Diagnosis and Management of Obstructive Sleep Apnoea – A Literature Review
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Abstract
Timely diagnosis of obstructive sleep apnoea and its management is an important factor in terms of orthodontic treatment outcomes. Symptoms of obstructive sleep apnoea being suggestive of the condition need to be given consideration along with predisposing factors that lead to obstructive sleep apnoea. Attention must be given to those patients identified with the predisposing factors who are likely to develop obstructive sleep apnoea. A variety of treatment options are available this day, beginning with lifestyle modifications, appliances, pharmacological management and ultimately surgical management depending on the severity of obstructive sleep apnoea which is identified through grading.

Key words: Obstructive sleep apnoea (OSA), obstructive sleep apnoea management.

INTRODUCTION
Obstructive sleep apnoea is an increasingly common disorder characterized by frequent episodes of airway obstruction associated with a reduced calibre of the upper airway and is vulnerable to further narrowing and collapse. Today, the field of orthodontia plays an important role in the diagnosis and treatment of sleep-disordered breathing syndromes and oral appliances have been used to treat mild to moderate obstructive sleep apnoea.

Definition of sleep apnoea
Apnoea is defined as cessation of airflow and hypopnoea as a reduction in airflow of > 50% for more than 10 sec [1]. Apnoeas and hypopnoeas may be central in origin or obstructive. Obstructive sleep apnoea (OSA) is said to be present when these episodes occur > 5 times per hour of sleep [1]. Obstructive sleep apnoea syndrome (OSAS) is the term applied when OSA is accompanied by day-time symptoms such as excessive somnolence [1].

Prevalence
Obstructive sleep apnoea affects approximately 1–4% of the middle-aged population, with a male-to-female ratio of 2:1[1]. An estimated 24 percent of men and 9 percent of women have obstructive sleep apnoea, whereas 4 percent of men and 2 percent of women have obstructive sleep apnoea with associated daytime sleepiness [2, 3]. The first detailed clinical description of OSA was made in 1966 but it was described incidentally many years prior to this, most famously in Dickens’ portrayal of Joe the fat boy in The Posthumous Papers of the Pickwick Club [1]. Predisposing factors for obstructive sleep apnoea [4].
Predisposing factors for obstructive sleep apnea [4]

<table>
<thead>
<tr>
<th>Factor</th>
<th>Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Obesity</td>
<td>Pharyngeal obstruction</td>
</tr>
<tr>
<td>Race</td>
<td>Craniofacial abnormality</td>
</tr>
<tr>
<td>Genetics</td>
<td>Laryngeal obstruction</td>
</tr>
<tr>
<td>Age</td>
<td>Endocrine</td>
</tr>
<tr>
<td>Male gender</td>
<td>Neuromuscular disorders</td>
</tr>
<tr>
<td>Alcohol</td>
<td>Connective disorders</td>
</tr>
<tr>
<td>Analgesics</td>
<td>Storage disease</td>
</tr>
<tr>
<td>Sedatives</td>
<td>Chronic renal disease</td>
</tr>
<tr>
<td>Anaesthetics</td>
<td>Nasal obstruction</td>
</tr>
<tr>
<td>Smoking</td>
<td></td>
</tr>
</tbody>
</table>

Symptoms of obstructive sleep apnoea [1]

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Loud snoring</td>
<td>Oesophageal reflux</td>
</tr>
<tr>
<td>Enuresis</td>
<td>Witnessed apnoeas</td>
</tr>
<tr>
<td>Day-time somnolence</td>
<td>Choking or dyspnoeic</td>
</tr>
<tr>
<td>Nocturnal sweating</td>
<td>Personality change</td>
</tr>
<tr>
<td>Nocturnal cough</td>
<td>Nocturia</td>
</tr>
<tr>
<td>Restless sleep</td>
<td>Unrefreshing sleep</td>
</tr>
<tr>
<td>Morning headaches</td>
<td>Insomnia</td>
</tr>
</tbody>
</table>

For majority of the symptoms, sleep fragmentation is the cause. The consequences of OSA are primarily a result of the fragmentation of sleep caused by multiple arousals, and the resultant hypoxaemia and hypertension that accompanies each arousal.

