Review Article

Late upper airway constriction and its prevention by interceptive functional appliance therapy- A review

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ABSTRACT

Introduction: Sleep-disordered breathing (SDB) is one of the poorly diagnosed disorders which affects the quality of life of patients. The SDB is a spectrum of problems ranging from simple snoring to obstructive sleep apnoea (OSA) which involves complete collapse of the pharyngeal airway passage during sleep resulting in airflow cessation and consequent arousals. A noteworthy etiological factor of OSA is mandibular retrognathism. Imaging studies on craniofacial structures in OSA patients have shown decreased oropharyngeal and hypopharyngeal depth, elongated soft palate, altered tongue posture, and increased hyoid bone-to-mandible distance. The management modalities of OSA are positive airway pressure (PAP) and oral appliance therapy. The modus operandi of mandibular advancement devices (MADs) is the same as of myofunctional therapy instituted by orthodontists for the treatment of skeletal Class II malocclusions with mandibular retrusion in growing patients. By virtue of the increase in the mandibular length and advancement in mandibular position, pharyngeal airway space is enlarged along with favorable changes in the soft palate, tongue posture, and hyoid bone position. The focus of the present review is to consolidate the available literature on the long-term effects of functional appliance treatment. It is hypothesized that interceptive functional appliance therapy in the growing stage could be a viable strategy for the prevention of OSA development during adulthood. The review also highlights the importance of the need for more studies emphasizing the long-term impact of functional treatment to establish the role of the same in reducing the OSA treatment burden during adulthood.

KEYWORDS: Airway constriction, Functional appliance, Pharyngeal airway, OSA, Upper airway

INTRODUCTION

Sleep-disordered breathing (SDB) is a complex phenomenon that is relatively poorly understood and underdiagnosed.[1] The SDB is an umbrella term that consists of obstructive sleep apnoea (OSA), upper airway resistance syndrome (UARS), central sleep apnoea syndrome, and sleep hypoventilation syndrome.[2] The OSA is a chronic disease characterized by mechanical constriction and collapse of the upper airway resulting in episodes of apnoea and arousals from sleep.[2] In OSA, the ventilatory effort is present but the airflow remains absent or almost absent. It is a troublesome illness that causes impairment of quality of life, exacerbation of cardiovascular and respiratory disorders, daytime somnolence, and road traffic accidents.[3] Epidemiological studies in the Western countries show that about 8% of middle-aged men and 2% of women are affected whereas studies from various parts of India report a prevalence of 2.4% - 4.96% in men and 1-2% in women.[3,4] However, pediatric OSA has a prevalence of 1-5%.[3-5] The clinical course of OSA has been described to comprise four chronological stages namely, stage of susceptibility, stage of pre-symptomatic disease, which may be characterized by snoring, stage of clinical disease, marked by apnoea, and lastly the stage of recovery, disability or death.[6] Although various screening tools like Friedmann's method based on the tonsil size, Kushida Index which factors in obesity along with transverse and sagittal dentition measurements, Mallampati scoring, questionnaires like the Epworth Sleepiness Scale, Berlin questionnaire are available, however polysomnography (PSG) remains the gold standard for the diagnosis of OSA.[2,7] The chronicity of the disease combined with

an asymptomatic phase that endanger cardiovascular and respiratory health necessitates a pre-emptive approach for its management. Keeping this in view, the role of an orthodontist becomes crucial in the screening of patients – either at risk of developing OSA or undiagnosed cases of established disease, other than in its management.

Etiopathology of SDB

The etiopathology of OSA is proposed to be multi-factorial.[6] The elements predisposing an individual to the clinical disease include genetic factors of obesity, ventilatory control, craniofacial abnormalities, epigenetic factors like environment, alcohol consumption, smoking, sedentariness, co-morbidities like congestive heart failure, hypothyroidism, various age-related factors, and lastly the hormonal factors like menopause.[6]

The principal cause of the abnormality is the collapse of the upper airway girth anywhere along the nasopharynx, oropharynx (which includes the velopharynx), and hypopharynx. The most frequent loci of obstructions are the velopharynx, at the level of the soft palate, and the oropharynx, at the base of the tongue.[2,8] During sleep, the tonicity of upper airway muscles and protective pharyngeal reflexes get diminished along with an increase in chemoreceptor set-point. The compensatory dilator action of genioglossus, geniohyoid, and pharyngeal muscles is lost leading to airway collapse thereby causing the patient to snore or wake up following a hypopnoea-apnoea event.[9] Adenotonsillar hypertrophy is considered a significant cause of OSA in children.[4] The American Academy of Sleep Medicine recommended at least five apnoea-hypopnoea episodes in an hour for the diagnosis of OSA in children.[10]

