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Long Term Follow Up Of Mandibular Advancement Splint Therapy in Obstructive Sleep Apnea

A thesis submitted in partial requirement for the degree of Master of Dental Science (Orthodontics)

by

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Discipline of Orthodontics
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2001
DEDICATION

This work is for Simone.

Thanks also to my family.
Acknowledgements

Sincere appreciation is expressed to:

Professor M.Ali Darendeliler

Dr Peter Cistulli

Dr Helen Gotsopoulos

Peter Petocz

Jin Qian, Liz Noakes, Gaye Hughes

My fellow students and colleagues
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<tr>
<td>AHI</td>
<td>Apnea Hypopnea Index</td>
</tr>
<tr>
<td>AI</td>
<td>Apnea Index</td>
</tr>
<tr>
<td>ANOVA</td>
<td>Analysis Of Variance</td>
</tr>
<tr>
<td>ASDA</td>
<td>American Sleep Disorders Association</td>
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<tr>
<td>BMI</td>
<td>Body Mass Index</td>
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<tr>
<td>CPAP</td>
<td>Continuous Positive Airway Pressure</td>
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<td>EMG</td>
<td>ElectroMyoGram</td>
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<tr>
<td>ESS</td>
<td>Epworth Sleepiness Scale</td>
</tr>
<tr>
<td>MAS</td>
<td>Mandibular Advancement Splint</td>
</tr>
<tr>
<td>MinSaO₂</td>
<td>MINimum oxyhaemoglobin SAturation level</td>
</tr>
<tr>
<td>mm</td>
<td>millimetres</td>
</tr>
<tr>
<td>MSLT</td>
<td>Multiple Sleep Latency Test</td>
</tr>
<tr>
<td>nonREM</td>
<td>Non-Rapid Eye Movement</td>
</tr>
<tr>
<td>NS</td>
<td>Not Significant</td>
</tr>
<tr>
<td>OA</td>
<td>Oral Appliance</td>
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<tr>
<td>OSA</td>
<td>Obstructive Sleep Apnea</td>
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<tr>
<td>RDI</td>
<td>Respiratory Disturbance Index</td>
</tr>
<tr>
<td>REM</td>
<td>Rapid Eye Movement</td>
</tr>
<tr>
<td>TMJ</td>
<td>Temporomandibular joint</td>
</tr>
<tr>
<td>UPPP</td>
<td>UvuloPalatoPharyngoPlasty</td>
</tr>
<tr>
<td>UPPG</td>
<td>UvuloPalatoPharyngoGlossoplastry</td>
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1 Introduction

Sleep and its' effect on health and quality of life are important health issues. Treatment modalities are many and varied ranging from counselling for lifestyle and weight control to major orthognathic surgical procedures (Battagel and L'Estrange, 1996; Sher et al., 1996). Oral appliance (OA) therapy is an important treatment for obstructive sleep apnea (OSA) and its' clinical use has grown in recent years with studies documenting their efficacy appearing regularly in medical journals (Schmidt-Nowara et al., 1996).

The US National Commission on Sleep Disorders Research estimated in 1993 that 40 million Americans suffered from chronic disorders of sleep, such as OSA, insomnia and narcolepsy (NIH, 1993). Sleep apnea was the most common form of disorder, affecting approximately 20 million people. Of these, it was estimated that more than 30 percent or 6.5 million people over the age of 30 experienced moderate to severe forms of sleep apnea. The prevalence of OSA (estimated at 7.5 percent of the population) was comparable to that for asthma and diabetes (6 – 12 percent). Using these statistics as a guide, and assuming global prevalence of OSA at 7.5 percent, JP Morgan Securities estimated there were more than 50 million people in the world suffering from varying degrees of OSA, including 1.5 million people in Australia (Gentle, 2001).

Sleep clinics in the US have increased in number from around 100 in 1985 to more than 2000, most of which are affiliated with hospitals (JP Morgan Securities, 2001). In addition, a US survey of 355 dentists, orthodontists and maxillofacial surgeons found 5 percent of their total patient load was evaluated or treated for either snoring or sleep apnea with an OA (Loube and Strauss, 1997). It is therefore conceivable that dental health professionals will have to deal with the long term consequences of OA use (Friedlander et al., 2000).
The literature review will cover sleep related breathing disorders with an emphasis on OSA and its' management by mandibular advancement splints (MAS) and the side effects and long term consequences of their use.

2 Literature Review

2.1 Sleep and Breathing

Sleep is not an homogeneous phenomenon but rather consists of two distinct states, referred to as non-rapid eye movement (non-REM) or quiet sleep and rapid eye movement (REM) or active sleep (Rechtschaffen and Kales, 1968). The two states are distinguished by a combination of behavioural and electrographic criteria. Non-REM sleep consists of four stages that are thought to represent progressively deeper sleep, with the deepest stages (3 and 4) being referred to as slow-wave sleep. REM sleep, during which dreaming occurs, is characterized by intense cerebral metabolic activity and central nervous system excitation, despite which incoming sensory information and outgoing motor activity are actively inhibited. In the normal adult, non-REM sleep and REM sleep alternate cyclically, with periods of REM sleep lasting 10 to 20 minutes and occurring every 90 to 120 minutes (Somers et al., 1993). Normal sleep is characterized by two to five percent of stage 1 sleep, 45 to 55 percent of stage 2 sleep, 13 to 23 percent of slow-wave sleep, and 20 to 25 percent of rapid eye movement sleep in young adults (Carskadon and Dement, 1994).

