REM* % time in REM (rapid eye-movement) sleep

Table 3 Comparative analysis of quality of life, stratified by the presence or absence of dysphagia

SWAL-QOL	Dysphagia absent $N = 48$	Dysphagia present $N = 18$	p value
SWAL-QOL total score	79.9 ± 16.3	74.0 ± 16.6	0.107
Domain 1 score	90.0 ± 20.6	84.7 ± 24.8	0.329
Domain 2 score	88.5 ± 14.0	72.4 ± 25.7	**0.015**
Domain 3 score	85.0 ± 14.3	77.9 ± 15.9	0.071
Domain 4 score	84.1 ± 24.4	77.8 ± 29.9	0.361
Domain 5 score	83.8 ± 25.4	74.3 ± 22.0	0.118
Domain 6 score	85.9 ± 20.7	84.4 ± 17.2	0.411
Domain 7 score	90.4 ± 19.4	90.6 ± 15.7	0.791
Domain 8 score	92.5 ± 18.1	90.8 ± 10.2	0.681
Domain 9 score	51.3 ± 37.3	40.3 ± 36.0	0.277
Domain 10 score	48.2 ± 33.7	41.2 ± 32.7	0.430

p value < 0.05 is given in bold

SWAL-QOL domains are described as mean ± standard deviation

SWAL-QOL, quality of life in dysphagia questionnaire

which resulted in a relatively small number of individuals being treated with CPAP and followed up. This might be explained by differences in FEES protocols. Most evaluation protocols proposed in the literature follow the criteria set out by Langmore [13]. Food bolus processing is similar in the various protocols available, but the sequence of bolus presentation varies. Most studies start by presenting the thin liquid consistency. Some authors prefer to include a nectar- or honey-thick consistency, because it is associated with a reduced risk of aspiration. Regarding bolus sizes, Langmore et al. report the initial use of 5 mL (cc) of each consistency [13, 15]. Other protocols use progressive volumes of 3.5 mL, 5 mL, and 10 mL in small sips [16], or administration of progressive volumes < 5 mL, 5 mL, 10 mL, 15 mL, 20 mL until penetration or aspiration occurs [17]. In the present study, progressive boluses of 5

Table 4 Comparative analysis of dysphagia-specific quality of life before and after CPAP

	Before $N = 11$	After $N = 11$	p value
Dysphagia			
Present	11/11 (1.0)	2/11 (0.18)	**0.004**
Absent	0/11 (0.0)	9/11 (0.82)	
SWAL-QOL	75.0 ± 16.3	88.9 ± 9.5	**0.028**
Domain 1 score	88.6 ± 14.2	90.9 ± 11.3	0.746
Domain 2 score	73.9 ± 26.5	86.3 ± 15.2	0.085
Domain 3 score	78.4 ± 16.4	87.1 ± 11.9	0.126
Domain 4 score	75.0 ± 31.6	87.5 ± 22.3	0.207
Domain 5 score	76.1 ± 23.4	95.5 ± 14.0	**0.026**
Domain 6 score	85.2 ± 19.4	86.9 ± 15.4	0.917
Domain 7 score	88.6 ± 15.3	97.3 ± 6.0	0.080
Domain 8 score	90.5 ± 19.7	98.7 ± 4.5	0.273
Domain 9 score	46.6 ± 39.9	70.5 ± 30.2	0.074
Domain 10 score	47.7 ± 34.6	74.9 ± 23.6	**0.046**

p values < 0.05 are given in bold

and 10 mL were administered for each consistency, according to the routine evaluation protocol used at the dysphagia clinic of the institutional Department of Otorhinolaryngology. We chose not to use 15- and 20-mL bolus sizes because, when we critically evaluated the work of Schindler et al., with a 50% prevalence of dysphagia in patients with moderate to severe OSA, we observed that most findings of premature spillage, residue, and/or penetration occurred exclusively with the 20-mL volume [1]. With respect to bolus sizes, the FEES protocol used in the present study was similar to that of Santoro et al. and Valbuza et al. using a similar protocol with the same bolus volumes (5 and 10 mL), these authors found a 64% prevalence of premature spillage [16, 17]. This difference may be explained by the smaller sample size of the latter study (14 patients with OSA).

