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Clinical Paper
Orthognathic Surgery

Does orthognathic surgery have an incidentally beneficial effect on mild or asymptomatic sleep apnoea?

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Abstract. A prospective study was performed to assess the effect of orthognathic surgery on mild obstructive sleep apnoea (OSA) in patients with an underlying dentofacial deformity treated for occlusal and/or aesthetic reasons. As the main outcome variables, changes in upper airway volume and apnoea–hypopnoea index (AHI) were evaluated at 1 and 12 months of follow-up, in patients undergoing orthognathic surgery with widening movements of the maxillomandibular complex. Descriptive, bivariate, and correlation analyses were performed; significance was set at $P < 0.05$. Eighteen patients diagnosed with mild OSA were enroled (mean age 39.8 ± 10.0 years). An overall upper airway volume widening of 46.7% after orthognathic surgery was observed at 12 months of follow-up. The AHI decreased significantly from a median 7.7 events/hour preoperatively to 5.0 events/h at 12 months postoperative ($P = 0.045$), and the Epworth Sleepiness Scale score decreased from a median 9.5 preoperatively to 7 at 12 months postoperative ($P = 0.009$). A cure rate of 50% was obtained at 12 months of follow-up ($P = 0.009$). Despite the limited sample size, this study provides evidence that in patients with an underlying retrusive dentofacial deformity and mild OSA, a slight decrease in AHI is obtained after orthognathic surgery due to upper airway enlargement, which could be added as a beneficial effect of orthognathic surgery.

Keywords: Dentofacial deformities; Obstructive sleep apnoea; Orthognathic surgery; Polysomnography; Three-dimensional imaging.

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Patients with an underlying dentofacial deformity (DFD), especially those with maxillary and/or mandibular hypoplasia, are more prone to suffer from obstructive sleep apnoea (OSA) syndrome, due to the lack of skeletal support of the anterior wall of the upper airway.[1,2] Although the role of the size and position of the mandible is more important than that of the maxillary bone in the context of OSA,

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the relevance of the maxilla is not negligible.[3] OSA patients suffer from recurring oxygen desaturations due to upper airway obstruction, with snoring, unrefreshing sleep, fatigue, and excessive daytime sleepiness. Among other comorbidities, systemic arterial hypertension and heart failure may subsequently develop, with a significant increase in mortality risk.

Orthognathic surgery combined or not with orthodontics aims to correct DFD through maxillomandibular complex repositioning. There have been many descriptions of the impact of forward and counterclockwise orthognathic surgery movements in increasing the upper airway volume,[1] and of their effectiveness in treating patients suffering from chronic sleep-related breathing disorders with underlying maxillary and/or mandibular hypoplasia.[4] In this context, orthognathic surgery is considered a first-line option for the treatment of patients with moderate and severe OSA (≥ 15 events/h),[5,6] with a high surgical success rate of 87.5%[1,4] (success being defined as a final apnoea–hypopnoea index (AHI) threshold of < 20 events/hour, and its reduction by 50%[7]). However, there is a lack of evidence supporting the management of mild OSA (5–15 events/h) by means of orthognathic surgery.

Many patients suffering from mild OSA remain undiagnosed due to the absence of clear symptoms, since they consider their daytime sleepiness or lack of energy as a normal and inherent condition, corresponding to what is also known as 'silent' OSA.[2] However, although sleep in mild OSA is not interrupted as often as in more severe cases, it still represents a serious health problem. Moreover, the normal tendency of the disease is to worsen over time, since the upper airway volume decreases after the age of 30 years due to a loss of muscle tone in the upper airway.[8] Thus, people who suffer from this condition should receive treatment in order to prevent it from becoming more serious.[1] According to the literature, the current gold standard for treating mild OSA is continuous positive airway pressure (CPAP) therapy,[7] although patient adherence is low (adherence being defined as > 4 h of night-time use of CPAP during 70% of all nights),[7,9] because it is noisy and uncomfortable. Mandibular advancement devices have been described as an effective treatment option for mild and moderate OSA[10] in patients with underlying mandibular sagittal hypoplasia, although it may induce periodontal and temporomandibular joint disorders, among other problems.[7,11] Besides, both CPAP and mandibular advancement devices do not permanently resolve the underlying anatomical problems and specific potential causes.[12]

Thus, as a continued effort into the investigation of the treatment of mild OSA, the purpose of this prospective study was to assess the eventual beneficial effect of orthognathic surgery on mild OSA in patients with an underlying retrusive DFD.