Non-REM sleep and REM sleep have several important physiologic influences on breathing, particularly on respiratory drive, stability, and ventilatory mechanics (Phillipson and Bowes, 1986). Overall respiratory drive is decreased during non-REM sleep, while during REM sleep, respiratory drive is often irregular. The influences of non-REM and REM sleep on respiratory drive and muscle activity are exerted on the muscles of the upper airway in addition to those of the chest wall. Upper airway
resistance is increased during non-REM sleep compared with wakefulness, and increased even more during REM sleep (Phillipson and Bowes, 1986). Since 1990, respiratory disturbances during sleep have come to be recognized as extremely common disorders with important clinical consequences. Sleep related breathing disorders describes a broad spectrum of conditions in which individuals have a complete cessation of breathing (apnea) or a marked reduction in airflow (hypopnea) while asleep (Collard et al., 1996; Carskadon and Dement, 1994).

2.2 Snoring

2.2.1 Definition and Epidemiology

Snoring is the noise generated by vibration of the relaxed soft palate, uvula and faucial pillars on inspiration during sleep (Lugaresi et al., 1984). Muscle relaxation narrows the airway space and the increased air velocity causes vibration of the soft tissue structures (Cistulli and Sullivan, 1994). Habitual snoring has been estimated to affect 20 percent of middle-aged men and 10 percent of middle-aged women and is a sign of a partially obstructed upper airway during sleep (Gislason et al., 1993). Ohayon et al. (1997) reported that 40 percent of the UK population snored. Both men and women who are habitual snorers tended to have a higher prevalence of apnea and hypopnea scores (Young et al., 1993). Snoring may occur without apneas but with significant sleep disturbance and sleepiness. This has been termed “upper airway resistance syndrome” (Guilleminault et al., 1992). Risk factors for being an habitual snorer are age, body mass index (BMI), weight gain, smoking habits, and physical activity (Koskenvuo et al., 1987). In men, the prevalence of snoring increases up to the age of 50 to 60 years and is then followed by a decrease. Weight gain is a risk factor for snoring in all age groups, while smoking is mainly associated with snoring in men before 60 years of age (Lindberg et al., 1998).
2.2.2 Clinical Consequences

Only 30 years ago, snoring was still regarded as a social nuisance that was harmless to the sleeper. (Kleitman, 1963). The recognition of OSA during the 1970s markedly increased interest in snoring as the most frequent complaint in patients with OSA. Snoring is now recognised as a symptom that may be related to clinical conditions with significant morbidity. Snoring has been identified as a risk factor of and possible risk factor for hypertension, ischaemic heart disease and stroke, although its’ aetiological role in these conditions is controversial (Waller and Bhopal, 1989).

Even in the absence of apneas, snoring has been found to be highly associated with excessive daytime sleepiness, subjective work performance problems, and morning headache (Ulfberg et al., 1996). Blood oxygen desaturation has occasionally been found in subjectively healthy snorers, and this implies that heavy snoring can be considered a preliminary stage of OSA (Andersson and Brattstrom, 1991). Furthermore, the treatment of non-apneic snorers by surgery has been reported to result in a significant decrease in daytime sleepiness (Janson et al., 1994; Boudewyns et al., 1997). In addition, the antisocial consequences of snoring are causing a demand for treatment (Schmidt-Nowara et al., 1995).

2.3 Sleep Apnea

2.3.1 Definition and epidemiology

An apnea is defined as a cessation of breathing during sleep that lasts for 10 seconds or more. An hypopnea, or reduction in airflow, is when a 50 percent or greater reduction in tidal volume occurs, simultaneously with a four percent or greater reduction in blood oxygen saturation, lasting 10 seconds or more (Guilleminault et al., 1980). The average number of episodes of apnea plus hypopnea per hour during sleep is called the Apnea Hypopnea Index (AHI) or the Respiratory Disturbance Index (RDI). OSA is diagnosed when at least five apneic episodes of 10 seconds duration per hour occur during seven hours of nocturnal sleep (Guilleminault and Dement, 1978).
The estimated prevalence of sleep-disordered breathing, defined as an RDI of five or higher, was nine percent for women and 24 percent for men from data derived from the Wisconsin Sleep Cohort Study. OSA has been conservatively estimated to affect two percent of women and four percent of men in the middle-aged work force (Young et al., 1993).

2.3.2 Pathophysiology

The definitive event in OSA is posterior movement of the tongue and palate into apposition with the posterior pharyngeal wall, resulting in occlusion of the nasopharynx and oropharynx (Hudgel, 1992). The resulting airway obstruction initiates a primary sequence of events that may repeat itself hundreds of times each night. (Fig 1.)