Regarding comparative analysis of sociodemographic and clinical characteristics according to the presence or absence of dysphagia, the only variable in which there was a trend toward statistically significant differences was female gender ($p = 0.069$). This finding is inconsistent with a previous study by Schindler et al. which showed no significant gender difference [1]. Some studies have reported a greater association between reflux and female gender in patients with OSA [18], but none have demonstrated any association between female gender and dysphagia.

There was no significant age difference between patients with (49.5 ± 11.3 years) and without dysphagia (49.3 ± 11.5 years) ($p = 0.953$). This finding contradicts a previous study of 72 patients, in which dysphagic patients were significantly older [1]. Other studies have also reported a higher prevalence of swallowing dysfunction in older patients [19]. However, our finding was consistent with the work of Valbuza et al. who found no relationship between age and dysphagia in patients with OSA [7]. In a study of 22 patients with OSA evaluated by FEES and esophageal manometry, there was no relationship between age and presence of dysphagia, but laryngeal penetration occurred only in patients with more advanced age [20]. One possible explanation for this fact would be longer duration of exposure to chronic vibratory trauma. It is known that, when healthy individuals are studied, aging may not be a determining factor for the presence of dysphagia [21], although we know it modifies several physiological swallowing features. Despite the fact that there is no specific age at which dysphagia may develop, we

Table 5 Characteristics of patients with dysphagia before and after CPAP

Patient number (initials, gender, age)	RDI (events/h)	Before CPAP[a]		After CPAP[a]	
		Premature spillage	Residue	Premature spillage	Residue
1 (LPB, male) 49-year old	34, 1	Yes, solid	No	No	No
2 (FFC, female) 60-year old	68, 1	Yes, thin liquid and puree	No	No	No
3 (WM, male) 29-year old	97, 4	Yes, thin liquid and puree	No	No	No
4 (ISD, female) 59-year old	36, 3	Yes, thin liquid	Yes, vallecula	No	No
5 (MSC, male) 50-year old	32, 1	Yes, puree	Yes, vallecula	Yes, puree	Yes, vallecula
6 (HRS, female) 70-year old	19, 3	Yes, thin liquid	No	No	No
7 (EN, male) 45-year old	40, 7	Yes, puree	Yes, vallecula	No	No
8 (PMV, male) 47-year old	90, 8	Yes, solid	Yes, vallecula and pyriform sinus	Yes, solid	Yes, vallecula
9 (ASH, male) 39-year old	32, 3	Yes, puree	No	No	No
10 (MCSS, female) 59-year old	41, 7	Yes, thin liquid and puree	Yes, vallecula	No	No
11 (AFM, male) 32-year old	43, 9	Yes, puree	No	No	No

[a]There were no cases of penetration of aspiration on FEES

decided to exclude patients over age 70 from the present study in an attempt to diminish a possible bias.

As in previous studies, there was no statistically significant association between greater severity of apnea and the presence of dysphagia in our sample [1, 20]. There was also no association between lower oxygen saturation and the presence of dysphagia. Some studies have reported an association between oxyhemoglobin desaturation, hypercapnia, and swallowing dysfunction in patients with OSA [22], suggesting that dysphagia may be caused not only by the chronic vibratory trauma of snoring but also by hypoxia/hypercapnia. Other recent studies corroborate this hypothesis; one found a high prevalence of swallowing dysfunction in patients with COPD (49% in a sample of 51 patients) [23]. However, critical appraisal of these studies which suggest a relationship between COPD and swallowing dysfunction reveals that the presence of comorbid OSA—a highly prevalent condition in patients with COPD—was not investigated in the sample [24].

On comparative analysis of quality of life, stratified by presence or absence of dysphagia, the only SWAL-QOL domain significantly worse in the dysphagia group was domain 2 (eating desire/eating duration), with $p = 0.015$. Domain 3 (frequency of symptoms) showed a trend toward statistical significance, with $p = 0.071$. These findings are similar to those of Schindler, who reported a significant difference for domains 1 (swallowing as a burden) and 3 (frequency of symptoms) in dysphagic patients with OSA [1]. The finding of a trend toward significance for domain 3 (frequency of symptoms) is interesting, as the questions asked in this domain (designed to capture signs and symptoms such as coughing, choking, nasal regurgitation of certain food consistencies, throat clearing, among others) can be easily applied to OSA patients, and some are consistent with common laryngopharyngeal reflux symptoms.