Materials and methods

Study design

To address the research purpose, the investigators designed and implemented a prospective study involving consecutive patients scheduled for orthognathic surgery for occlusal or aesthetic reasons between June 2018 and December 2021 at the Maxillofacial Institute, Teknon Medical Center (Barcelona, Spain). All patients underwent a home sleep apnoea test (HSAT) to detect OSA. The HSAT is equated to polysomnography according to the American Association of Sleep Medicine.[13,14] Those patients diagnosed with mild OSA were included in the study.

Patients of either sex, over 18 years of age, with completed maxillomandibular growth and diagnosed with mild OSA (5–15 events/h) and an underlying DFD eligible for orthognathic surgery (single-jaw or bimaxillary) were included in the study. Patients with any craniofacial syndrome, those missing pre- or postoperative HSAT, cone beam computed tomography (CBCT) scans, or follow-up visits, and those who were not willing to sign the informed consent, were excluded.

The study was approved by the Ethics Committee of Teknon Medical Center, Barcelona, Spain (Ref. 2020/06-CMX-CMT) and was conducted in accordance with the ethical standards laid down in the Declaration of Helsinki (1964 and later amendments). All participants signed an informed consent document prior to study enrolment.

Surgical procedure

The standard three-dimensional virtual surgical planning protocol was applied (Dolphin Imaging version 11.95 premium; Dolphin Imaging and Management Solutions, Chatsworth, CA, USA),[15] and the upper incisor–soft tissue nasion plane (UI–STP) was used as an absolute reference to guide anteroposterior positioning of the maxillomandibular complex.[16] Intermediate and final surgical splints were designed and printed in-house. The patients were operated on under general anaesthesia following the mandible-first protocol. A mandibular bilateral sagittal split osteotomy was performed, with or without a maxillary Le Fort I osteotomy. The minimally invasive 'twist' technique was applied in those patients receiving a Le Fort I osteotomy.[17]

Study variables

The main outcome variables assessed were the changes in upper airway volume and AHI.

All patients underwent the standard clinical and radiographic evaluation workflow for orthognathic surgery planning and follow-up in the study department. The protocol comprises clinical evaluation and a CBCT scan (i-CAT; Imaging Sciences International, Hatfield, PA, USA) at three time-points: preoperatively after orthodontic treatment (T0) and postoperatively at 1 month (T1) and 12 months of follow-up (T2). The upper airway volume (total and nasopharyngeal, oropharyngeal, and hypopharyngeal volumes (mm^3)) was measured in each CBCT by two experienced specialists (M.G.H. and A.V.O.).

The HSAT test was performed at the same three time-points (T0, T1, and T2), and an experienced neurophysiologist (G.Z.A.) evaluated the polysomnographic and nocturnal oximetry parameters: AHI (mild OSA, ≥ 5 events/hour; moderate OSA, ≥ 15 events/h; severe OSA, ≥ 30 events/h)), oxygen desaturation index (ODI), the percentage of time spent at arterial oxygen saturation < 90% (CT90%), and the lowest oxygen saturation ($LSpO_2$). Only those patients diagnosed with mild OSA (5–15 events/h) were selected for this study.

Additionally, the following examinations were performed at the same three time-points (T0, T1, and T2): body mass index (BMI, kg/cm^2), neck perimeter (cm), heart rate (bpm), blood pressure (minimum and maximum, mmHg), and daytime drowsiness based on the Epworth Sleepiness Scale (ESS) (normal score 0–10 out of 24 points).[18]

The following demographic variables were recorded: sex, age, smoking and alcohol consumption habits, initial dental class according to the Angle classification (I–III). Surgical parameters were also documented: single-jaw or bimaxillary surgery, type of surgery, direction and amount of each

Table 1. Demographic and clinical data of the sample (N = 18).

