

Orthodontics & Obstructive Sleep Apnoea PART 2

Obstructive Sleep Apnoea (OSA) is characterised by a collapse of the upper airway leading to potential complete cessation of airflow. It's wide range of causes were discussed in Part 1.

Orthodontic treatment has been suggested as a potential pathogenetic factor for OSA, in particular, extractions.

Orthodontic extractions and OSA

At present, there is a lack of evidence to suggest that conventional orthodontic treatment is an aetiological factor in the development of OSA¹.

Extractions have been implicated as a potential cause of OSA. A reduction in the size of the airway, it has been postulated, occurs as the anterior dentition is retracted due to posterior displacement and encroachment of the tongue into the airway space, such as has reportedly occurred after extractions in bi-dentally protrusive patients. Reductions in the cross-sectional area of the oropharynx have been noted in such adults³ – however, there is considerable misinformation about this airway related sequelae¹.

However, bi-dental protrusion is not a common malocclusion. Extractions are more frequently required for patients presenting with significant crowding or other malocclusions. In these, there has been no significant change in airway dimensions⁴. Arch width, length and perimeters have been shown to either increase, decrease or remain unchanged following extractions, leaving the argument that extractions may cause OSA unsubstantiated in the vast majority of cases⁵.

Rapid bodily growth and changes occurring during adolescence will also significantly influence airway dimensional changes. This is highlighted by studies showing airway changes in children and adolescents, with and without extractions, which have been reported to increase following orthodontic treatment ^{6,7}. In addition, complex neuromuscular control of the airways during sleep, which may protect against OSA despite a reduced airway dimension, further complicates the discussion and highlights that changes in airways are multifactorial⁸.

Treatment of OSA - Orthodontic Appliances

The orthodontic management of OSA varies according to causative factors, and can range

from simple intra-oral appliances to more complex surgical management.

Maxillary Expansion

Common skeletal discrepancies known to increase the risk of OSA include retrognathia of the maxilla and/ or mandible. It is logical to assume that expanding or protracting the maxilla may reduce airway resistance and improve the symptoms of OSA.

Maxillary constriction is considered a risk factor for OSA as it may increase nasal resistance, alter tongue posture and be associated with more narrow oropharyngeal airways. Orthopaedic maxillary expansion in growing children has been used in the treatment of maxillary constriction. Evidence has shown that opening of the mid palatal suture during expansion displaces the lateral walls of the nasal cavity, leading to increases in the nasal cavity bone width and volume and reducing the resistance to nasal flow. This has the potential to assist in the management of OSA^{9, 10, 11, 12, 13, 14}. A higher tongue posture may also occur post lateral expansion, increasing the upper airway volume.

Numerous studies have identified the potential for maxillary expansion to reduce the severity of OSA and improve the quality of life¹⁵, with one study illustrating a reduction in mean AHI from 12.2 to less than 1 in a group of 6 to 12 year olds¹⁶. It was stressed in these studies, however, that improvements found after maxillary expansion were in children who did not have existing adenoid-tonsillar hypertrophy.



Figure 1 - Rapid Maxillary Expansion (RME) appliance

In adolescent populations, mini-screw assisted maxillary expansion (MARPE) has been gaining popularity and exhibits significant potential for long term increases in nasopharyngeal volume¹⁷. However long term studies are yet to confirm this.

Surgically Assisted Rapid Maxillary Expansion (SARME) in adults with OSA may be a viable option if there are concurrent maxillary transverse discrepancies identified as part of a thorough orthodontic exam. SARME has been shown to improve some Polysomnography (PSG) parameters¹8 in patients – however, it is important to remember that OSA alone, just as with orthognathic surgery, is not the only determinant for consideration of SARME. SARME may produce substantial short term volume increases in the nasal cavity, however, studies investigating this concept were noted to be at high risk of bias, and the effect of SARME on respiratory function is yet to be determined¹9.

In summary, at this time there is no indication that prophylactic maxillary expansion prevents the development of OSA in the future.

Functional Appliances

Protraction of the mandibular arch via functional appliances or advancement devices may increase the patency of the oropharyngeal airway (figure 2). However, there is no firm evidence in the contemporary literature to suggest that prophylactic orthodontic protraction of the mandible/mandibular arch alone will cure OSA. Given the multifactorial aetiology of OSA, and the fact that functional appliances do not 'grow' mandibles with any clinical significance, this is hardly surprising.

