Surgical treatment for obstructive sleep apnea: effect on sleep architecture

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Abstract

Purpose Investigate the effect of surgical treatment of obstructive sleep apnea syndrome (OSA) on sleep architecture.

Methods Observational retrospective analysis of polysomnographic data of adults diagnosed with OSA, submitted to surgical treatment. Median (25–75th percentile) was used to present the data.

Results Data were available for 76 adults, 55 men and 21 women, with median age of 49.0 years (41.0–62.0), body mass index of 27.3 kg/m^2 (25.3–29.3) and AHI of 17.4 per hour (11.3–22.9) before surgeries. Preoperatively, 93.4% of patients had an abnormal distribution of at least one of the sleep phases. After surgical treatment, we found a significant increase in median N3 sleep percent from 16.9% (8.3–22–7) to 18.9% (15.5–25.4) (p = 0.003). Postoperatively, 18.6% patients that had an abnormal preoperative N1 sleep phase distribution had a normalization of this sleep phase, as also occurred to N2, N3 and REM sleep phases in 44.0%, 23.3% and 63.6% of patients, respectively.

Conclusion This study aims to show the impact of OSA treatment, not only on respiratory events but also on other polysomnographic data often underestimated. Upper airway surgeries have shown to be effective in sleep architecture improvements. There is a trend for sleep distribution normalization, with increase of time spend in profound sleep.

Keywords Drug induced sleep endoscopy · Obstructive sleep apnea · Sleep stages · Surgery

Introduction

Sleep is a complex phenomenon, essential for normal physical and mental status. It is composed by two distinguishable neurophysiologic states, non-rapid eye movement (NREM) and rapid eye movement (REM), which cycle between each other a number of times through the night [1, 2]. In general, NREM sleep is considered to be the time during which the brain recovers from prior wakefulness, while REM sleep plays an important role in memory consolidation [1, 3].

Obstructive sleep apnea syndrome (OSA) is a common medical condition. It is caused by recurrent upper airway collapses, which causes airflow obstruction and consequent need to arouse to breathe. When untreated, OSA decreases the restorative effect of sleep, resulting in excessive daytime sleepiness and poor quality of life [4]. Studies have shown that when comparing with normal individuals, patients with OSA usually exhibit altered sleep architecture, with increased light sleep (N1 and N2) and reduced deep sleep (N3) and REM sleep [5–7].

There is no treatment for OSA that is completely effective and fully tolerated. Continuous positive airway pressure (CPAP) is the first line recommended therapy, although the adherence among patients is usually low [4, 8, 9]. This therapy is responsible for improvements not only in respiratory events, but also in sleep architecture, by decreasing light sleep and increasing deep sleep [10, 11]. Alternatives to CPAP can go from oral appliances, positional therapy, lifestyle changes and surgeries. Surgical options include soft tissue surgeries, like uvulopalatopharyngoplasty or tongue base surgeries, bone or skeletal surgeries, like maxillomandibular advancement surgery, but also stimulation surgeries [5, 11]. Surgical treatments significantly improve respiratory parameters in patients with OSA, however the effects on

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sleep architecture have not been well studied. On Fig. 1 is shown a flowchart about treatment of OSA.

When considering CPAP alternatives, many authors recommend meticulous assessment of the level(s), degree and patterns of upper airway collapse to better guide treatment selection and outcomes. In 1991, Croft and Pringle introduced a way to assess upper airway in patients with OSA under sedation: drug induced sleep endoscopy (DISE) [13]. Evidence shows that DISE can help to improve treatment outcomes, and it is a valuable tool in treatment selection [12, 14].

Despite the recognized effects of upper airway surgeries on respiratory parameters, the effects of these procedures on sleep architecture are not so well known. The objective of this study was to compare changes on sleep architecture before and after upper airway surgeries, alternative treatment for patients with OSA when CPAP did not achieved the desired goal. We theorize that upper airway surgeries in patients with OSA may improve sleep architecture.

Methods

This was an institutional review board-approved retrospective study, performed at a single medical center (Professor Doutor Fernando Fonseca Hospital, Lisbon) between 2015 and 2020. Local ethics committee approved the study, which was designed and conducted in compliance with the principles of Good Clinical Practice regulations and the Helsinki declaration.