	n (%)
Age (years)	
Mean ± SD	39.8 ± 10.0
Range	20–56
Sex	
Male	14 (77.8)
Female	4 (22.2)
Type of dentofacial deformity	
Class I	0 (0)
Class II	11 (61.1)
Class III	7 (38.9)
Previous knowledge of OSA	
Yes	2 (11.1)
No	16 (88.9)
Previous use of CPAP	
Yes	2 (11.1)
No	16 (88.9)
Snoring	
Yes	2 (11.1)
No	16 (88.9)
Previous OSA surgery (soft/hard tissue)	
Yes[a]	2 (11.1)
No	16 (88.9)
Smoker	
Yes	2 (11.1)
No	11 (61.1)
Ex-smoker (> 2 years)	5 (27.8)
Alcohol intake	
Yes (socially, > 1 unit/week)	9 (50)
No	9 (50)
Type of intervention	
Bimaxillary surgery	15 (83.3)
Single-jaw surgery	3 (16.7)
Surgical approach	
Orthodontics first	10 (55.6)
Surgery early	1 (5.6)
Surgery first	7 (38.9)
Sagittal movements	
Maxillary advancement	14 (77.8)
Mandibular advancement	18 (100)
Chin advancement	7 (38.9)
Rotational movements	
Clockwise rotation	0 (0)
Counterclockwise rotation	15 (83.3)
No rotation	3 (16.7)

CPAP, continuous positive airway pressure; OSA, obstructive sleep apnoea; SD, standard deviation.

[a]One patient underwent septoplasty, while the other underwent septoplasty and tonsillectomy.

movement, and eventual intraoperative or postoperative complications.

The data were recorded in an anonymized electronic case report form (e-CRF).

Statistical analysis

Demographic characteristics and other parameters were presented in terms of descriptive statistics. Continuous variables were reported as the number (n), mean, standard deviation (SD), minimum, maximum, and median, while relative frequencies (percentages) were used for qualitative variables. The inferential analysis included (1) the analysis of variance (repeated measured ANOVA) general linear model for repeated measures to compare the change in the skeletal and volumetric parameters over follow-up; (2) the Wilcoxon test to evaluate changes in the variables resulting from HSAT (this non-parametric test was adequate due to the more asymmetric distribution and frequency of atypical cases in this set of variables; Bonferroni correction was applied); and (3) Spearman's non-linear correlation coefficient to estimate the degree of association between the changes in the different groups of variables. For all purposes, a two-sided P-value < 0.05 was considered significant. The data analysis was performed using IBM SPSS Statistics version 25.0 (IBM Corp., Armonk, NY, USA).

Results

A total of 18 consecutive patients eligible for orthognathic surgery and diagnosed with mild OSA were enroled in the study. There were 14 males (77.8%) and four females (22.2%), with a mean age of 39.8 ± 10.0 years (range 20–56 years). Preoperatively, 61.1% of the sample presented an underlying Class II DFD and 38.9% a Class III DFD. None of the patients presented alcohol abuse (half were teetotal and half social drinkers), while two of them were active smokers (< 10 cigarettes/day). Only two patients knew they suffered from OSA; both of them had undergone previous septoplasty, and one of them had received a tonsillectomy, however the sleep-related breathing problem persisted. Both used CPAP therapy, but with low adherence. The remaining patients (88.9%) were diagnosed with mild OSA based on the preoperative HSAT findings (Table 1).

Different surgical approaches were used: orthodontics first (55.6%), surgery first (38.9%), and surgery early (5.6%). Most patients underwent bimaxillary surgery (83.3%); single-jaw surgery (mandible) was performed in three patients (16.7%). Specifically regarding the expansive maxillomandibular movements, all patients in the bimaxillary group received mandibular advancement and counterclockwise rotation, 93.3% received maxillary advancement, and 60% maxillary counterclockwise rotation (Fig. 1), while in the single-jaw group, all patients received mandibular advancement. Additionally, an advancement genioplasty was performed in seven patients (38.9%) (Table 1).

The upper airway volume was observed to have increased significantly at 1 month postoperative (mean volume gain 19,794.4 ± 8138.6 mm^3, P < 0.001). Relapse was also statistically relevant, but insufficient to offset the total volume gain (T2–T0: 13,963.8 ± 6496.8 mm^3, P < 0.001) (Table 2). The overall total upper airway increase for the 18 patients at 1 year of follow-up was 46.7%: 28.7% in the nasopharynx, 52.2% in the oropharynx, and 56.8% in the hypopharynx.