Role of mandible in SDB

It is hypothesized that craniofacial abnormalities place the pharynx 'at risk' for airway constriction irrespective of other soft tissue influences.[8] Patients with OSA have been shown to have an elongated soft palate, and inferiorly positioned hyoid bone, enlarged tongue, decreased or narrow pharyngeal airway space, steep mandibular plane, and retrognathic maxilla and mandible.[8] A recent systematic review by Neelapu et al. also noted that key cephalometric features noted in established OSA patients were reduced pharyngeal airway space, lower hyoid bone, and increased anterior facial height.[11] A significant weighted mean difference, albeit with heterogeneity were seen in OSA cases versus controls with mandibular position with respect to the cranial base and its length, maxillary length, tongue area, and soft palate area, and upper airway length.[11] These findings were in accordance with the meta-analysis done by Miles et al.[12]

The backward position of the tongue in subjects with retrognathic mandible pushes the soft palate posteriorly and decreases the dimension of the upper airway.[13,14] It has been found that there is a direct relationship of apnea with the distance between the hyoid bone and mandibular plane.[8] Patients with OSA demonstrated a significantly higher distance than healthy persons and as the perpendicular distance between the hyoid bone and mandibular plane increases, the probability of having apnoea increases.[8] This was reinforced in a meta-analysis depicting an inferior location of the hyoid bone in relation to the mandibular plane and sella.[11]

Further, a small or retrognathic mandible during childhood is considered as a risk factor of OSA in adulthood.[15] Literature suggests that constricted airway contributes to OSA in about 2% of children and has long-term adverse effects.[16] A study by Nelson and colleagues demonstrated that snorers have a greater hyoid to mandibular plane distance during the prepubertal and pubertal periods.[17] In other words, smaller and retruded mandible if corrected during childhood may help prevent OSA later in life. On this basis, several management modalities are targeted at altering the mandibular length and its position which may be surgical and non-surgical. The treatment of choice is purely governed by patient age and treatment acceptance.

Management of SDB

The desired outcomes of OSA treatment are to resolve patient signs and symptoms, reduce apnoea-hypopnea events to below 5 per hour and normalize oxyhemoglobin saturation in the blood. The approach is multi-targeted. It begins with behavior modification i.e. weight loss till BMI is below 25kg/m^2, exercise, avoidance of alcohol, sedatives before sleep, and sleep position.[2] Depending on the severity of OSA, positive airway pressure (PAP) therapy or oral appliance (OA) therapy is instituted.[2] When conservative and non-invasive measures are not deemed suitable for the patient, surgical interventions like uvulopharyngoplasty (mild to moderate cases), tracheostomy (severe cases), mandibular advancement, and/or elongation surgeries, and distraction osteogenesis are indicated. The American Academy of Sleep Medicine recommends PAP as the treatment of

choice for mild, moderate, and severe OSA.[10] However, based on patients' anatomy, their preferences, and other risk factors, oral appliance therapy and surgery may alleviate the problem.[2] Among various oral appliances, mandibular advancement splints are most commonly used for the treatment of OSA. Newer therapies like muscle exercises, hypoglossal muscle stimulation, and expiratory nasal valves have also emerged for OSA management.[2]

Although the weight of literature tips the balance in favor of CPAP therapy over mandibular advancement appliances in terms of efficacy; Sarell et al.[18] and Guralnick et al.[19] have demonstrated poorer patient adherence to the usage duration of continuous PAP. Reckoning with patients' poor compliance, the cost-effectiveness of PAP drops down to a level lower than that of mandibular advancement devices.[20] Mandibular advancement appliances position the lower jaw anteriorly, thereby improving upper respiratory tract obstruction.[21] Similar mandibular advancement appliances are also widely used in growing children for the interception of their Class II malocclusions associated with mandibular deficiency.[22] Sagittal mandibular development is also seen to have a significant influence on the inclination of the soft palate.[14] We hypothesize that if the effects of interceptive functional orthopedic treatment are retained adequately in the long run, we can utilize it for the prevention of OSA in adulthood. The rationale behind preventive mandibular advancement is three-pronged,

1. Intermittent hypoxia and post-apneic reoxygenation during apnoeic episodes in untreated OSA cause vascular endothelial damage, which can progress to atherosclerosis and, consequently, coronary artery disease and ischemic cardiomyopathy. Even incident diabetes, hypertension, and increased mortality are associated.[6] To worsen things, it has a subclinical stage. Thus, prevention would control future systemic health burdens.

