

Oral appliances in sleep apnea

Dr Clyde Keevy BChD (Pret)

General Dental Practitioner: In private practice Morningside, Sandton

Sleep-disordered breathing (SDB) spans a continuum from snoring through upper airway resistance syndrome (UARS) to obstructive sleep apnea-hypopnea syndrome (OSAHS).¹ When we look at the treatment of Sleep-disordered breathing and Obstructive Sleep Apnea (OSA) we need to remember that the main aim of the treatment is the removal of the obstruction and to volumetrically increase the pharyngeal airway space.

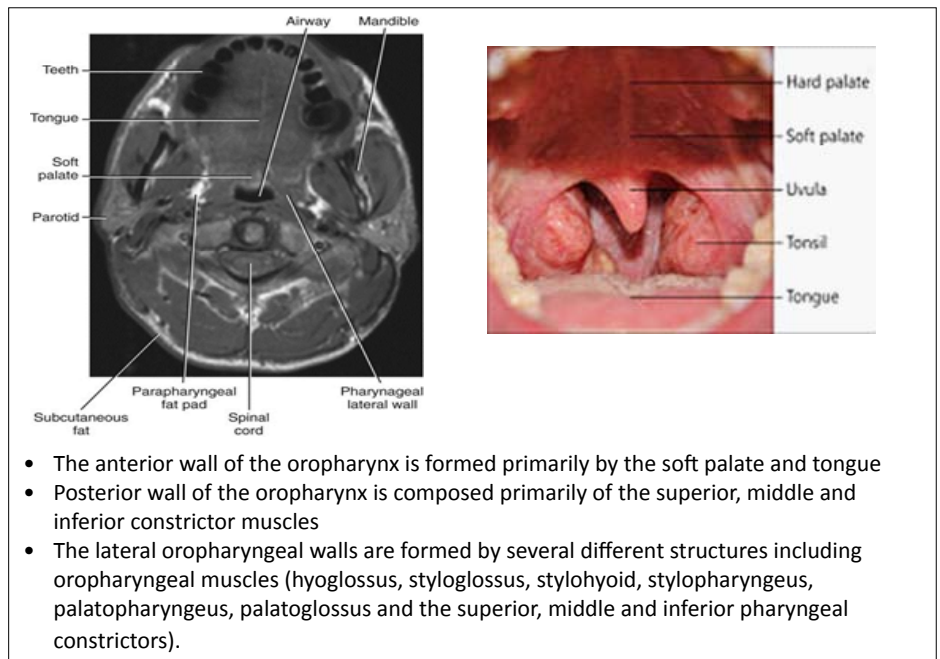
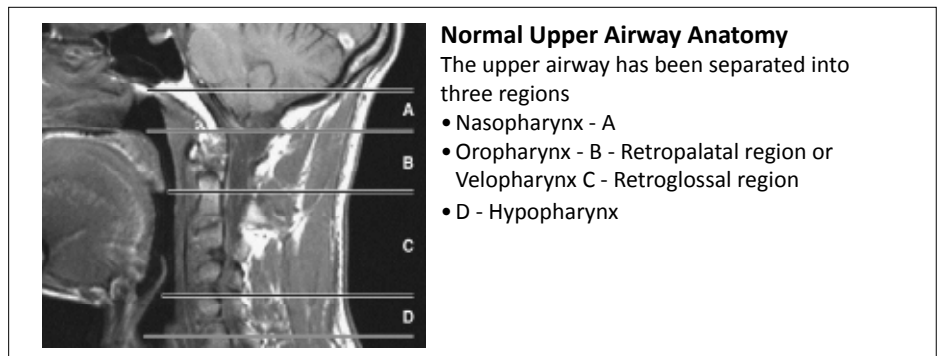
Pharyngeal patency is critical as a conduit for airflow

- With the exception of the upstream and downstream ends of the entire respiratory airway tract (the nares and the small intrapulmonary airways) the pharynx is the only collapsible segment of this anatomically defined system
- Normally, the pharynx remains open at all times, except during momentary closures associated with swallowing, regurgitation, eructation, and speech.
- The sleep state is associated with a decrease in neuromotor output to pharyngeal muscles.

The patency of the pharynx is controlled by dilator and constrictor muscles without any rigid bony structures to help control patency. Muscles start relaxing during Non-REM Stage 3 sleep and in REM sleep they are in a state of muscle paralysis also called REM atonia.