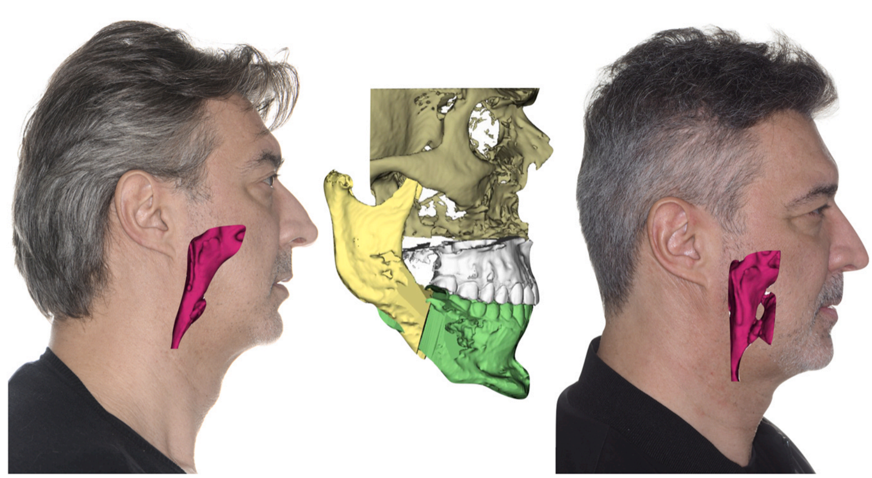

Fig. 1. Clinical case illustrating the facial appearance and upper airway enlargement after orthognathic surgery with maxillomandibular advancement and counterclockwise rotation.

Table 2. Changes in total airway volume and the anthropometric variables ; mean ± SD values.

	T0	T1	T2	T1–T0	T2–T1	T2–T0
Total airway volume (mm^3)	29,875.9 ± 8178.7	49,670.2 ± 11,409.9	44,047.4 ± 8216.5	19,794.4 ± 8138.6 $P < 0.001$***	− 5830.5 ± 4155.9 $P < 0.001$***	13,963.8 ± 6496.8 $P < 0.001$***
Weight (kg)	82.3 ± 16.4	79.9 ± 15.4	80.8 ± 14.7	− 2.38 ± 2.35 $P = 0.001$**	0.82 ± 3.12 $P = 0.845$	− 1.56 ± 3.96 $P = 0.338$
BMI (kg/m^2)	26.6 ± 4.7	25.8 ± 4.5	26.1 ± 4.4	− 0.77 ± 0.72 $P = 0.001$**	0.29 ± 0.97 $P = 0.681$	− 0.49 ± 1.19 $P = 0.298$
Neck perimeter (cm)	37.6 ± 3.7	37.6 ± 3.9	37.5 ± 3.2	0.03 ± 2.97 $P = 1.000$	− 0.06 ± 1.63 $P = 1.000$	− 0.03 ± 2.57 $P = 1.000$

BMI, body mass index; SD, standard deviation; T0, preoperative; T1, postoperatively at 1-month follow-up; T2, postoperatively at 12-month follow-up; T1–T0, difference between 1 month postoperative and preoperative; T2–T1, difference between 12 months postoperative and 1 month postoperative; T2–T0, difference between 12 months postoperative and preoperative. *$P < 0.05$; **$P < 0.01$; ***$P < 0.001$.

Table 3. Polysomnographic changes over time; median (range) values.

	T0	T1	T2	T1–T0	T2–T1	T2–T0
AHI (events/h)[a]	7.7 (6.1–8.5)	4.6 (3.4–5.7)	5.0 (4.1–7.3)	− 2.4 (− 4.0 to − 1.1) $P = 0.018$*	0.1 (− 0.3 to 2.2) $P = 0.448$	− 2.0 (− 2.5 to − 0.1) $P = 0.045$*
ESS score[b]	9.5 (8–11)	8 (7–10)	7 (5–8)	− 1 (− 2 to 0) $P = 0.093$	− 1 (− 2 to 0) $P = 0.072$	− 3 (− 6 to 0) $P = 0.009$**
OSA severity, *n* (%)[c]	No OSA, 0 (0%) Mild, 18 (100%)	No OSA, 10 (55.6%) Mild, 7 (38.9%) Moderate, 1 (5.6%)	No OSA, 9 (50%) Mild, 8 (44.4%) Moderate, 1 (5.6%)	Worse, 0 (0%) Stable, 8 (44.4%) Better, 10 (55.6%) $P = 0.006$**	Worse, 4 (22.2%) Stable, 11 (61.1%) Better, 3 (16.7%) $P = 1.000$	Worse, 0 (0%) Stable, 9 (50%) Better, 9 (50%) $P = 0.009$**

