IN BRIEF

- Obstructive Sleep Apnoea (OSA) requires a multidisciplinary approach from the outset as compared with a step-by-step interdisciplinary approach.
- Randomised control trials support the use of a mandibular advancement appliance in selected cases of OSA.
- GDPs should be aware of signs and symptoms of OSA and manage the patient by referring to the chest physician for a definitive diagnosis.

The role of orthodontics and oral and maxillofacial surgery in the management of obstructive sleep apnoea — a single case report

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Obstructive sleep apnoea (OSA) is a potentially fatal breathing disorder, yet is widely under-diagnosed. It is a multifactorial condition associated with high morbidity, a degree of mortality and is associated with an increase risk of car crashes. A case is presented which demonstrates the inter-disciplinary approach to this condition and the successful treatment through mandibular advancement by orthodontic means followed by orthognathic surgery. The experience of this patient illustrates the importance of the disciplines of orthodontics and oral and maxillofacial surgery in the multidisciplinary management of these patients.

Epidemiological studies in the UK estimate that 5.7% of men and 1.2% of women in the age group of 35-69 have obstructive sleep apnoea/hypopnoea syndrome.[1] This syndrome is characterized by repeated episodes of complete or partial upper airway obstruction during sleep, leading to a diminished airflow to the lungs. Each episode lasts 10–30 seconds.[2] Obstruction is generally caused by the soft palate and/or the base of the tongue collapsing against the pharyngeal walls, thereby narrowing the oropharyngeal inlet. A combination of anatomical (ie craniofacial anatomy) and/or pathophysiological factors (ie functional impairment of the muscles that dilate the upper airway) has been implicated in the aetiology of OSA.[2]

The risk factors associated with this condition are age, sex, obesity, genioglossus tonicity, pharyngeal flaccidity and lung compliance.[3] The prevalence of OSA is highest in middle-aged males with a predisposition to obesity.[4] Certain facial types have been identified as being at risk. Cephalometric parameters have been used to identify patients who are anatomically compromised.[4]

It has been proposed that three categories of OSA obstruction exist, based on the anatomical site of the obstruction.[3] each reducing the posterior airway space (linear measurement between base of tongue and posterior pharyngeal wall, as measured in the saggital plane of a lateral cephalogram). Type I oropharynx obstruction is characterized by a large soft palate or a large hard palate. Type II is a combination of oropharyngeal obstruction and hypopharyngeal obstruction: ie Type I & Type III. Type III hypopharynx obstruction presents as a retrognathic mandible with posterior positioning of the tongue and or macroglossia.[3]

The common clinical findings of sleep-disordered breathing include pronounced snoring with repetitive brief arousals from sleep and producing excessive daytime somnolence. Medical complications secondary to recurrent nocturnal hypoxia and hypercapnia can include hypertension, arrythmias and cerebrovascular disorders.[5] The common clinical signs, which may be associated with this condition, include obesity, a large neck circumference, excessive fat deposition in the palate, an enlarged tongue, a long soft palate and a retrognathic mandible.[2]

Although this syndrome could be suspected by a dentist from the clinical findings, the definitive diagnosis should be confirmed by a physician before commencing any form of treatment. A diagnosis can be made from the clinical history, Epworth Sleepiness Scale (ESS) and an overnight-approved sleep study (eg polysomnography).

The ESS is based on a questionnaire completed by the patient, which includes eight questions related to the likelihood of daytime somnolence under varied circumstances. This methodology is regarded as both subjective and weak in its prediction of the severity of this condition.[6]

Polysomnography is the only definitive measure to quantify the presence and severity of OSA. It detects a drop in oxygen saturation and fluctuations in pulse rate reflecting surges in blood pressure, all of which are recorded on a tracing. However,

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polysomnography is a very expensive diagnostic tool.[6] Therefore while polysomnography is regarded as the gold standard for diagnosis of OSA, many clinics now use the simpler and less expensive technique of pulse oximetry.