All patients included in this study had more than 18 years, were diagnosed with OSA on a baseline level II PSG, failed or could not tolerate CPAP therapy, were revaluated by nocturnal level II PSG after surgical treatment, were submitted to DISE to asses UA obstruction, were eligible for UA surgery. Patients with one or more of the following conditions were excluded: prior sleep surgery, uncontrolled nasal obstruction, obesity (body mass index ≥ 30 kg/m^2), neurologic or neuromuscular disorders, hypothyroid, patients who were taking medication or had an underlying condition that might affect the sleep architecture (namely taking medication that could affect sleep–wake cycle, periodic limb movement disorder or depression).

Clinical records of the patients were reviewed to gather data, namely: age, gender, body mass index, relevant
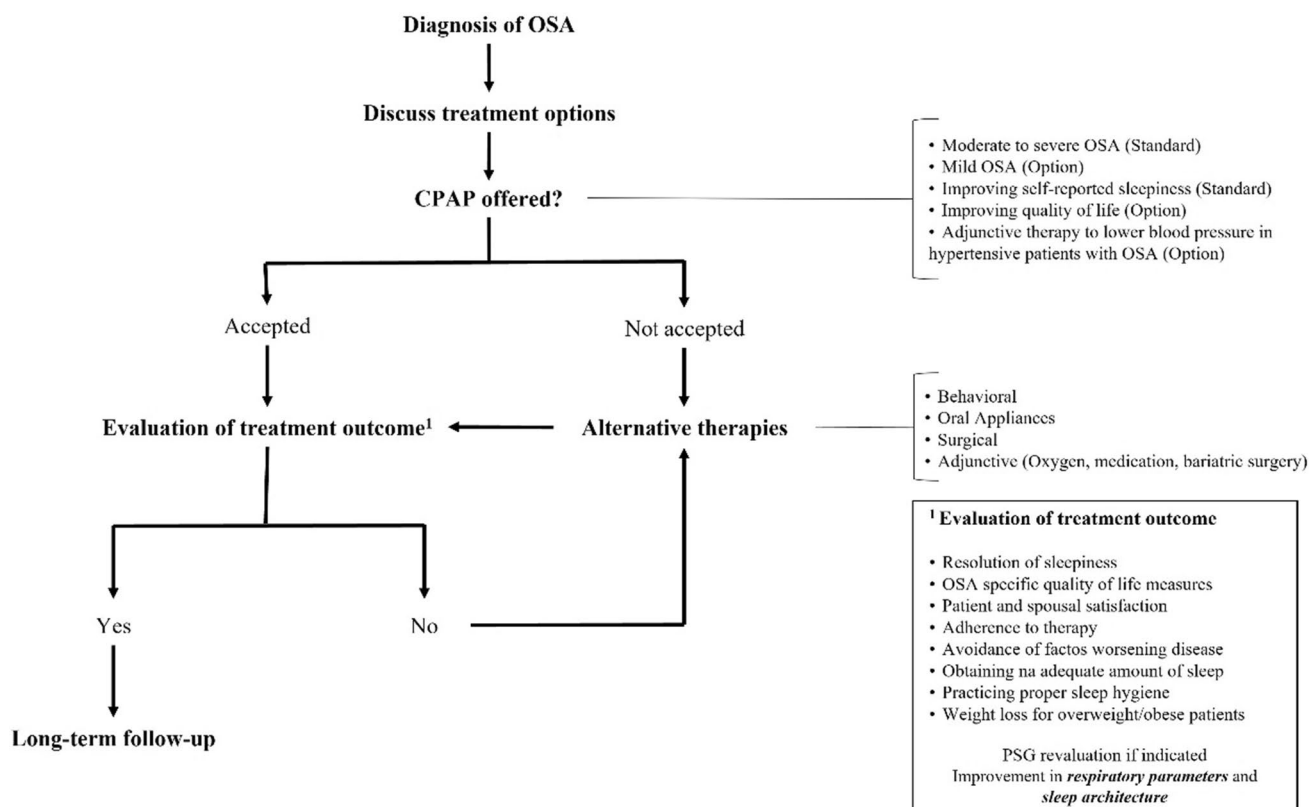

Fig. 1 Flowchart for evaluation and treatment of patients with OSA

personal medical history, DISE results, surgeries performed and preoperative and postoperative PSG data (total sleep time (TST), sleep efficiency, sleep latency, total and percentage of TST spent in each sleep stage, REM latency, wake after sleep onset (WASO) and arousal index, total apnea–hypopnea index (AHI), oxygen desaturation index (ODI), percentage of sleep with peripheral capillary oxygen saturation (SpO_2) < 90%).