AHI, apnoea–hypopnoea index; ESS, Epworth Sleepiness Scale; OSA, obstructive sleep apnoea; T0, preoperative; T1, postoperatively at 1-month follow-up; T2, postoperatively at 12-month follow-up; T1–T0, difference between 1 month postoperative and preoperative; T2–T1, difference between 12 months postoperative and 1 month postoperative; T2–T0, difference between 12 months postoperative and preoperative. *$P < 0.05$; **$P < 0.01$; ***$P < 0.001$.

[a]Normal < 5 events/h.

[b]Normal < 10.

[c]No OSA, < 5 events/h; mild OSA, 5–14.9 events/h; moderate OSA, 15–29.9 events/h; severe OSA, ≥ 30 events/h.

The changes in anthropometric variables are reported in Table 2. Body weight and BMI decreased significantly during the first month (− 2.38 ± 2.35 kg and − 0.77 ± 0.72 kg/m^2, respectively; both $P = 0.001$), but recovered during follow-up, returning to baseline values. Non-significant changes were observed for the haemodynamic parameters investigated, although an important decrease in minimum blood pressure was observed (from 80.9 ± 10.3 mmHg to 74.4 ± 6.3 mmHg; $P = 0.069$).

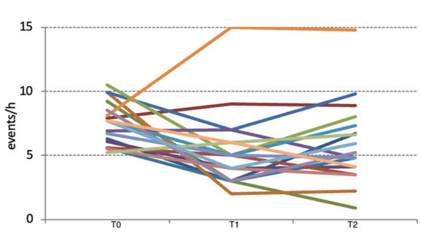

Fig. 2. Graph showing the change in AHI of each patient over time, from preoperative (T0) to 1 month (T1) and 12 months (T2) postoperative.

The changes in polysomnographic variables are summarised in Table 3. The AHI decreased significantly from a median 7.7 events/h at T0 to 4.6 events/h at T1 and 5.0 events/h at T2 (significant change from T0 to T2, $P = 0.045$) (Fig. 2). The ESS score decreased in the same way, from a median 9.5 at T0 to 8 at T1 and 7 at T2 (significant change from T0 to T2, $P = 0.009$). Cure of OSA was documented in 55.6% of the patients at 1 month after the operation ($P = 0.006$), while 50% of the global sample was definitively cured at the 12-month visit ($P = 0.009$).

The correlation analysis between the airway volume and polysomnographic changes showed that the patients with cured OSA after surgery presented significantly larger airway gains. From T0 to T1, the improvement was related to the gain in the total airway ($P = 0.043$), but from T0 to T2, this relationship weakened ($P = 0.236$) (Figs. 3 and 4).

Logistic models were estimated to identify factors influencing the probability of cure, but only the surgical approach was found to be relevant: in patients with mild OSA, the orthodontics first approach (80% of those with cure) increased the odds of cure by 24 times when compared to the surgery first approach (14.4% of those with cure) ($P = 0.018$).

Discussion

The results of this study showed that orthognathic surgery with widening movements of the maxillomandibular complex in patients with mild OSA enlarged the upper airway (+ 46.7%) and significantly decreased the AHI (by a median 2.0 events/h; $P = 0.045$) and ESS score ($P = 0.009$), although the decreases were slight.