2. When it is diagnosed later in adulthood, the only major options remain either surgical or device-supported. However, a lot of acceptance issues with both the approaches have been reported, fear of surgery, maintenance of device, and cost burden being major deterrents for many patients.[6]

3. Oral appliance therapy during adulthood has clear dental and skeletal side effects with long-term use.[23]

Therefore, if the identification of high-risk individuals is made early, it will serve a dual purpose i.e. enlargement of upper airway space along with addressing the malocclusion, facial dysmorphism, trauma to protruding incisors, and so on.

Role of functional appliances in SDB

Functional appliances are widely used for the interception of Class II malocclusions associated with mandibular deficiency. Multiple types and designs like removable or fixed, conventional or bone-anchored, have been employed to correct the mandibular deficiency.[24-27] These appliances perform by enhancing the proprioceptive sensory feedback of stomatognathic muscles that control the mandibular position and transmit the generated forces to the dentition and basal bone and thus altering the position and growth of the mandible.[28,29] Rapid palatal expansion has also been shown to be effective for slight to moderate OSA, as a co-adjuvant therapy to adenotonsillectomy in severe cases with maxillary constriction.[4,30] The effects of functional appliances on craniofacial structures have been assessed using lateral cephalometry and CBCT.[22,24,31] Ghodke et al. evaluated the effect of twin-block therapy on Class II malocclusion patients with mandibular deficiency and found significant improvements in the depth of the oropharynx and hypopharynx and inclination of the soft palate following correction of mandibular retrusion.[13] When the mandible was displaced anteriorly by a twin-block appliance, it influenced the position of the hyoid bone and consequently the tongue position thus improving the upper airway morphology.[13] Schutz et al. found that after Class II correction, the anterior displacement of the mandible and the hyoid bone caused anterior traction of the tongue, which increased the posterior airway space by 3.2 mm and reduced airway resistance.[32] Similarly, Horihota et al.[33] and Ullusoy et al.[34] noted a beneficial impact on the airway by activator treatment. In contrast, Lin et al. reported no significant changes in the anteroposterior dimensions of the pharyngeal airway passage following the advancement of the mandible by bionators.[35] Nevertheless, in a more recent study, twin-block appliance therapy resulted in the advancement of the mandible with a lowered posturing of the tongue.[36] Jena et al. also reported enlargement of pharyngeal airway space following twin-block and Mandibular Protraction Appliance-IV (MPA-IV) treatment.[37] Pavoni et al. compared the effects of a modified monobloc appliance in Class II patients with a control Class II group.[38] Besides reporting a widened airway space, they showed more anterior positioning of the tongue, a decrease in tongue height, and an increase in mandibular length (condylion to menton) by 4mm.[38] The latest systematic review on the functional

appliance and upper airway revealed that the correction of the mandibular deficiency by functional appliance therapy did not affect the nasopharyngeal airway dimensions; however, they had a significant effect on the improvement of the oropharyngeal airway.[39]

There is a significant positive correlation between oropharyngeal airway volume and mandibular length.[23,40] Increase in mandibular length by functional appliances has been demonstrated by Baccetti et al.[41] and Marsico et al.[42] A study evaluating the effects of skeletally anchored Forsus fatigue-resistant device (FFRD) and Herbst appliance reported a significant increase in the lower pharyngeal dimension with the former.[43] Celikoglu reported significantly increased oropharyngeal area measurements with both Herbst appliance and skeletally anchored Forsus FRD.[43] Similar observations including an increase in mandibular length, vertical movement of the mandible, and increase in the oropharyngeal area were noted by Ince-Bingol et al. using a mini-plate anchored Forsus device.[27] They highlighted the possible role of such therapies in addressing respiratory problems in skeletal Class II patients.[27] Moreover, conventional fixed functional appliances also report a favorable change in tongue posture and forward positioning of hyoid bone amounting to enlarged oropharyngeal airway size. Larger oropharyngeal volume was also reported to occur using mandibular anterior repositioning appliance (MARA) appliance in another study.[44] However, literature reported that the correction of mandibular deficiency with fixed functional appliances is mostly based on the mesial movement of mandibular dentition, which might influence changes in tongue posture.[26] Kannan et al. in a recent systematic review concluded that notable increases in airway space are noted with activator, bionator, twin block, and Frankel II but not with MPA-IV, Herbst, and functional mandibular advancer (FMA) appliance.[24] Thus interception of the mandibular deficiency by various removable functional appliances is more effective in the improvement of upper airway dimensions.