The management of these patients is best carried out by a multidisciplinary approach. Behavioral management includes the elimination of aggravating factors: for instance treating the obesity, reducing alcohol consumption, smoking cessation, changing body position during sleep and a reduction in central nervous system depressants. Medical management normally involves the application of continuous positive airway pressure (CPAP) by mechanical means.

Two surgical approaches have been described:

1. Pharyngeal surgery normally involving uvulopalatopharyngoplasty (UPPP) that is soft tissue surgery to the soft palate or
2. Orthognathic surgery, which is bony surgery directed at altering the skeletal bones of the face.

Riley *et al*. evaluated patients and concluded that there is an excellent chance of correcting the obstructive process using orthognathic surgery. It proved to be as effective as CPAP. Presurgical evaluation, which includes polysomnography, physical examination, cephalometric analysis and fiberoptic pharyngoscopy is crucial in determining the site of obstruction.[7] Orthodontic management relies upon the elimination of local factors that may prevent the jaw adopting a forward resting position and the use of intraoral mandibular advancement appliance (MAA).[4] The method of treatment selected is dependent upon the site of the obstruction and may involve one or more combinations of the above treatment modalities.

The examination prior to determining the treatment approach should exclude pathologic entities of upper airway (eg neoplasms, cysts) as well as identifying the site of obstruction.

In view of the range of management options, patients with this condition require a multi-disciplinary approach to determine the most appropriate treatment plan, targeted at relieving the site of obstruction. Ideally clinicians from the specialties of chest medicine, ear nose and throat surgery (ENT), orthodontics and oral and maxillofacial surgery should work collaboratively.

The aim of this paper is to illustrate one patient's experience following interdisciplinary referrals, which resulted in a step-by-step management of his OSA, as compared to a preferred multi-disciplinary approach from the outset.

CASE STUDY

A 56-year-old male presented to his general practitioner complaining of chronic loud snoring, daytime somnolence and social problems. He was treated conservatively with nasal sprays and a change in sleeping position. Neither resolved his problems. He was then referred to the chest physician, who diagnosed mild to moderate obstructive sleep apnoea using polysomnography. This tracing showed multiple episodes of oxygen desaturation throughout the night, the majority of which was spent below 95% PO2 and 30 secs desaturation below the more dangerous area of 90% PO2. Marked heart rate fluctuations associated with desaturation episodes were also indicated on the tracing. Apnea/hypopnea index (AHI) was 33.

The chest physician treated the patient using CPAP, delivering air under gentle continuous pressure overnight from a pump at the bedside over 5-6 hours. The patient reported an elimination of all symptoms overnight and this was confirmed by an oximetry trace, which showed complete abolition of all desaturation episodes. The patient, however, complained of a dry mouth and nose. In addition, he complained about the noise of the machine, discomfort from the mask and abrasions on the bridge of the nose. Arrangements were therefore made for a humidifier to be inserted into the CPAP circuit and he was provided with a more cushioned mask. A smaller, quieter machine was also supplied, but the patient became intolerant of the machine and there was a recurrence of symptoms.

He was subsequently referred to an ENT surgeon who provided an uvulopalatopharyngoplasty, which the patient felt made his condition worse. This was confirmed by polysomnography.

At this stage, 2 years after the initial diagnosis, a referral was made to the orthodontic department with a view to possible management with a mandibular advancement appliance.

Relevant findings from the history include hypertension controlled with amlodopine. He did not smoke nor drink excessively. The orthodontic examination noted a full set of teeth except for both lower first permanent molars. A severe Class II Division I, incisor relationship superimposed on a severe class II skeletal base, with an overjet of 10 mm, a deep and complete overbite to the palate. There was an in-standing upper right lateral incisor with evidence of wear on this tooth. Mesial drifting of both lower second molars and a backward displacement of the mandible on closing associated with the avoidance of the in-standing lateral incisor was noted.