Although the SWAL-QOL was initially developed as a dysphagia-specific quality of life questionnaire, studies have shown a statistically significant correlation with instruments for assessment of general quality of life, such as the SF-36 [12, 25].

Significant improvements in dysphagia were observed after 3 months of CPAP ($p = 0.004$). In our review of the literature, we did not find any previous studies demonstrating improvement of dysphagia in a group of subjects with OSA treated with CPAP; only a single report of two cases, but both patients were reported to have had concomitant weight loss [8]. In our study, the 12 reassessed patients had no change in body mass index (BMI) after 3 months. One patient was excluded from analysis at reassessment because she was unable to adapt to CPAP.

Regarding improvements in quality of life after CPAP therapy as assessed by SWAL-QOL, significant changes after CPAP were observed for the overall SWAL-QOL score ($p = 0.028$) and for domains 5 (communication) and 10 (fatigue), with $p = 0.026$ and $p = 0.046$, respectively. There was also a trend toward significant improvement in domains 2 (eating desire/eating duration) and 9 (sleep), with $p = 0.085$ and $p = 0.074$, respectively. These findings are consistent with the literature and were expected, as overall quality of life, fatigue, and sleep patterns are well known to improve after proper use of CPAP.

This study suffers from some limitations. There was no control group recruited and consequently we have not been able to compare the presence of signs of dysphagia between OSAS patients and asymptomatic subjects, and also to compare before and after CPAP between these two groups. Additionally, differences regarding FEES protocols could influence the prevalence of signs of dysphagia in different studies. Besides, the fact that we did not use a standardized classification for the amount and location of residue, such as the pooling score (PS) [26], and our limited sample could be considered a potential bias of our study. Nevertheless, this is the first study to our knowledge to evaluate the reversibility of endoscopic signs of dysphagia in patients with OSA after the use of CPAP.

Conclusion

In this sample of patients with OSA, the overall prevalence of dysphagia was 27.3%. Treatment of OSA with CPAP was able to reverse the endoscopic findings of swallowing dysfunction and to improve quality of life measured by SWAL-QOL.

Compliance with Ethical Standards

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

References

1. Schindler A, Mozzanica F, Sonzini G, Plebani D, Urbani E, Pecis M, et al. Oropharyngeal dysphagia in patients with obstructive sleep apnea syndrome. Dysphagia. 2014;29(1):44–51.
2. Friberg D, Gazelius B, Hökfelt TNB. Abnormal afferent nerve endings in the soft palatal mucosa of the sleep apnoics and habitual snorers. Regul Pept. 1997;71:29–36.
3. Teramoto S, Sudo E, Matsuse T, Matsuse T, Ohga E, Ishii T, et al. Impaired swallowing reflex in patients with obstructive sleep apnea syndrome. Chest. 1999;116(1):17–21.
4. Friberg D, Ansved T, Borg K, Carlsson-Nordlander B, Larsson SE. Histological indications of progressive snorers disease in an upper airway muscle. Am J Respir Crit Care Med. 1998;157:586–93.