Long term stability of airway changes

Numerous endeavors to analyze the long-term results of functional therapy have been made with different kinds of appliances viz. bionator,[45] activator with headgear,[46] Faramund[25] functional appliances, etc. The details of each study that evaluated the long-term stability of functional appliance therapy on the upper airway are summarized in table-1. The stability of the effects of fixed functional appliance has also been studied by Drosen et al.[47] The findings of Yassaei et al.[48] indicate that these achieved changes of enlarged airway space, and tongue and hyoid placement changes seemed to be maintained in long-term, up to 4-years on average. The subjects studied till the time of their growth completion following Herbst appliance treatment showed a significant increase in the parameters representing pharyngeal airway width compared to the control groups. This was attributed by the authors to the possible long-lasting alteration of the post-treatment mandibular growth pattern and consequent increase in lower posterior facial height.[47] Hanggi et al.[46] carried out a follow-up of up to 12-years of patients treated by activator-headgear combination therapy. They reported that the increase in parameters like pharyngeal area, pharyngeal length, and the smallest distance between the tongue and the posterior pharyngeal wall remained stable till up to 22-years of age on average.[47] Furthermore, the study by Han et al.[45] using bionator therapy indicated that growth in pharyngeal airway dimensions in growing adolescents was maintained even after growth completion.

Can OSA be prevented?

Keeping in view the plausible long-term effects of functional appliances,[45,46,48] it may be postulated that widening of a narrow upper airway passage by functional appliances in children could eliminate the risk of developing OSA in adulthood. Thus early intervention of mandibular deficiency by various removable functional appliances provides an opportunity to enlarge the upper airway dimensions and decrease the potential risk of obstructive sleep-disordered breathing problems for growing individuals in the future.

Clinical recommendations

Screening assessment for OSA symptoms in every orthodontic patient may help to identify the high-risk patients. Any such case should be referred to the sleep physician and polysomnography should be done for a final diagnosis. Orthodontists can team up with sleep physicians and assist the sleep physician to manage the patient better.

Table-1: The details of various studies evaluating the long term stability of upper airway changes following functional appliance treatment.

Functional appliance type	Author, year	Study Design			Appliance used	Airway assessment tool	Conclusion
		Sample	Study design	Timing of long-term follow-up			
Removable	Hanggi et al.,[46] 2008	Study group- 32 patients Control group- 32 patients -	Retrospective	Patient aged 18years or above at follow-up.	Activator-headgear	Lateral cephalometry	Activator-headgear therapy has the potential to increase pharyngeal airway dimensions, such as the smallest distance between the tongue and the posterior pharyngeal wall or the pharyngeal area. Importantly, this increase seems to be maintained long term, up to 22 years on average in the present study.
	Yassaei et al.,[48] 2012	-	-	-	Faramund appliance	Lateral cephalometry	Treatment with functional appliance has the potential to increase pharyngeal airway dimensions and changes in tongue and hyoid position and these achieved changes seemed to be maintained in long-term, up to 4 years on average.
	Han et al.,[45] 2014	Study group (Class II)- 24 Control group (Class I)- 24	Retrospective	Growth completion assessed on hand-wrist radiographs.	Bionator	Lateral cephalometry	The increase in pharyngeal airway dimensions in growing Class II adolescents after functional therapy is maintained until the completion of growth.
Fixed	Drosen et al.,[47] 2018	Study group (Class II) -13 patients Control group – 13 Class I and 13 Class II patients	Retrospective	Patient aged 18years or above at follow-up.	Herbst appliance	Lateral cephalometry	In the long term, Herbst treatment resulted in a significant post-treatment increase of PA width, possibly due to an increased lower posterior facial height development compared to untreated individuals.

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