clinical OD **Case Study**


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Treatment of Obstructive Sleep Apnea With Maxillomandibular Advancement

Introduction

Obstructive sleep apnea (OSA) is caused by a complete or partial obstruction of the airway during sleep, resulting in sleep disturbance and daytime somnolence.[1–3] Some suggest that the subsequent reduction of cognitive function from OSA is a major contributor to motor vehicle crashes and occupational accidents.[4] Further, untreated OSA may exacerbate poorly controlled disease and has been associated with diabetes, stroke, hypertension and cardiovascular morbidity and mortality.[4,5] The negative and sometimes devastating effects of OSA makes it an important quality of life, clinical and public health issue.[6,7] Dentists and dental specialists play an important role in the multidisciplinary treatment of this complex sleep disorder. The aims of this paper are two-fold. First, background information on OSA with regard to its pathophysiology, prevalence, signs and symptoms, diagnosis and treatment will be provided. Second, this paper will highlight the treatment of a patient with severe OSA using maxillomandibular advancement (MMA).

Pathophysiology of OSA

During sleep, patients with OSA have repetitive periods of apnea, defined as a cessation or near cessation (> 50 percent) of airflow from baseline, regardless of oxygen saturation.[8] Some patients may also exhibit periods of hypopnea, defined as: 1) ≥ 30 percent decrease in airflow from baseline lasting a minimum of 10 seconds and with ≥ four percent desaturation from baseline, or 2) ≥ 50 percent decrease in airflow with ≥ three percent desaturation or arousal from sleep.[8] Apnea and hypopnea are the direct result of pharyngeal collapse, secondary to relaxation of the musculature during periods of sleep. Increased inspiratory efforts from pharyngeal collapse and reduction in oxygen saturation are accompanied by the characteristic loud snoring, gasping and waking at night. Repeated awakenings cause fragmentation of normal sleep and are often not noticed by the patient. The cyclical nature of this sleep pattern results in the typical symptoms of OSA.[8]

Prevalence

The majority of OSA prevalence studies have been done in Western populations; thus, the worldwide importance of OSA, as well as racial and ethnic prevalence patterns, is not well understood.[4] However, a review found that roughly one of every five adults had at least mild OSA and one of every 15 had at least moderate OSA.[4] The authors of the review also estimated from population-based studies a two- to three-fold greater risk of OSA for men compared with women. Further, it was estimated that five percent of adults

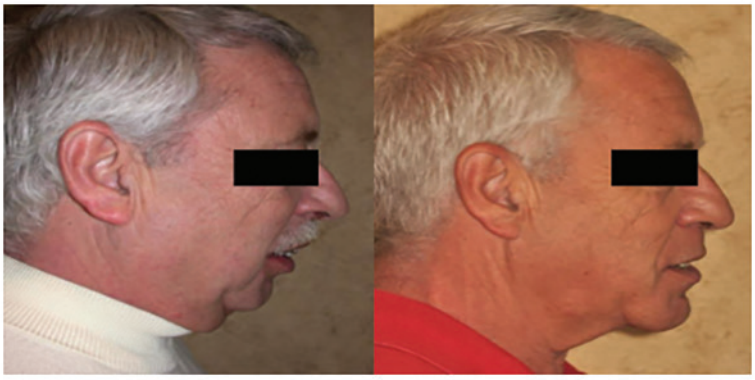


Figure 1.
Pre- and post-operative profile view photographs depicting changes from a retrognathic to orthognathic profile.

in Western countries were likely to have undiagnosed OSA.[4]