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Consequences of obstructive sleep apnoea [1]

<table>
<thead>
<tr>
<th>Category</th>
<th>Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neuropsychological</td>
<td>Sleepiness, impaired memory/cognition, increased accident risk, anxiety, depression, intracranial hypertension, chronic headache</td>
</tr>
<tr>
<td>Cardiovascular</td>
<td>Hypertension, ischaemic heart disease, right heart failure, cerebrovascular disease</td>
</tr>
<tr>
<td>Respiratory</td>
<td>Hypoxaemia, hypercapnia, pulmonary hypertension</td>
</tr>
<tr>
<td>Endocrine</td>
<td>Reduced growth hormone/testosterone levels, diabetic instability</td>
</tr>
<tr>
<td>GIT</td>
<td>Gastro-oesophageal reflux</td>
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</tbody>
</table>

Pathophysiology

The pathophysiology of obstructive sleep apnoea is related closely to the anatomy of the upper airway. The oropharynx lacks the cartilaginous skeletal support of the lower airway, likely because it must be malleable enough to perform diverse functions of speech, mastication, swallowing, and breathing [5]. Although the hyoid bone sits at the base of the tongue and could potentially provide rigid support, this floating bone is held in place by ligamentous and muscular attachments [6]. Although this enables dynamic mobility of the tongue base and larynx, it leaves the upper airway unsupported [7]. As a result, the oropharynx is structurally predisposed to collapse during sleep and has been cited as the most common location of obstruction in obstructive sleep apnoea, although obstruction in the hypopharynx is also quite common [8-10].

Evaluation and diagnosis

A routine health checkup must include questions on snoring and daytime somnolence and examined for obesity, hypertension, and retrognathia. Patients considered to be at risk need to be further assessed. Questionnaires that are used to identify obstructive sleep apnea are Berlin, STOP-Bang and Epworth questionnaires. Polysomnography is the standard for testing recommended by the American Academy of Sleep Medicine. Atypical laboratory-based polysomnogram includes measures of [3]: (1) airflow through the nose using a nasal cannula connected to a pressure transducer or through the nose and mouth using a thermal sensor;(2) respiratory effort using thoracic and abdominal inductance bands;(3) oxygen haemoglobin saturation by finger pulse oximetry;(4) snoring using a microphone affixed over the trachea or by filtering out low frequency signals from the nasal cannula-pressure transducer system;(5) sleep stage and arousal using electroencephalogram,electrooculogram, and chin electromyogram; (6) electrocardiogram findings;(7) body position;(8) leg movement. Home sleep apnea testing is increasingly used to diagnose OSA, and consists of measures of airflow,
respiratory effort, and oxygen saturation, but not measures of sleep or leg movements. It is a type of out of centre sleeping test (OCST). The sensors are self-applied by the patient at home following instruction from a technologist or via an instructional video. Apnea-hypopnea index is an objective parameter that serves as the standard for classifying obstructive sleep apnea severity.

In recent years, drug-induced sleep endoscopy (DISE) has become a widely used diagnostic tool and is routinely administered by multiple hospitals for the diagnosis of OSA. DISE allows for an assessment of the degree and localization of airway obstruction during a defined period of sedation time. Particularly owing to the high degree of visual feedback, performing DISE is considered to improve the selection of the required operative procedure [11].

According to the International Classification of Sleep Disorders [12], OSA can be diagnosed by either of 2 sets of criteria. The first set of diagnostic criteria for OSA includes the presence of at least 1 of the following: (1) the patient has sleepiness, nonrestorative sleep, fatigue, or insomnia symptoms, (2) the patient wakes with breath holding, gasping, or choking, (3) a bed partner or other observer reports habitual snoring, breathing interruptions, or both during the patient’s sleep, and (4) the patient has been diagnosed with hypertension, a mood disorder, cognitive dysfunction, coronary artery disease, stroke, congestive heart failure, atrial fibrillation, or type 2 diabetes mellitus; and polysomnography or OCST shows at least 5 predominantly obstructive events (obstructive or mixed apneas, hypopneas, or respiratory effort–related arousals (RERAs) per hour of sleep during a PSG or per hour of monitoring on OCST.

Grading of OSAS
Grade 0 (AHI 1–10) is a typical heavy snorer with a few episodes of arousal. Grade 1 is moderately severe OSA (AHI 10–30). Grade 2 (AHI 30–80) is severe OSA with recurrent episodes of apnoea throughout sleep but with SpO2 returning to baseline with each arousal. Grade 3 is characterised by sustained hypoxaemia, worse during rapid eye movement sleep, suggesting a degree of hypoventilation [1].

Treatment Options
Lifestyle modifications
Patient must be advised on moderating caffeine and alcohol intake. Obese patients can be advised on the importance of weight loss and following a healthy diet. It may be considered as the sole initial treatment in asymptomatic or minimally symptomatic patients. The effects of sleep position should also be discussed including how sleeping in a side position may provide some benefit [13, 14]. Lifestyle interventions, bariatric surgery, and weight loss medication are each associated with improved OSA severity [15-17].