The patient was seen on a combined orthodontic and oral and maxillofacial surgery clinic. Clinical findings and investigations suggested the following contributing aetiological factors; a narrowed upper arch, a retrognathic lower jaw, an in-standing upper right lateral incisor and mesially tilted lower second permanent molers with associated occlusal interferences. The retrognathic lower jaw is suggestive of a Type III OSA obstruction.

It was decided to commence management by orthodontic means in order to

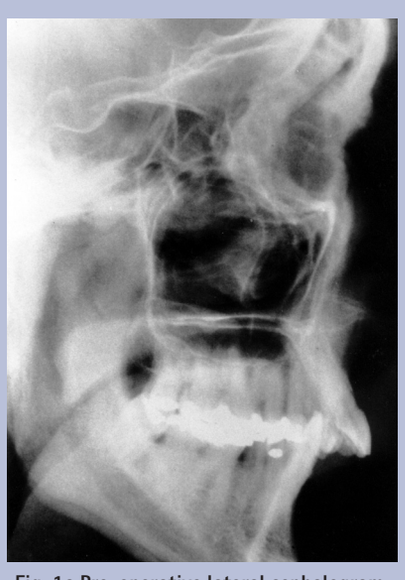

Fig. 1a Pre-operative lateral cephalogram showing evidence of increased overjet and Class II discrepency

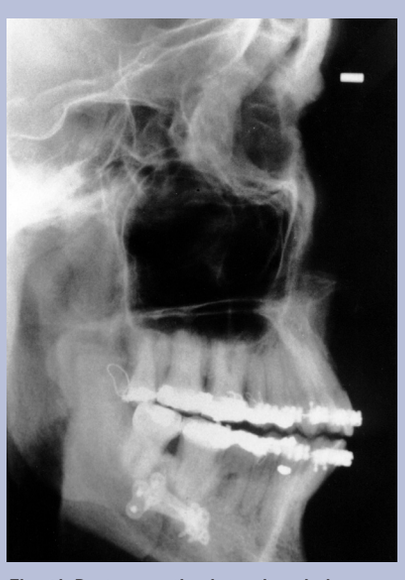

Fig. 1b Post-operative lateral cephalogram after surgical mandibular advancement

assess the patient's response to mandibular repositioning, before embarking on definitive orthognathic mandibular advancement treatment.

The orthodontic treatment aims were to facilitate an increased posterior airway space by the forward posturing of the mandible and tongue and to discourage the posterior mandibular displacement on jaw closure. This was to be achieved by maxillary arch expansion, proclination of the upper right lateral incisor and the wearing of a functional orthodontic appliance.

Almost immediately following the fitting of the orthodontic appliance, the patient reported a complete resolution of all symptoms. This was confirmed by a polysomnography trace, which showed a reduction from a previous record of 33 to 3.

Following the success with the orthodontic intervention, the multi-disciplinary team agreed with the patient to treat the underlying mandibular retrognathia by orthognathic means thereby increasing the posterior airway space.

The pre-surgical orthodontic aims were; to decompensate the teeth within arches, to correlate both arches and to use fixed appliances in preparation for a mandibular advancement procedure. In a Class II case, with mandibular retrognathia, pre-surgical decompensation, usually involves retroclination of lower incisors and proclination of upper incisors requiring Class III elastic traction. However with OSA patients these mechanics might be expected to aggravate the OSA. To overcome this problem, mild Class II elastic traction was applied at night to maintain the forward jaw position and Class III elastic traction was worn during the day. This probably delayed the orthodontic surgical preparation. Alternatively the patient could have maintained a patent airway at night by having CPAP, however the patient was reluctant to revert to this treatment modality.

