Recent Developments in Oral Appliance Therapy of Sleep Disordered Breathing

WOLFGANG SCHMIDT-NOWARA, M.D.

ABSTRACT Oral appliances are increasingly gaining a place in the treatment of sleep disordered breathing caused by upper airway obstruction. This review of publications since 1995 documents substantial progress in the scientific basis for this therapy. Imaging by several techniques has shown that mandibular advancing oral appliances open the airway in awake and anaesthetized subjects, creating the presumption that this effect is maintained in sleep. Three controlled cross-over treatment trials have shown that patients consistently prefer oral appliance over continuous positive airway pressure therapy, especially when the treatment effect is strong. Appliance design and use indicates a preference for adjustable mandibular advancing appliances. Complications of therapy appear to be infrequent, but evidence for safety of long-term use is still limited. Oral appliance therapy can be an effective therapy for sleep disorders caused by upper airway obstruction. Considering the accumulated evidence, it is no longer tenable to label oral appliance therapy an ‘experimental’ procedure.

KEYWORDS: oral appliances, upper airway obstruction, sleep disordered breathing

Oral appliances (OA) are increasingly gaining a place in the treatment of sleep disordered breathing (SDB) caused by upper airway obstruction. Substantial progress since the original descriptions of this concept in Europe and the US has been documented in the growing literature. A review of the topic in 1995 represented a milestone in the development of this concept, not only because it summarized the conclusive evidence of efficacy of OA treatment for snoring and obstructive sleep apnea (OSA), but also because it was accompanied by practice parameters developed by the American Sleep Disorders Association (ASDA). The review considered 21 publications, comprising data from 320 patients, and demonstrated that in most, if not all cases, OA improved OSA, appeared to be safe, and were accepted for long-term use. The practice parameters stated that OA would be suitable first line therapy for simple snoring and mild OSA, and that they would be appropriate alternative therapy in more severe cases when continuous positive airway pressure (CPAP) was not accepted and surgery was not indicated.

Since 1995, a steady stream of reports has confirmed the 1995 conclusions and has extended the scientific basis for OA therapy. This article will summarize these newer developments and will update recommendations for OA therapy at the end of this millennium. The author relies on his acquaintance with the work in this area, supplemented by a search of Medline citations since 1994.

The term ‘oral appliance’ was introduced to encompass all appliances placed in the mouth so as to modify upper airway anatomy and function during sleep for the relief of upper airway obstruction. In 1995, the literature described primarily mandible advancing appliances, but also included a series of reports describing one tongue appliance. More recent reports are almost entirely restricted to mandible advancing appliances. Unless otherwise specified, in this article the term OA refers to an appliance with dental attachments and a mandible advancing design. Furthermore, the term ‘sleep disordered breathing’ in this discussion is restricted to the spectrum of respiratory abnormalities that appear during sleep because of upper airway obstruction.

NEW DEVELOPMENTS: MECHANISMS OF EFFECT

Oral appliances are presumed to open the airway by creating an anterior displacement of the upper airway structures and maintaining this during sleep. A number of studies confirm this concept in waking patients, but
observations during sleep are limited to documentation of improved breathing. A variety of imaging techniques in wake patients have demonstrated airway opening with OA use. Cephalographic studies, summarized in the 1995 review, demonstrated variously an increase in the retroglossal and retropalatal segments of the airway with OA. More recent cephalographic studies have produced the same findings. More importantly, MR imaging has produced a more complete, three-dimensional description of these changes. In a study performed by the author and published in an abstract, OA in 13 chronically treated patients increased the retroglossal and retropalatal segments of the airway by 25 and 27%, respectively. Endoscopic measurements have also demonstrated a change in upper airway size and shape with mandible advance during anesthesia or in awake patients. The observations of Isono provide the best evidence to date that OA do, in fact, open the airway during sleep, if anesthesia can be accepted as a surrogate of sleep.

A direct effect on airway size may not be the only benefit of mandibular advancement. Lowe has shown that OA can produce increased EMG activity in the genioglossus, presumable due to stretching of the displaced muscle, and this may translate in a reduced compliance or greater resistance to a collapse of the airway. Another mechanism may be the prevention of a change in mandibular posture during sleep. In normal subjects, progressively deeper sleep produces increasing downward rotation and retropositioning of the mandible, which in turn narrows the airway. In OSA patients, airway obstruction with apnea produces the same changes in mandibular posture. Since an OA prevents this retrusion of the mandible, this mechanism for sleep-induced airway narrowing is opposed. This observation might explain the clinical observation that OA can be effective with relatively little advance from the centric position.

OTHER DEVELOPMENTS

Other recent publications have significantly augmented the clinical research database since 1995. A major accomplishment is the comparison of OA to CPAP in randomized controlled trials. These studies will be reviewed later in this article. In addition, the concept of OSA severity as a predictor of treatment success is now well established. Marklund showed that treatment success declined as the apnea-hypopnea index (AHI) exceeded 30. Lowe also reported a better success when AHI was less than 30 compared to the more severe patients. A beneficial effect on sleepiness has been documented objectively with improvement of the maintenance of wakefulness test after OA. The ability to correct surgical uvulopalatopharyngoplasty (UPPP) treatment failures has been explicitly addressed in another study, and the answer is yes. The experience of OA in these patients is as good as in unselected patients: a predominance of successful responses and some patients with insufficient treatment effect.

It has been suggested for some time that OA may be effective in the upper airway resistance syndrome, because the effect on snoring appears to be robust. In an article published in this journal, Loube convincingly demonstrated the reversal of upper airway resistance syndrome (UARS), defined by esophageal manometry, with OA therapy. This single patient observation needs to be replicated. UARS is classically defined by esophageal manometry, but flow-time curve analysis or recording of snore arousals may be useful surrogates. These newer techniques provide opportunities to examine the effect of OA on mild upper airway obstruction, a group ideally suited for OA therapy.

COMPARISONS TO CPAP

Four new studies have addressed the relative effectiveness of OA compared to nasal CPAP for OSA. Effectiveness includes the concepts of treatment efficacy, and also acceptance and adherence to treatment. These were randomized controlled treatment trials; three studies used a cross-over design, a fourth used a parallel group design (Table 1). The reports of efficacy are remarkably consistent with each other as well as with earlier case series (Table 1). OA often, but not always, reduced AHI, whereas CPAP was almost uniformly successful in eliminating obstructed breathing events. However, because CPAP treatment acceptance and adherence were limited, the overall proportions of effectively treated patients were similar in each treatment arm. In the cross-over trials, patients could compare treatments and express a preference. In each of the three trials, the majority of patients preferred OA therapy (Table 1). This included patients in whom CPAP produced a lower AHI than OA. In two studies, sleep-disordered breathing severity was relatively mild, and it is quite possible that more severely affected patients would have expressed a different preference. One trial of an adjustable OA included a lengthy titration period to optimal therapy. In this study, dropout rates of OA users were significantly greater than CPAP, and this has been attributed to the delay in achieving effective treatment.

These studies illustrate the complexity of comparing two substantially different treatments. It is tempting to recommend OA for patients with mild to moderate disease, because patients are likely to prefer it to CPAP, even if it does not make breathing entirely normal. But should patients use a therapy with less than optimal efficacy? Arguably, any treatment that is used is better than a perfect but shunned therapy. It is not known whether mild residual abnormalities affect outcome, although the trend of recent research is to demonstrate beneficial