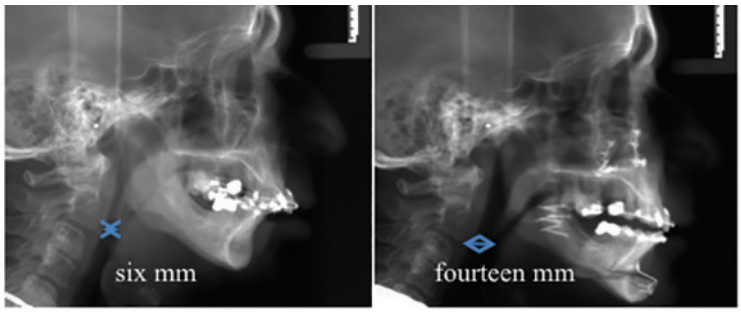
Signs and Symptoms

Many common physical findings in the head and neck region include nasal obstruction, hypertrophic tonsils, adenoid hypertrophy, hypertrophic uvula, hyperplastic soft palate, macroglossia, retrognathism, micrognathism and a neck circumference of greater than 17 inches.[9] Other physical conditions that may present in OSA patients include gastroesophageal reflex, hypertension, truncal/central obesity, nocturia, diabetes and coronary artery disease.[4,5] Symptoms include snoring, witnessed apneas, daytime somnolence, morning headaches, nocturnal gasping, restless sleep, insomnia, nightmares, irritability, memory impairment, impaired concentration/attention, depression, and impotence.[9]

Diagnosis

Diagnosis is done by physicians and starts with a comprehensive medical history and exam of the head, neck, respiratory and cardiovascular systems. Validated questionnaires including the Epworth Sleepiness Scale[10] and STOP-Bang[11] aid in identifying patients with OSA. Computed tomography (CT) scans, complimented with cephalometric radiographs, can volumetrically assess airway patency.[12] However, the gold standard for OSA diagnosis is polysomnography in a sleep medicine clinic.[13]

OSA severity is measured by the apnea-hypopnea index (AHI) and the respiratory disturbance index (RDI). However, due to limitations and methods of measurements of the RDI, the AHI is more commonly used. The American Academy of Sleep Medicine's OSA classifications[14] are: 1) mild OSA – AHI of 5-15; 2) moderate OSA – AHI 15-30; 3) severe OSA – AHI > 30.



Pre-operative Post-operative

Figure 2.
Pre- and post-operative lateral cephalometric radiographs demonstrating a substantial increase in PAS from six mm to 14 mm.

Treatment

Traditionally, OSA has been treated by a variety of conservative modalities including, but not limited to, oral appliances, continuous positive airway pressure (CPAP), bilevel positive airways pressure (BiPAP), automatic continuous positive airway pressure (autoCPAP), and lifestyle and diet modification.[9] Although these can be effective, many patients fail conservative treatment due to poor compliance. When these options fail, health care providers can look at surgical options, classified as Phase 1 and Phase 2 surgery.[15]

Phase 1 surgery is traditionally done by Otolaryngology (ENT) and Sleep Medicine medical specialists with success rates of 40-55 percent.[15] Treatment modalities include nasal surgery, tonsillectomy, uvulopalatopharyngoplasty (UPPP), radiofrequency ablation, suprahyoid myotomies and partial glossectomy.[15] Many patients demonstrate some improvement of their OSA after Phase 1 surgery; however, much of this treatment tends to fail, as it does not always address the level of obstruction.

Phase 2 surgery or maxillomandibular advancement (MMA) involves the interdisciplinary care of orthodontists, sleep medicine physicians and oral and maxillofacial surgeons. This typically involves a period of pre-surgical orthodontics and pre-operative work-up, similar to that of patients undergoing orthognathic surgery for dentofacial deformities. Once this phase and the final planning is completed, the patient can move to the surgical phase. Surgery includes advancement of the maxilla via Lefort 1 osteotomy (advancing the soft palate and palatoglossus muscles), advancement of the mandible via bilateral sagittal split osteotomy (BSSO) (advancing the anterior belly of the digastric, mylohyoid, geniohyoid and genioglossus muscles) and advancement of the chin via genioplasty (advancing the suprahyoid muscles).[16] The surgical advancement of the osseous architecture results in significant dimensional changes in the nasopharynx, oral pharynx and laryngopharynx, creating a much larger posterior airway space (PAS).[12] These volumetric changes at all three levels of the upper airway space are the reason for the high rates of surgical success, defined as AHI < 20, and OSA cure, defined as AHI < 5,[3] with accompanying long-term surgical stability.[17]