Polysomnography

All patients were submitted to nocturnal level II PSG preoperatively and postoperatively, and data was analyzed at the neurology department of the Professor Doutor Fernando Fonseca Hospital, Lisbon, according to the American Sleep Association [15]. The following variables were continuously measured and recorded by a computerized polysomnograph (Compumedics, model Siesta): electroencephalography (EEG) (4 channel), electro-oculography (EOG) (2 channel), electromyography (EMG) of anterior tibialis and chin muscles and electrocardiography. Respiratory effort was measured by chest and abdominal movements, oxygen saturation by a finger probe connected to a pulse oximeter, and airflow was measured by means of nasal pressure cannula, and body position.

Accordingly with EEG, EOG and EMG signals, sleep was scored in four different stages: N1, N2, N3 and REM. Stage N1 was scored when alpha rhythm was attenuated and replaced by low amplitude, mixed frequency signal of 4–7 Hz for more than 50% of the period, decrease in muscle tone and slow eye movements. N2 was defined when either one or more K complexes unassociated with an arousal was noted or when one or more trains of sleep spindles appeared. N3 was scored when slow wave activity was detected in at least 20% of the period. Finally, REM was defined when all of the following were present: rapid eye movements, low-amplitude and mixed-frequency activity in EEG and muscle tone at the lowest of any other stage [16]. Sleep stage distribution was expressed as the amount of a given sleep stage relative to total sleep time (TST).

DISE

DISE was performed in the operating room, with patients in a supine position, with a pillow, in a silent and dark room. Standard anaesthesiological monitoring was used (oxygen saturation, electrocardiogram and blood pressure). Sleep endoscopy was manually performed using propofol, with a delivering dose up to freeze at the observation window (observing two or more cycles of snoring-obstructing hypo/apnea-oxygen desaturation-breathing for each segment of upper airway). The correct depth of sedation level was guided by bispectral index (BIS) in the recommended range

of 50–70 [12]. Endoscopic video recordings were saved and were later reviewed and graded for obstruction level and severity. Grade and patterns of upper airways collapse were evaluated at four levels: velum, orolateral pharyngeal walls, tongue base and epiglottis. It was also defined the different patterns of collapse as anteroposterior, lateral and concentric. To grade the collapse it was used a semi-quantitative system with 0–25, 25–50, 50–75 and 75–100% of obstruction. Significant or complete obstruction was considered when more than 75% collapse was seen.

Surgeries

Patients who failed or refused a trial with CPAP and were eligible for surgery were submitted to surgery accordingly with clinical evaluation and DISE results. Depending on the level of UA collapsibility, different techniques were proposed. Palatoplasty was performed when a velum and/or orolateral pharyngeal walls collapse were seen, tongue base surgery when a tongue base collapse was captured or a partial epiglottectomy when an epiglottis collapse was seen. It was used suspension palatoplasty, described by Vicini et al. [17]. For tongue base collapse was performed tongue base coblation or tongue base ablation radiofrequency. Partial epiglottectomy was performed using carbon dioxide laser. Multilevel surgeries were performed concurrently. All surgeries were performed by the same experienced team of surgeons.

Statistical analysis

Statistical analysis was performed with SPSS version 26.0 software (International Business Machines Corporation, USA). Data were presented as median (25–75th percentile). Comparisons between groups were made by Wilcoxon Signed-Rank test, and Spearman's Rank Correlation calculated correlations. Multivariate analyses were conducted to adjust for potential confounding factors. Statistical significance was accepted at $p < 0.05$.