Continuous positive airway pressure
The American Academy of Sleep Medicine describes continuous positive airway pressure as the standard for treatment of moderate to severe sleep apnea and an option for the management of mild obstructive sleep apnea [10]. The device generates pressure which is delivered via a mask worn over the nose or both nose and mouth. The pressure may be continuous or bilevel and may be automatic titrating or delivered at a preset pressure. Continuous positive airway pressure improves apnea-hypopnea index and daytime somnolence among other symptoms [18]. The advantage of this method is that it is efficacious in most patients, regardless of disease severity, level of airway collapse, or body weight; improves sleepiness, quality of life, and blood pressure. The main limitation of continuous positive airway pressure is failure of patients to use it regularly [19].

Bi-level positive pressure (BPAP)
Bi-level positive airway pressure (BPAP) is an alternative treatment for patients with CPAP intolerance [20]. While CPAP provides a fixed pressure throughout the breathing cycle, BPAP offers a higher level of inspiratory positive airway pressure (IPAP) and a lower level of expiratory positive airway pressure (EPAP) during respiration [21, 22].

Autotitrating positive airway pressure (APAP)
Autotitrating positive airway pressure (APAP) is another alternative treatment for patients with CPAP intolerance [20]. It is characterized by the ability to automatically adjust the pressure between 5 and 20 mmHg. As APAP can increase the pressure as needed while patients are in fast eye movement period (REM) or in supine sleep, and maintain a lower pressure during other sleep conditions, it provides more comfort and better compliance compared with the traditional fixed pressure CPAP [23].

Oral Appliances
There are numerous differences in the design features of commercially available oral appliances. Differences predominantly relate to the degree of customization to the patient’s dentition and one-piece (monobloc) designs (no mouth opening) versus two-piece design (separate upper and lower plates).

Mandibular repositioning devices
Mandibular advancement device (MAD) can be used as a first-line treatment for mild to moderate OSAS patients [20]. Fabricated to fit the upper and lower teeth, these devices provide adjustable forward advancement of the mandible during sleep. These devices comprise moulded gum shields which cohere to
the teeth to protrude the mandible by approximately 75% of maximum, enlarging the retroglossal space. Potential complications include temperomandibular joint dysfunction and dental damage. Also it has lower efficacy than positive airway pressure in most patients, especially in those with severe OSA or class 2 or 3 obesity [24].

Tongue retaining devices

It is a bubble shaped device made of soft polyvinyl. The patient’s teeth rest in custom fitted grooves which are extended to form a bubble shape that sticks out from between the lips. The patient positions his teeth in the grooves, sticks his tongue forward into the bubble until suction grabs and holds the tongue in place [25]. It is most useful in patients with very large tongues, poor dental health, no teeth, chronic joint pain or if their sleep apnea is worse when lying on their backs than when they lie on their sides at night. The device cannot be used in people who are tongue-tied, so overweight that they are more than 50% above their ideal body weight, grind their teeth at night, or have chronically stuffy noses [25].

Appliance designs

1). Thornton adjustable positioned

The Thornton adjustable positioner (TAP) allows for progressive ¼ mm advancements of the jaw via an anterior screw mechanism at the labial aspect of the upper splint. This appliance has a separate section for the mandible and maxilla. Each portion of the appliance is placed in the mouth separately and then the patient sticks out his/her chin until the hook and bar hardware can be connected. This appliance is easily retained by tooth grinders, even those who have worn away much of their tooth structure [25].

2). The elastic mandibular advancement

This appliance design uses specially designed, patented elastic bands to reach the desired positions with considerable freedom of movement. The elastic mandibular advancement (EMA) is the thinnest and least bulky of all the appliances. It is similar to clear acrylic orthodontic retainers, and moves the jaw forward in fairly significant steps, and can be difficult to tolerate [25].

3). Oral pressure appliance

It is a combination appliance which combines a nonadjustable mandibular repositioning device with continuous positive airway pressure (nasal CPAP). Instead of using nasal CPAP, which delivers air pressure through a mask over the nose or the nose and mouth, the air pressure is delivered through a small conduit that fits across the roof of the patients’ mouth [25].