Case Study

A 57-year-old business executive presented to an oral and maxillofacial surgeon (MC) with a chief complaint of "I hate my life" (Figure 1). He was suffering from severe OSA with a polysomnography-confirmed AHI of

45, a six mm PAS from cephalometric analysis (Figure 2) and minimal upper airway space from analysis of the CT scan (Figure 3). He also had uncontrolled hypertension. Treatment modalities that had failed included diet/lifestyle modification, oral appliances and CPAP. Phase 1 surgery was subsequently performed by an otolaryngologist. The surgery included nasal septoplasty and UPPP. Due to a lack of resolution or change in the severity of his OSA and a desire for a better quality of life, he pursued further surgical options to help alleviate the burden of his disease.

Treatment was planned by an orthodontist and MC. After 18 months of orthodontics to level and align the teeth and coordinate the arches, the patient was ready for MMA. The surgery consisted of a Lefort 1 advancement, a BSSO advancement and a genioplasty advancement. This was tolerated well without complications in the post-operative phase. Orthodontics continued for six months to provide the final detailing and finishing in a pre-planned end-to-end occlusion. He then underwent a second sleep study. His AHI had decreased to 15 (surgical success) and his PAS had increased to 14 mm on lateral cephalometric evaluation (Figure 2). He also had a substantial increase in his upper airway based on the post-operative CT (Figure 3). At 59 years of age and treatment completion, the patient lost 40 lbs (18.2 kg). His systolic and diastolic blood pressures decreased by 15 and 25 points, respectively. He subsequently retired from work and subjectively felt like a new person (Figure 1). With regards to his OSA, he continues to progress well to date.

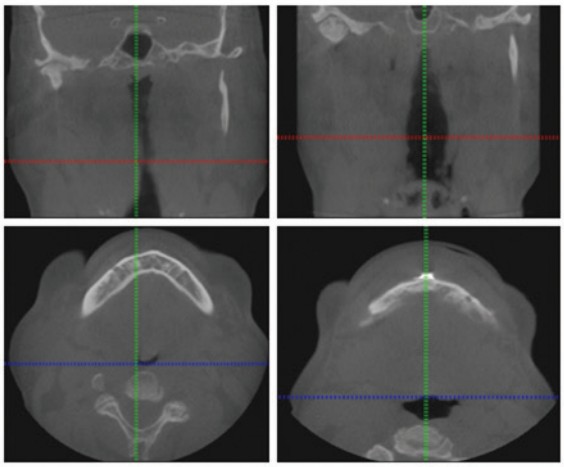


Figure 3.
Pre- and post-operative coronal and axial cone beam CT cuts demonstrating a significant increase in upper airway dimensions.

Discussion

OSA continues to be prevalent in society[3] and the literature suggests that change in severity, mostly toward progression, does occur in individuals with mild or moderate OSA.[4] Thus, it is important to initiate interventions to reduce or reverse OSA progression before the development of significant morbidity. Nevertheless, the patient in this report had failed both conservative management and Phase 1 surgery due to poor compliance and treatment that was not directed at the cause of his problem. MMA is effective in treating severe OSA, with recent literature suggesting an 85.5 percent success rate (50 percent reduction in AHI to less than 20 events per hour) and a 38.5 percent cure rate (AHI to less than five events per hour).[3] Statistically significant improvements in total airway volume and overall airway dimensions are demonstrated with MMA.[18] Large advancements of the maxillomandibular complex can further reduce airway collapse during sleep.[1] After a 10 mm advancement, our patient's AHI decreased to 15, his posterior airway space increased dramatically (Figure 2 and Figure 3), and his quality of life improved substantially. A minimal amount of relapse is expected with these patients due to the large movement; however, the changes are considered clinically minor and skeletal stability is preserved.[17]

Conclusions

MMA is an effective surgical option for patients with severe OSA, with the potential to reduce daytime sleepiness and significantly improve quality of life.[2,19,20] Treatment should be individually tailored in order to appropriately target the level of obstruction and to alleviate the associated systemic health issues. Given that conservative treatment can fail, MMA can be considered as first line surgical therapy in cases of severe OSA.

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