Results

Study population

Seventy six adults were eligible for inclusion in this study: 55 men and 21 women. They had a median age of 49.0 years (41.0–62.0), median body mass index of 27.3 kg/m^2 (25.3–29.3), median neck circumference of 40.0 cm (38.0–42.0) and median Epworth Sleepiness Scale score (ESS) of 7.0 (3.0–12.0). Median preoperative AHI was 17.4 (11.3–22.9), and 40.8% (31 patients) had a preoperatively mild OSA, 48.7% (37 patients) moderate OSA and 10.5%

(8 patients) a severe OSA. 57.9% of patients had significant multilevel collapse (> 75% of collapse at more than one level). All 76 patients were submitted to palatoplasty, 27.6% (21 patients) to tongue base surgery and 9.2% (7 patients) to epiglottis surgery. Surgery techniques performed are presented in Table 1.

Prevalence of sleep architecture disturbances

Preoperatively, 93.4% of patients (71/76) had an abnormal distribution of at least one of the sleep phases: 77.6% of patients had an increased N1 phase, 32.9% an increased N2 phase, 39.5% a decreased N3 phase and 28.9% a decreased REM phase.

Postoperatively, 18.6% (11/57) patients that had an abnormal preoperative N1 sleep phase distribution had a normalization of this sleep phase, as also occurred a N2 sleep phase normalization in 44.0% (11/24) patients, N3 sleep phase normalization in 23.3% (7/30) patients and REM sleep phase normalization in 63.6% (14/21) patients. Changes in sleep stages after surgical treatment are presented in Table 2.

No statistically significant differences were seen in preoperative sleep architecture between subgroups of patients based on OSA severity (mild, moderate and severe), type of surgeries performed (palatoplasty, palatoplasty and tongue base or palatoplasty and epiglottis surgery) or sex.

There were no significant correlations between age, ESS or neck circumference and any of the sleep phases (time spent in or percentage of TST of N1, N2, N3 or REM).

Effect of upper airway surgeries on sleep architecture

To evaluate the impact of upper airway surgeries on sleep architecture, it was compared preoperative with postoperative PSG data. After surgeries AHI decreased from 17.4 (11.3–22.9) to 13.0 (8.7–24.0). Time spent in N3 significantly increased from 70.2 min (49.9–97.7) to 83.9 (62.8–114.8) (p = 0.026), no statistically significant changes were seen in time spent in N1, N2 or REM sleep. It was observed statistically significant changes in N3 sleep percent, that increased from 16.9 (11.3–22.1) to 18.9 (15.5–25.4) (p = 0.003). Preoperative and postoperative data are presented in Table 2.

Significant correlations were found between arousal index and time spent in N1 (r = 0.41, p < 0.001), N2 (r = 0.39, p = 0.001) and N3 (r = -0.21, 0.049), as also occurred between AHI and time spent in N1 (r = 0.32, p = 0.005) and N2 (r = 0.29, p = 0.013).

Nineteen patients (25.0%) were considered as nonresponder to surgical treatment, as no decrease of AHI was reported. When comparing responders with non-responders, there are statistically significant differences in postoperative PSG data: responders AHI of 10.0 (6.6–17.9) compared with 24.5 (18.1–39.6) (p < 0.001) in non-responders group, as also occurred for time spent in N1 of 46.0 (28.1–57.7)

Table 1 Upper airway surgeries performed

Surgery	Percentage	Number
Suspension palatoplasty	100	76
Tongue base surgery	27.6	21
Coblation	13.2	10
Radiofrequency	14.5	11
Parcial epiglottectomy	9.2	7