Currently, mandibular advancement devices and CPAP are recommended for the management of mild OSA, in addition to changes in habits.[14,19,20] However, the

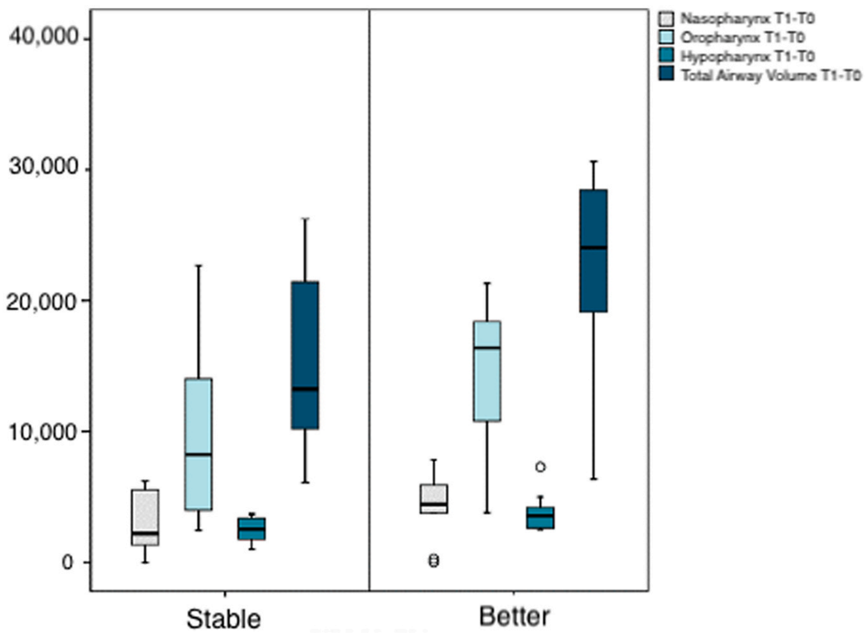

Fig. 3. Distribution of the volumetric changes in the airway according to the change in level of OSA severity during the first month after surgery (T1–T0).

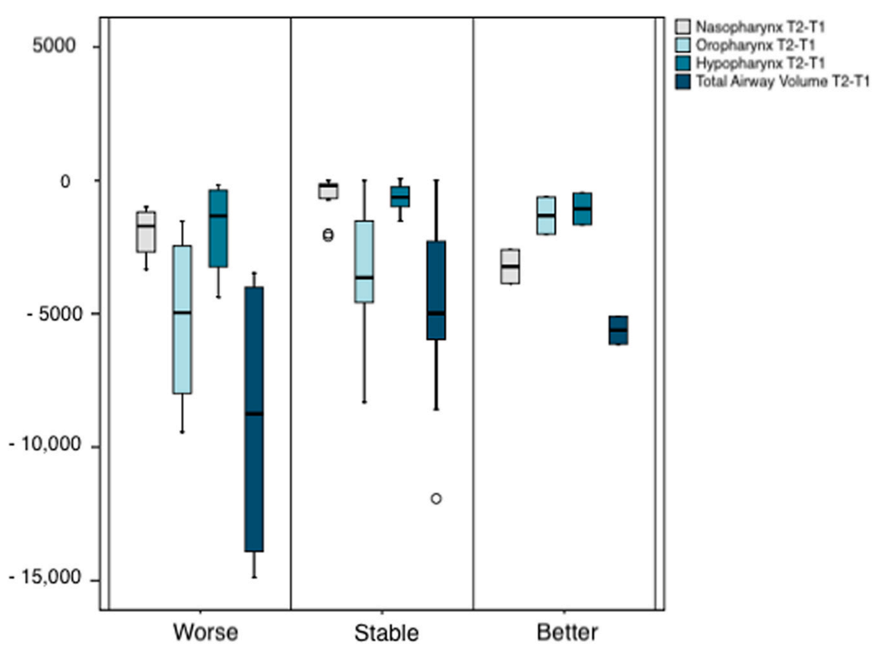

Fig. 4. Distribution of the volumetric changes in the airway according to the change in level of OSA severity during the 1-year postoperative period (T2–T1).

results of the present study suggest that orthognathic surgery could have a beneficial effect on mild OSA in selected patients with an underlying retrusive DFD. Further studies are required to confirm this suggestion. On the other hand, it has been proven in the literature that the upper airway volume decreases rapidly after 30 years of age.[8] Considering the age of the patients in the study sample (39.8 ± 10 years), they would probably experience a reduced upper airway volume over time and therefore a worsened severity of their AHI and OSA. Thus, orthognathic surgery could also be considered as a preventive treatment option in patients at potential risk of suffering sleep-related breathing disorders, or to avoid worsening in those who have already been diagnosed with OSA.