Pharmacological management

Steroids and leukotriene receptor antagonists

Nasal steroids have been used as a non-surgical alternative to pediatric OSAS, especially for patients with mild OSAS [23]. The most common cause of OSAS in children is adenoid hypertrophy. Resection of adenoid and tonsils which was considered the first choice of treatment in the past is no longer advocated and instead anti-inflammatory therapy is the preferred treatment. Chan and colleagues conducted a randomized double-blind controlled trial of mild OSAS children and found out that AHI of the nasal mometasone furoate group decreased from 2.7 ± 0.2 to 1.7 ± 0.3, while AHI of the placebo group increased from 2.5 ± 0.2 to 2.9 ± 0.6 (P = 0.039) [26]. The American Academy of Sleep Medicine noted that topical nasal steroids may improve AHI in patients with OSAS and concomitant rhinitis and may, therefore, be an adjuvant to the primary treatment of OSAS [27]. Leukotriene receptor antagonists, as a proved safe, well-tolerated anti-inflammatory drug, can be an effective preventive treatment of inflammatory diseases such as asthma or allergic rhinitis. Also, it may be used as an alternative to adenoidectomy for OSAS children [28]. Gozal and colleagues showed that AHI of children in montelukast group reduced from 9.2 ± 4.1/h to 4.2 ± 2.8/h (P < 0.0001) while AHI in the placebo group did not change [29].

Transcutaneous electrical stimulation (TES)

TES is mainly used to treat CPAP intolerant patients. Studies have shown that pharyngeal collapse is associated with the loss of genioglossus tone at the beginning of sleep, which becomes the theoretical basis for nerve stimulation to recover airway patency, increase sleep breathing and eventually treat OSAS [30, 31]. Female patients with mild to moderate OSAS are more likely to be responders. Lequeux and colleagues [32] combined transcutaneous neuromuscular stimulation with muscle training, and the mean apnea hypopnea index (AHI) decreased from 32.9 to 20.6 (P = 0.017) after 10 weeks, with 7 patients < 10.

Surgical Procedures

1) Uvulopalatopharyngoplasty (UPPP) and related soft tissue procedures: Involves resection of the uvula and a portion of the soft palate; other soft tissue procedures focus on reducing volume of the lateral pharyngeal walls or base of tongue to increase pharyngeal volume. The advantages include it is extensively studied; results in improvement in OSA severity in many patients; adherence to therapy is ensured. It is lower in efficacy than PAP in most patients and effectively manages airway collapse only at the level of the velopharynx. Postoperative pain is common with small risk of velopharyngeal insufficiency and relapse can occur with weight gain [24].
2) Maxillomandibular advancement: This includes LeFort I maxillary and bilateral mandibular osteotomies with forward fixation of the facial skeleton. It is highly efficacious regardless of disease severity, level of airway collapse, or body weight; adherence to therapy is ensured. The drawback of this procedure is that it is complex and requires a recovery time of 2 – 10 weeks. Potential complications include malocclusion, poor cosmetic result, and facial numbness or paresthesia.

3) Tracheostomy: Although curative in most patients with OSA, regardless of disease severity, level of airway collapse, or body weight, and adherence to therapy is ensured, it is rarely used. Unacceptable cosmetic result, effect on speech, need for long-term tracheostomy care, make this a less approach procedure to relieve OSA.

4) Radio frequency or somnoplasty: Radiofrequency tissue volume reduction (RFTVR) is a surgical method which uses radiofrequency heating to create targeted coagulative submucosal lesions resulting in shrinkage of the inner tissues leaving the outer tissues intact [25]. The result is relief of nasal obstruction when used to shrink the nasal turbinates and diminished snoring when used to reduce the soft palate or elimination of OSA when used for tongue reduction [33].

5) Genioglossus tongue advancement: The procedure produces a larger space between the back of the tongue and the throat thereby creating a widerAirway [34]. Complications resulting from this procedure are very uncommon. This operation is often performed in tandem with at least one other procedure such as the Uvulopalatopharyngoplasty (UPPP) or hyoid suspension [25].

6) Hyoid suspension: This is a procedure which was developed specifically for the treatment of OSA. The operation advances the tongue base and epiglottis forward, thereby, opening the breathing passage at this level [34, 35]. If the hyoid bone is pulled forward in front of the voice box, it can open the airway space behind the tongue.

**CONCLUSION**

Current literature indicates that myofunctional therapy can be successfully used to decrease AHI in both adults and in children. Although obstructive sleep apnoea should be undertaken by a qualified dentist only after a referral from a qualified physician trained in sleep medicine that has performed a face-to-face evaluation of the patient, a variety of treatment options are available based on the tolerance of the patient and severity of the OSA. Although mandible advancing oral appliance and CPAP have been considered as alternative treatment pathways, there is scope for a patient to alternate between them as needed in situations such as travel when CPAP may be inconvenient. Additionally there are some recent lines of evidence suggesting combining the two treatment modalities simultaneously may be of additional benefit.

**REFERENCES**