Table 2 Pre-operative and postoperative comparisons of PSG data

Variable	Pre-operative	Post-operative	p-value
TST (min)	456.8 (385.5–514.6)	423.8 (392.3–467.4)	0.158
Efficiency (%)	91.3 (83.2–94.6)	90.9 (85.0–94.6)	0.353
Stage 1 NREM (min)	47.3 (33.4–71.9)	47.5 (29.4–68.9)	0.895
Stage 2 NREM (min)	233.7 (191.1–269.3)	198.5 (115.5–307.9)	0.421
Stage 3 NREM (min)	70.2 (49.9–97.7)	83.9 (62.8–114.8)	**0.026**
REM (min)	84.6 (58.6–113.8)	75.6 (59.7–104.5)	0.350
Stage 1 NREM (%)	11.3 (7.2–16.4)	11.2 (7.7–16.4)	0.987
Stage 2 NREM (%)	50.8 (43.5–57.8)	49.1 (40.9–55.6)	0.171
Stage 3 NREM (%)	16.9 (11.3–22.1)	18.9 (15.5–25.4)	**0.003**
REM (%)	18.7 (13.4–24.4)	18.1 (14.0–22.6)	0.968
Sleep latency (min)	7.5 (5.0–12.0)	7.0 (3.0–16.0)	0.672
REM latency (min)	93.0 (69.5–139.5)	87.8 (68.9–120.6)	0.588
Arousal index (n/h)	41.3 (19.7–63.2)	37.4 (12.6–53.5)	0.132

Data are presented as median (25–75th percentile). Wilcoxon Signed Rank Test

Min minutes, *Mini* minimum, *n/h* number per hour, *TST* total sleep time |

Bold p-value: statistical significance accepted

compared with 53.7 (43.3–78.9) (p = 0.026) and time spent in N3 of 87.2 (65.8–116.4) compared with 65.6 (54.2–97.4) (p = 0.029), respectively. Postoperative PSG data of responders and non-responders are presented in Table 3.

Discussion

CPAP is the main medical treatment for OSA, but its effectiveness is affected by an overall adherence lower than 50% [18]. There are some alternative treatments, which include oral appliances, positional therapy, lifestyle changes and surgeries. Most of the patients with OSA have multilevel sites of obstruction, including oropharynx, hypopharynx and larynx [12, 19]. Multilevel surgeries for OSA are becoming more frequent, mainly because they are starting to be considered safe and successful [19, 20].

This study is a focused attempt to demonstrate the effects of upper airway surgeries directed by DISE results on sleep architecture. There are some studies focused on other OSA treatment options that already showed improvements across several sleep architecture parameters [5, 11, 21]. The first randomized placebo-controlled trial, performed by McArdle and Dougles in 2001, demonstrated improvements in sleep architecture in patients with OSA, after 1 month of treatment with CPAP. They describe significant reductions in N1 sleep percent, increases in N3 sleep percent, and reductions in arousal index [11]. Also Bohorquez et al. describes significant reductions in N1 sleep percent and arousal index, but also increases in time spent in N2 and N3 sleep, when patients with OSA were successfully treated with upper airway stimulation therapy [5]. These studies based their results on consistent use of treatment devices, which make their findings difficult to reproduce. Long term longitudinal data

suggests that CPAP compliance rates are as low as 30–40% [5].

This study shows that a highly significant proportion of people (93.4%) with OSA experience abnormal distribution of at least one of the sleep phases. This population tend to spend more time in light sleep, with increased N1 sleep (77.6% of population) and N2 sleep (32.9% of population), and less time in deep sleep, with decreased N3 sleep (39.5% of population) and REM sleep (28.9% of population). Some studies show similar results [5–7]. One study included 391 patients and compared polysomnographic data between patients with OSA, primary snoring and without OSA or snoring. They concluded that people with OSA tend to experience more light sleep and less deep sleep when compared with people without OSA, and even with primary snorers. Their findings may be related with increased arousal index usually found in this type of patients, which could interfere with sleep continuity and achievement of deep sleep [7].

In this analysis, after upper airway surgeries, sleep architecture improved. It was observed a N1 sleep normalization in 18.6% of patients that had a preoperative abnormal distribution (11/57), as also occurred for N2 sleep in 44.0% of patients (11/24), N3 sleep in 23.3% of patients (7/30) and REM sleep in 63.6% of patients (14/21). It was found a statistically significant increase in time spent in N3 sleep. Other studies focused on other OSA treatment options, namely CPAP or upper airway stimulation therapy, show similar results [5, 11, 21], but there is limited information about the impact of upper airway surgeries on sleep architecture, in patients with OSA. A study that included 31 patients with severe OSA submitted to genioglossus advancement and hyoid suspension plus uvulopalatopharyngoplasty, also shows a postoperative increase in slow wave sleep and a decrease in lighter sleep [22].