This study is novel in assessing the impact of orthognathic surgery on patients suffering from mild OSA, since orthognathic surgery is currently only indicated in individuals with moderate or severe OSA.[21] Surgical effectiveness in moderate and severe OSA is evaluated in terms of success, defined as a final AHI < 20 events/h and an AHI reduction of at least 50%.[22] The literature has reported a high success rate of orthognathic surgery in patients with moderate and severe OSA, reaching 87.5%.[1] However, when results are focused on the cure rate, which means achieving a final AHI < 5 events/h, the studies on patients with moderate and severe OSA report cure rates between 40% and 50%.[20,23,24] The present study involving a sample of patients with mild OSA obtained a cure rate of 50% at 12 months of follow-up ($P = 0.009$), which is comparable to the results obtained in samples with moderate and severe OSA. On assessing surgical success in mild OSA, only the cure rate can be used, since mild OSA implies a maximum AHI of 15 events/h, not reaching the AHI of 20 events/h considered for the success rate.

On specifically considering the AHI in the study sample, a significant decrease was observed at 1 year after the operation, with the median score at T2 being 5.0 events/h ($P = 0.045$) (Fig. 2). However, one patient showed an increase in AHI from 8.1 events/hour to 15.8 events/h. The final HSAT was repeated and confirmed the worsened AHI value. Unfortunately, the baseline HSAT could not be performed again to corroborate the initial relatively low AHI. Although after surgery the total upper airway volume of this patient increased from 26,021 mm^3 to 42,940 mm^3 at 12 months of follow-up, the patient presented other risk factors including a high BMI (27.73 kg/m^2), active smoker status (< 10 cigarettes/day), and social drinking, which may have had a more relevant role in the OSA disease than their previous underlying DFD.

Surprisingly, most of the patients in this study (88.9%) were not aware that they had mild OSA. This is probably because they did not link their daily fatigue to any sleep-related disorder, apart from the mildness of the OSA symptoms in these individuals. Thus, general recommendations were prescribed once the patients were diagnosed with mild OSA: weight loss, giving up smoking and alcohol use, and resting in lateral decubitus. However, although during the first visit those patients who were obese were advised to lose weight and were referred to the dietician for this purpose, the mean BMI of the sample at T0 (26.6 ± 4.7, range 19.6–40.4 kg/m^2) (Table 1) was higher than the normal range (18.5–24.9 kg/m^2),[25] and was higher than 25 kg/m^2 in 66.7% of the sample. Although the mean BMI did not change significantly over the study period, it could have introduced some bias to the upper airway volume and AHI outcomes, since obesity is also related to OSA.[25]

Regarding the baseline skeletal profile of the patients, it should be noted that most of them presented with an underlying Class II DFD (61.1%), which is the typical facial pattern in patients suffering from OSA. However, the remaining patients (38.9%) presented a Class III deformity. This means that although their mandible was longer in the sagittal plane than their maxillary bone, they presented a bimaxillary retrusive skeletal facial profile. Therefore, all patients received maxillomandibular widening surgery in terms of advancement and/or counterclockwise rotation. The logistic model analysis revealed a higher probability of cure when the orthodontics first approach was used instead of the surgery first approach ($P = 0.018$). This could be because when orthodontic decompensation is performed before surgery, it is usually possible to perform a wider skeletal movement during surgery.

This study has some limitations, such as the small sample size, the single-centre design, and the presence of confounders including smoking, alcohol use, and obesity, with the inherent biases involved. Moreover, quality of life and the polysomnographic outcomes compared to other treatments such as mandibular advancement devices or CPAP were not assessed.

In conclusion, despite the limited sample size, this study provides evidence that in patients with an underlying retrusive DFD who are suffering from mild OSA, a slight reduction in the number of apnoea–hypopnoea events could be added as a beneficial effect following orthognathic surgery, due to the upper airway enlargement achieved, with a mild OSA cure rate of 50%.

Ethical approval

This study was approved by the Ethics Committee of Teknon Medical Center (Barcelona, Spain) (Ref. 2020/06-CMX-CMT).

Patient consent

Written informed consent was obtained from all patients prior to surgery.

Funding

None.

Competing interests

None.

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