A 9 mm mandibular advancement was performed by a bilateral saggital split ramus osteotomy using rigid bone plate fixation. The effect of this partial mandibular movement is to bring forward the genial tubercles, the anterior and greater part of the mandibular body and the lingula. This advances the origin of the genioglossus, the geniohyoid and mylohyoid muscles, the insertion of digastric and the attachment of the sphenomandibular ligament. The major mandibular agonists are not significantly affected but the structures in the floor of the mouth including the sublingual and submandibular salivary glands are affected. The hyoid bone moves forward to a lesser extent than the mandible and this and the advancement of many of the origins of the tongue musculature brings forward the tongue leading to an increase in oro-pharyngeal size.

Fig. 2 Continuous positive airway pressure (CPAP) machine

DISCUSSION

Polysomnography diagnosed this patient as having mild to moderate OSA.

Appropriate management for OSA depends on:

1. Cause of this condition
2. Severity of this condition

CPAP is the definitive treatment for OSA, however the functional orthodontic mandibular advancement appliance appeared to provide an immediate resolution of obstructive sleep apnoea symptoms. Probably as a result of forward movement of the mandible plus mylohyoid and genial muscles, to be followed by definitive orthognathic surgery.

This suggests that this treatment modality could be effective in most cases where the cause is a retrognathic mandible. A randomized control trial by Ferguson *et al*. concluded that MAA was an effective treatment in some patients with mild to moderate OSA and is associated with fewer side effects and greater patient satisfaction than CPAP.[8] A further randomized control trial by Johnston *et al*. concluded that MAA to be particularly successful in those with mild to moderate OSA, but less successful in patients with severe OSA.[9]

Mandibular advancement appliances may therefore be appropriate for those patients not severe enough for CPAP and in cases where patients refuse other procedures such as CPAP or surgery. They also provide an interim measure prior to other treatment procedures.[3]

Initial reports in the treatment of OSA with surgery to the soft palate were encouraging. However, as further trials of this procedure were reported, disappointing results began to emerge.[10] Surgery to the soft palate alone in the unselected patient provides little benefit in the management of mild OSA. Surgeons must use great care in discerning the level of obstruction in the patient with mild OSA to achieve excellent surgical outcomes.[11] A prospective randomised study, which compared mandibular advancement appliance with surgery to the soft palate, indicates a success rate of 95% with MAA and 70% with UPPP.[12]

Although the patient had previously undergone soft palate reduction, he still opted for orthognathic mandibular advancement, as he did not wish to be committed to protracted use of a mandibular advancement appliance. Surgical mandibular advancement was offered for the functional and aesthetic benefits of treating his skeletal discrepancy and his malocclusion. These included improvement of facial aesthetics and protection of upper incisors by placing them within the control of the lower lip.

Although this case was surgically treated successfully, surgical orthodontic treatment for OSA is not a mainstream treatment for this condition. It is only suitable for OSA patients with an underlying Class II discrepancy.

Risks associated with surgical mandibular advancement are significant, in particular with regard to sensory deficits resulting from inferior dental nerve trauma.

A study by Yu *et al*. concluded that increases in the saggital dimensions of oropharynx were unpredictable and tended to decrease with time. The best long-term results were found in patients with narrowing of the oropharynx preoperatively.[13]

There is a conflict of evidence with regard to mandibular setbacks provoking OSA. A 3-year post operative analyses by Achilleos *et al.*[14] reported a reduction of the saggital dimension of the oropharyngeal airway space; as a more short term study by Turnbull *et al.*[15] showed no significant change.

A systematic review indicates that there is an urgent need for high quality randomized controlled trials to be carried out in the field of surgery for OSA. More research should also be undertaken to identify and standardize techniques to determine the site of airways obstructions.[16]

Because of the complexity of investigating the cause of this condition and the multi-disciplinary management need, it would appear to be more beneficial for

patients to be managed by specialists in hospital services.

CONCLUSION

As a result of late orthodontic and orthognathic intervention, the patient achieved a delayed successful outcome. This case illustrates the importance and potential benefits of the specialties of chest medicine, ENT, orthodontics and oral and maxillofacial surgery working together as a multi-disciplinary team for the efficient diagnosis and treatment of these patients.