Table 3 Postoperative PSG data comparisons between responders and non-responders to surgical treatment

Variable	Responders	Non-responders	p-value
AHI (n/h)	10.0 (6.6–17.9)	24.5 (18.1–39.6)	**0.001**
TST (min)	423.8 (395.6–474.5)	423.0 (379.9–456.0)	0.504
Efficiency (%)	90.9 (85.2–94.4)	91.1 (82.0–95.2)	0.736
Stage 1 NREM (min)	46.0 (28.1–57.7)	53.7 (43.3–78.9)	**0.026**
Stage 2 NREM (min)	175.7 (108.8–258.4)	257.3 (175.2–348.8)	0.052
Stage 3 NREM (min)	87.2 (65.8–116.4)	65.6 (54.2–97.4)	**0.029**
REM (min)	78.1 (60.5–106.7)	63.2 (58.2–95.0)	0.277
Stage 1 NREM (%)	10.4 (6.8–14.3)	13.8 (10.3–18.1)	**0.026**
Stage 2 NREM (%)	48.1 (40.6–54.6)	50.7 (42.2–58.3)	0.338
Stage 3 NREM (%)	21.2 (16.7–26.4)	15.9 (12.7–23.9)	**0.019**
REM (%)	18.5 (14.0–22.8)	17.1 (13.9–19.7)	0.439

Data are presented as median (25–75th percentile). Wilcoxon Signed Rank Test

AHI apnea hypopnea index, *Min* minutes, *n/h* number per hour, *TST* total sleep time |

Bold p-value: statistical significance accepted

In this study, statistically significative positive correlations were found between arousal index and time spent in and percentage of TST of N1 and N2, as also occurred between AHI and time spent in and percentage of TST of N1 and N2. It was also found a statistically significative negative correlation between arousal index and time spent in and percentage of TST of N3. When non-responders were compared with responders, statistically significant differences were found, with a higher time spent in N1 and lower time spent in N3, for the first group. These results may help to explain why upper airway surgeries caused improvements in sleep architecture, as they may be related with reductions in AHI and arousal index. It is known that OSA causes multiple arouses to breathe, which may lead to sleep fragmentation and this could impact in sleep continuity. Upper airway surgeries cause improvements in AHI and arousal index, which decreases sleep fragmentation to restore normal breathing, with subsequent improvements in sleep depth and consolidation. Similar results have been reported with CPAP therapy and airway stimulation therapy [5, 11, 21].

There are long term effects of improved sleep architecture already reported. Abnormal sleep architecture results in imbalanced sympathetic drive, as it is believed that NREM sleep contributes to autonomic stability. Improving sleep architecture may prevent cardiovascular autonomic homeostasis imbalances [23, 24]. Previous studies have linked sleep architecture disturbances with the risk of clinical cognitive impairment and dementia [25, 26]. Improving sleep architecture could lead to long-term benefits, namely potential reductions in cardiometabolic and neurocognitive risks, but future research is needed.

There are some limitations in our study, some of them because of its retrospective nature. For example, some patients were excluded due to incomplete preoperative polysomnographic data. Another limitation is the follow up duration was not standardized. Long-term follow up could allow for neuroplastic changes to occur, that could optimize sleep and breathing, and so adjusting for follow up duration could be helpful.

The fact that patients were submitted to different procedures, presents itself as an additional limitation. Sleep architecture outcomes may depend on the type of surgeries performed. Future studies should try to get procedure specific outcomes, as this could guide optimal selection of surgeries for sleep architecture benefits. However, no significant differences were seen in preoperative sleep architecture between subgroups of patients based on type of surgeries performed, but there were no patients that were exclusively submitted to tongue base surgery or epiglottis surgery.

Future studies should include randomized prospective trials, with control group to confirm the findings and evaluation of long term effects of sleep architecture optimization. Future studies could also perform sensitivity analysis considering patients that underwent revision surgeries, to find if they significantly confounded results.

Conclusion

Sleep architecture abnormalities are frequent in people with OSA. Upper airway surgeries caused improvements in sleep architecture, through increase in time spent in N3. These changes may have-long term clinical implications, namely cardiometabolic and neurocognitive. Additional prospective researches are needed to confirm these findings.

Declarations

Conflict of interest The authors have no competing interests.

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