5. Jobin V, Champagne V, Beauregard J, Charbonneau I, McFarland DH, Kimoff RJ. Swallowing function and upper airway sensation in obstructive sleep apnea. J Appl Physiol. 2007;102(4):1587–94.
6. Jäghagen EL, Berggren D, Isberg A. Swallowing dysfunction related to snoring: a videoradiographic study. Acta Otolaryngol. 2000;120:438–43.
7. Valbuza JS, de Oliveira MM, Zancanella E, Conti CF, Prado LBF, Carvalho LBC, et al. Swallowing dysfunction related to obstructive sleep apnea: a nasal fibroscopy pilot study. Sleep Breath. 2011;15(2):209–13.
8. Okada S, Ouchi Y, Teramoto S. Nasal continuous positive airway pressure and weight loss improve swallowing reflex in patients with obstructive sleep apnea syndrome. Respiration. 2000;67(4):464–6.
9. American Academy of Sleep Medicine. Sleep-related breathing disorders in adults: recommendations for syndrome definition and measurement techniques in clinical research. The Report of an American Academy of Sleep Medicine Task Force. Sleep. 1999;22(5):667–89.
10. American Academy of Sleep Medicine. International classification of sleep disorders: diagnostic and coding manual. 2nd ed. Westchester: American Academy of Sleep Medicine; 2005.
11. Berry RB, Budhiraja R, Gottlieb DJ, Gozal D, Iber C, Kapur VK, et al. Rules for scoring respiratory events in sleep: update of the 2007 AASM Manual for the scoring of sleep and associated events. Deliberation for the Sleep Apnea Definitions Task Force of the American Sleep Medicine. J Clin Sleep Med. 2012;8(5):597–619.
12. Portas JG. Validação para a língua portuguesa-brasileira dos questionários: Qualidade de Vida em Disfagia (Swal-Qol) e Satisfação do paciente e qualidade no cuidado do tratamento da Disfagia (Swal-Care). Tese (mestrado) São Paulo: Fundação Antônio Prudente; 2009.
13. Langmore SE, Kenneth SMA, Olsen N. Fiberoptic endoscopic examination of swallowing safety: a new procedure. Dysphagia. 1988;2(4):216–9.
14. Faul F, Erdfelder E, Lang A-G, Buchner A. G*Power 3: a flexible statistical power analysis program for the social, behavioral, and biomedical sciences. Behav Res Methods. 2007;39(2):175–91.
15. Langmore SE. History of fiberoptic endoscopic evaluation of swallowing for evaluation and management of pharyngeal dysphagia: changes over the years. Dysphagia. 2017;32(1):27–38.
16. Santoro PP, Furia CLB, Forte AP, Lemos EM, Garcia RI, Tavares RA, et al. Otolaryngology and speech therapy evaluation in the assessment of oropharyngeal dysphagia: a combined protocol proposal. Braz J Otorhinolaryngol. 2011;77(2):201–13.
17. Castro E, Fonseca L, Matos J, Bernardo T, Silva A. Videoendoscopia da deglutição: protocolo de avaliação. Rev Port Otorrinolaringol. 2012;50:197–204.
18. Basoglu OK, Vardar R, Tasbakan MS, Ucar ZZ, Ayik S, Kose T, et al. Obstructive sleep apnea syndrome and gastroesophageal reflux disease: the importance of obesity and gender. Sleep Breath. 2015;19(2):585–92.
19. Payne RJ, Kost KM, Frenkiel S, Zeitouni AG, Sejean G, Sweet RC, et al. Laryngeal inflammation assessed using the reflux finding score in obstructive sleep apnea. Otolaryngol Head Neck Surg. 2006;134(5):836–42.
20. Oliveira LA, Fontes LH. Cahali MB Swallowing and pharyngo-esophageal manometry in obstructive sleep apnea. Braz J Otorhinolaryngol. 2015;81(3):294–300.
21. Kobayashi H, Sekizawa K, Sasaki H. Aging effects on swallowing reflex. Chest. 1997;111(5):1466.
22. Teramoto S, Ishii T, Matsuse T. Relationship between swallowing function and gas exchange during day and night in patients with obstructive sleep apnea syndrome. Dysphagia. 2001;16(4):249–53.
23. Gonzalez Lindh M, Blom Johansson M, Jennische M, Koyi H. Prevalence of swallowing dysfunction screened in Swedish cohort of COPD patients. Int J Chron Obstruct Pulmon Dis. 2017;12:331–7.
24. Teramoto S. A possible pathological link among swallowing dysfunction, gastro-esophageal reflex, and sleep apnea in acute exacerbation in COPD patients. Int J Chron Obstruct Pulmon Dis. 2016;11:147.
25. Ginocchio D, Alfonsi E, Mozzanica F, Accornero AR, Bergonzoni A, Chiarello G, et al. Cross-cultural adaption and validation of the Italian version of SWAL-QOL. Dysphagia. 2016;31(5):626–34.
26. Farneti D. Pooling score: an endoscopic model for evaluating the severity of dysphagia. Acta Otorhinolaryngol Ital. 2008;28(3):135–40.

Fabio Azevedo Caparroz MD

Milena de Almeida Torres Campanholo MD

Danilo Anunciatto Sguillar MD

Leonardo Haddad MD, PhD

Sung Woo Park MD

Lia Bittencourt MD, PhD

Sergio Tufik MD, PhD

Fernanda Louise Martinho Haddad MD, PhD