Headgear appliances, used for skeletal and dental effects, do not exhibit significant changes on the volume of the upper airway and thus do not pose an increased risk of OSA¹.

Therefore, the current recommendation is that they may be recommended only if an underlying skeletal discrepancy or malocclusion exists, for which there is an orthodontic indication for such advancement appliances, with the potential additional benefit of improving the airway patency and OSA parameters.



Figure 2 - Mandibular advancement device maintaining airway patency (Source: Division of Sleep Medicine at Harvard Medical School.)

Splints

Nocturnal Mandibular Advancement Splints (MAS) are used in the management of OSA, particularly in patients with mild to moderate OSA who have intolerance to Positive Airway Pressure (PAP) devices²⁰. They hold the mandible and associated soft tissues forward, resulting in an increased size of the upper airway at the oropharyngeal level. However, long term use may alter the occlusion, particularly proclination of the lower anterior teeth. In patients with minimal overjet, this may result in an anterior crossbite. Reductions in overbite and the development of posterior open bites may also occur. A tongue stabilising device (TSD) is another appliance that used suction to protrude the tongue forward and thereby increasing upper airway structure and function. The literature supports that TSD's can yield a mild improvement in AHI scores, however it is much less effective than MAS and requires further research to evaluate its role in the management of OSA²¹.





Figure 3 & 4- Mandibular Advancement Splint (MAS) and Tongue Stabilising Device (TSD) ²¹

Long term use of PAP therapy has been implicated in affecting the development of the craniofacial skeleton^{22,23}. Consistent wear of the typical nasal mask with PAP devices may act as a maxillary retrusion appliance, restricting any potential growth in the maxilla and negatively influencing the nasopharyngeal airway dimension. Care must be taken when such devices are used long-term in a growing patient.

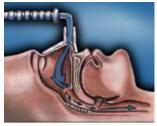




Figure 5- Positive Airway Pressure (PAP) therapy (Source: Mayo Clinic)

Therefore, where the use of an extra-oral or intra-oral appliance is suggested by a sleep physician it must be closely monitored. An orthodontist may also need to be consulted before commencement of treatment or to recover any unwanted side effects.

Orthodontists may encounter patients who are already being treated for OSA with oral appliances and must be aware that the patient may no longer be able to wear such an appliance during orthodontic treatment. In this case, the patient will need to revert to PAP therapy - hence, communication with their treating physician is essential to ensure the patient is being appropriately managed.

Treatment of OSA- Orthognathic Surgery

The link between orthognathic surgery and obstructive sleep apnoea has long been debated. Of particular concern is the link between mandibular Bilateral Sagittal Split Osteotomy (BSSO) set-back surgery and its impact on the pharyngeal airway dimension. Some studies highlight that the retrolingual airway dimension does decrease after mandibular setback surgery²⁴ – however, there was no association with any significant changes in snoring incidence or apnoea events²⁵. Other studies have found that mandibular BSSO set-back of less than five millimetres does not increase the risk of OSA. According to a STOP-BANG questionnaire, however, movements greater than or equal to 5mm may increase the risk²⁵. Another review also supported the decrease in posterior airway volume, and noted that if a patient exhibits other risk factors such as obesity, a short neck or large tongue, a mandibular BSSO set-back procedure may predispose a patient to OSA²⁷.

Conversely, it was found that mandibular BSSO advancement surgery did improve sleep quality, but only in patients presenting with pre-existing sleep disorders²⁸.

Bimaxillary advancement surgery, on the other hand, has been shown to have beneficial effects in terms of upper airway size, AHI scores and quality of life measured on the Epworth sleepiness scale ^{29,30} and is comparable to those under ventilation therapy^{31, 32, 33}. Patients with severe OSA who are unable to tolerate PAP devices/intra-oral splints, or who have had failed conservative therapy and have an underlying sagittal skeletal discrepancy, may be candidates for maxillomandibular advancement.

Final Remarks

Obstructive sleep apnoea is a multifactorial syndrome affecting children and adults differently. The role of the dental professional, with their extensive training and regular patient contact, is to identify at risk patients. There are multiple methods by which dentists and orthodontists may assist patients with OSA – however, it should ideally be in collaboration with, and at the direction of, a sleep physician.

References available upon request





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