Without any rigid structures to control patency the collapse of the pharyngeal airway during inspiration gives rise to a drop in intra-pharyngeal pressure and the development of pharyngeal closure and obstruction. Pharyngeal closure can be partial or complete leading to Upper Airway Resistance Syndrome (UARS) or OSA respectively.

These airway obstructions can cause recurrent arousals from sleep, ultimately resulting in excessive daytime sleepiness, neurocognitive impairment, a higher risk of motor vehicle accidents as well as cardiovascular and metabolic disease events.²



How are we treating?

1. Multidisciplinary

- To improve upper airway patency during sleep, a variety of treatment options, ranging from non-invasive to surgical, are available.
- A multidisciplinary approach to the treatment and possible causes that might need different approaches is recommended. Many patients suffering from OSA may have other medical conditions and it is therefore vitally important that all the relevant medical professionals be associated with the treatment such as:
 - Neurologists
 - Ear Nose and Throat Surgeons
 - Cardiologists
 - Physicians
 - Psychologists
 - Dentists

2. Positive Air Pressure- PAP, CPAP, AutoPAP, BiPAP etc (Gold Standard)

Continuous positive airway pressure (CPAP) is generally considered the treatment of first choice in severe OSA cases. Compliance with this relatively intrusive therapy varies from poor to very good.³

3. Oral Appliances, Mandibular Advancement Device (MAD)

Most oral appliances used in a clinical setting are mandibular advancement devices which keep the mandible and its attached musculature in a protruded position.²

The mandibular advancement device seems to be a promising treatment alternative to conventional Continuous Positive Airway Pressure

(CPAP) therapy.³ There are two main types of appliances:

- Active Appliance - Custom Made
- Static Appliances - Home Made or Custom Made

4. Combinations of PAP and MAD

Combination therapy of MAD and nasal CPAP is effective in normalising respiratory disturbances of sleep apnea in selected OSA patients who are intolerant to CPAP.⁴

Results of studies with different MAD

Different studies have shown the efficacy of treatment of OSA with an oral appliance.

1. A total of 75 patients had polysomnography at baseline and with the appliance. A significant reduction in AHI from 44 ± 28 events/h to 12 ± 15 events/h ($p < 0.0005$) and a reduction in the arousal index from 37 ± 27 events/h to 16 ± 13 events/h ($p < 0.05$) was found. Epworth scores fell from a mean of 11 ± 5 to 7 ± 3 ($p < 0.0005$).⁵
2. A total of 14 Chinese subjects (8 males, 6 females) diagnosed with OSA by overnight polysomnography (PSG), were fitted with the MAD. The mean \pm standard deviation baseline apnoea-hypopnoea index (AHI) was 38.4 ± 17.2 and minimum arterial oxygen saturation (SaO₂) was $75.5 \pm 11.1\%$. At the second polysomnogram, AHI was significantly reduced to 10.9 ± 14.7 . Minimum SaO₂ was significantly increased to $86 \pm 8.4\%$.³
3. Fourteen of 15 subjects demonstrated significant improvement in the degree of OSA, based on the apnea-hypopnea index (AHI) (34.7 ± 5.3 to 12.9 ± 2.4 events/h, $p < 0.002$).⁶
4. Twenty-seven patients (20 male, 7 female), were recruited from a tertiary hospital sleep clinic. The apnea-hypopnea index (AHI) was reduced with mandibular advancement splint (MAS) (11.68 ± 8.94 , $P = 0.001$) and tongue stabilising device (TSD) (13.15 ± 10.77 , $P = 0.002$) compared with baseline AHI (26.96 ± 17.17). The arousal index decreased for MAS (21.09 ± 9.27 , $P = 0.004$) and TSD (21.9 ± 10.56 ,

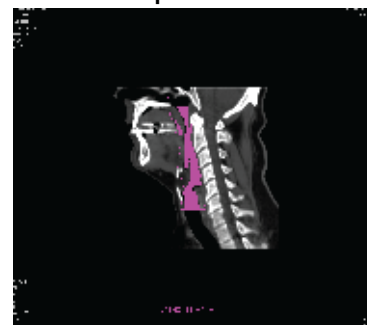
$P = 0.001$) compared with baseline (33.23 ± 16.41). Sixty-eight percent of patients achieved a complete or partial response with MAS, compared with 45% with TSD. The Epworth Sleepiness Scale (ESS) score was decreased with MAS ($P = <0.001$) and TSD ($P = 0.002$). Subjective improvements in snoring and quality of sleep were reported, with a better response for MAS than TSD. Compliance was poorer for TSD, and the side effect profiles of the 2 modalities were different. All patients were satisfied with MAS compared to TSD, and 91% of patients preferred the MAS.⁴