The outcome should identify which subgroups of patients with OSA benefit most from each type of treatment.

Although continuous airway pressure continues to remain the gold standard treatment, it can be concluded that mandibular advancement appliance and orthognathic surgery do play a role in the treatment of obstructive sleep apnoea in specific cases. However there is a lack of scientific evidence to support orthognathic surgery, in contrast to MAA for which there are now several well designed randomized controlled trials supporting their use.

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1. Davies R J, Stradling J R. The epidemiology of sleep apnoea. *Thorax* 1996; **51** Suppl 2: S65-S70.
2. Friedlander A H, Friedlander I K, Pogrel M A. Dentistry's role in the diagnosis and co-management of patients with sleep apnoea/hypopnoea syndrome. *Br Dent J* 2000; **189:** 76-80.
3. Miles P G, Nimkarn Y, De Leeuw B J. Dentistry's role in obstructive sleep apnoea. Review and case report. *Aust Dent J* 1996;**41:** 4.
4. Robertson C J. Obstructive sleep apnoea. Part 1: Diagnosis, aetiology and current treatment. *N Z Dent J* 1996; **410:** 110-113.
5. Peker Y *et al.* An independent association between obstructive sleep apnoea and coronary artery disease. *Eur Resp J* 1999; **14:** 179-184.
6. Amandeep J. The relationship between obstructive sleep apnoea and Dentistry: Aetiology and Diagnosis & 2. Management; *Dent Update* 1998; **25:** 380-383, 474-477.
7. Riley R W, Powell N B. Maxillofacial surgery and obstructive sleep apnoea syndrome. *Otolarygol Clin N Amer* 1990; **4:** 809-823.
8. Ferguson K A, Takashi O, Lowe A A, Keenan S P, Fleetham J A. A randomized crossover study of an oral appliance vs nasal-continuous positive airway pressure in the treatment of mild-moderate obstructive sleep apnea. *Chest* 1996; **109:** 1269-1275.
9. Johnston C.D, Gleadhill I.C, Cinnamond M.J, Gobbey J, Burden D.J. Mandibular advancement appliances and obstructive sleep apnoea: a randomized clinical trial. *Eur J Orthod* 2002; **24:** 251-262.
10. Coleman J. Oral & Maxillofacial surgery for the management of OSA syndrome. *Otolaryngol Clin North Am* 1999; 32: 235-241.
11. Senior B A, Rosenthal L, Lumley A, Gerhardstein R, Day R. Efficacy of uvulopalatopharyngoplasty in unselected patients with mild OSA. *Otolaryngol head Neck Surg* 2000; **123:** 179-182.
12. Wilhelmsson B, Tegelberg A, Walker-Engstorm ML, Ringquist M, Anderson L, Krekmanov L,Ringuist L. A prospective randomised study of dental appliance compared with uvulopalatopharyngoplasty in the treatment of OSA. *Acta Otolaryngol* 1999; **119:** 503-509.
13. Yu L F, Pogrel M A, Ajayi M. Pharyngeal Airway Changes Associated with Mandibular Advancement. *J Oral Maxillofac Surg* 1994; **52:** 40-43.
14. Achilleos S, Krogstad O, Lyberg T. Surgical mandibular setbacks and changes in uvuloglossopharyngeal morphology and head posture: a short- and long-term cephalometric study in males. *Eur Jr Orthod* 2000; **22:** 383-394.
15. Turnbull N R, Battangel J M. Effects of Orthognathic surgery on pharyngeal airway dimensions and quality of sleep. *J Ortho* 2000; **27:** 235-247.
16. Bridgman S A, DunnK M, Ducharme F. Surgery for obstructive sleep apnoea. (Cochrane Review). *The Cochrane Library* 2002; Issue 3.