5. The effects of a combination of CPAP and MAD was tested. The combination of MAD and nasal CPAP was well tolerated by all participants. Compared to CPAP alone, the optimal CPAP pressure required to eliminate all obstructive events on the combination therapy was reduced from 9.4 ± 2.3 to 7.3 ± 1.4 cm H₂O ($p = 0.001$). The residual apnea hypopnea index on the MAD alone decreased from 11.2 ± 3.9 to 3.4 ± 1.5 on the combination therapy ($p < 0.001$). The number of oxygen desaturations was also less with the combination therapy than with MAD ($p < 0.001$). Both the MAD and the combination therapy were effective in reducing daytime sleepiness from 12.7 ± 2.1 at baseline to 9.7 ± 3.1 ($p = 0.04$) and 7.5 ± 4.1 ($p = 0.007$), with MAD and combination therapy respectively.

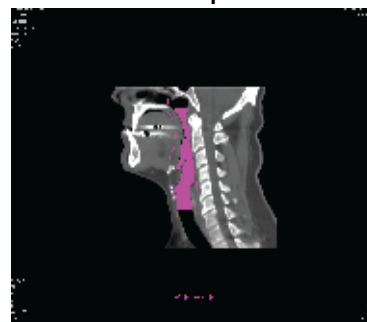
They conclude that although their study provides no insight into the mechanism by which the combination therapy achieves its desired effect, it is plausible that by improving the patency of the velopharyngeal segment of the upper airway through mandibular advancement, the CPAP unit needs less pressure to maintain patency. The inability of MAD to normalise AHI on its own, particularly in the supine position, suggests also that the addition of CPAP to oral appliance therapy may increase lung volumes and hence reverse upper airway collapsibility during non-REM sleep in patients with OSA. Irrespective of the underlying mechanism, the combination of CPAP and MAD device clearly reduced the optimal CPAP pressure by 29% while reducing the AHI by 86% from baseline.⁷

VOLUMETRIC INCREASE IN PATIENT WITH ACTIVE MANDIBULAR ADVANCEMENT DEVICE (KEEVY APPLIANCE)

pre-MAD

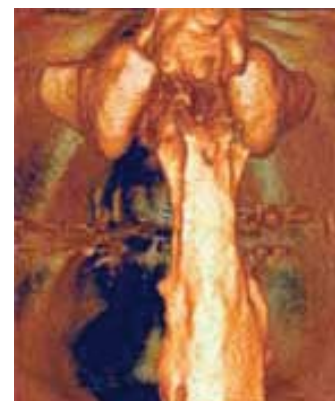


MAD in place



There is a 12.657cm^3 or 32% volumetric increase in the Oro- and Laryngopharyngeal space.

Three-dimensional upper-airway changes associated with various amounts of mandibular advancement in awake apnea patients



Conclusion

Most patients with sleep apnea are being offered nasal Continuous Positive Airway Pressure (CPAP) as the treatment of choice. However, compliance with nasal CPAP varies, and is particularly poor in non-apneic snorers, patients who suffer from UARS and those with mild to moderate sleep apnea. Oral appliances constitute an attractive non-invasive alternative for patients with sleep apnea, provided the efficacy, compliance, long-term tolerance, and satisfaction with these appliances are established.⁵

A very important aspect of a successful appliance is the patient's comfort. To achieve this goal the appliance must be custom-fitted for the patient to minimise the discomfort.

The use of a MAD device has been shown in various studies to have a significant effect in reducing AHI, reducing RDI, increasing arterial oxygen saturation and reducing Epworth scores. The use of a MAD also produces a volumetric increase in the pharyngeal airway space and helps to splint the airway during advancement

of the mandibula. This helps to eliminate the effect of collapse of the pharynx during muscle relaxation. It also has an influence on reducing patient sleepiness and improving the overall wellbeing of patients.

The use of MAD independently or in combination with a CPAP should therefore be considered as treatment option for all patients with SDB, as independent treatment or in combination with CPAP.

Dr Keevy developed and patented an Active MAD for the use in SDB.

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