Figure 1. Primary sequence of events, physiologic responses and clinical features of OSA. (Phillipson and Bowe, 1982)
With the onset of sleep, the upper airway occludes resulting in cessation of airflow despite continuing respiratory efforts. As a result of apnea, progressive asphyxia develops until there is a brief arousal from sleep, restoration of upper airway patency and resumption of airflow (Bradley and Phillipson, 1985). With the relief of asphyxia, the patient quickly returns to sleep, only to have the sequence of events repeated (Guilleminault and Dement, 1980). Several underlying mechanisms contribute to the onset and termination of upper airway occlusion during sleep. Upper airway collapse occurs when the subatmospheric pharyngeal pressure generated during inspiration exceeds by some critical amount the stabilising forces generated by the dilator and abductor muscles of the upper airway (Suratt et al., 1983). There are predisposing factors as well as structural and functional factors contributing to the development of a critical airway collapsing pressure (Hudgel, 1992).

2.3.2.1 Predisposing Factors

Different factors are apparent in the severity of OSA. Pre-existing pulmonary disorders such as asthma and chronic obstructive airways disease make breathing more difficult, so favouring decreased blood oxygen tensions (Bradley and Phillipson, 1985). Substances which depress the central nervous system, such as alcohol, sedatives, and sleeping pills have been shown to contribute to relaxation of the pharyngeal musculature, and therefore airway occlusion (Battagel and L'Estrange, 1996). Obesity and excess fat in both peripharyngeal and subcutaneous regions will further diminish the available airway space and may be involved in a lack of control of the oral and pharyngeal musculature (Horner et al, 1989). Ryan and Love (1996) found obese patients with large necks have a more collapsible velopharynx during wakefulness, which may predispose to upper airway obstruction during sleep. Hypothyroidism has a well established link with OSA and therefore all individuals should undergo thyroid function tests (McNamara et al., 1994). Other risk factors including male gender (Young et al., 1993), sleeping position (Pae et al., 1994), neck circumference (Davies and Stradling, 1990; Stradling and Crosby, 1991), familial tendency (Redline
et al., 1995), Marfan's syndrome (Cistulli and Sullivan, 1993), acromegaly
(Rosenow et al., 1994), non insulin dependent diabetes (Brooks et al., 1994)
and heavy smoking (Wetter et al., 1994) have been associated with
obstructive sleep apnea.

2.3.2.2 Anatomical Structures

It has been suggested that snoring or OSA patients may have anatomic head
and neck abnormalities that can be assessed by means of cephalometric
analysis. However, evidence for a direct casual relationship between
craniofacial structure and OSA has not been established (Miles et al., 1996).

Nevertheless, one of the most widely published deviating cephalometric
variables in these patients is the size and position of the mandible.
Mandibular micrognathia or retrognathia has frequently been described
(Coccagna et al., 1978; Conway et al., 1972; Rivlin et al.,1984; Andersson
and Brattstrom, 1991; Battagel and L'Estrange, 1996; Hochban and
Bradenburg, 1994). Tsuchiya et al. (1992) showed mandibular retrognathia
in non-obese patients was associated with a high apnea index. Lowe et al.
(1986) found that patients with obstructive sleep apnea often have posteriorly
positioned maxillae and mandibles, steep mandibular planes, high upper and
lower facial heights, and anterior open-bites, all associated usually with a long
tongue. Battagel et al. (2000) compared subjects with OSA and snoring
versus controls and found that, of the hard tissue measurements, only the
cranial base angle and mandibular body length showed significant intergroup
differences. Both measurements were significantly larger in the control
group.

In contrast, Pae and Ferguson (1999) found a decreased lower face height
and deep overbite but not a sagittal skeletal discrepancy may be associated
with severe OSA in non-obese patients. Similarly, neither Bacon et al. (1990)
nor Tangugsom et al. (1995) found a reduction in mandibular body length in
OSA patients. Sakakibara et al. (1999) found that upper airway soft tissue
enlargement may play a more important role in the development of OSA in
obese subjects whereas bony structure discrepancies, such as anteroposterior dimensions, may be the dominant contributing factor for OSA in non-obese subjects. Brander et al. (1999) showed that differences in upper airway size between obese and non-obese subjects were independent of bony craniofacial structure. Consequently, it would appear simplistic that there is a particular skeletal pattern observable in a lateral cephalogram to predict OSA.

When the airway and associated structures were examined by Battagel et al. (2000) both snorers and OSA subjects exhibited narrower airways, reduced oropharyngeal areas, shorter and thicker soft palates, and larger tongues than their control counterparts. The soft palate was larger and thicker, both lingual and oropharyngeal areas were increased and the hyoid was further from the mandibular plane in OSA subjects. Their study concluded that whilst the dento-skeletal patterns of snorers resembled those of subjects with OSA, some differences in soft tissue and hyoid orientation were apparent. There was not, however, a recognizable gradation in size of the airway and its associated structures from control through snoring to OSA subjects. They suggested that there may be a cephalometrically recognizable predisposition towards the development of sleep disordered breathing, but that this is only one facet of the condition. This aspect of cephalometric analysis was supported in a study of non apneic subjects by Trenouth and Timms (1999) where oropharyngeal airway was positively correlated with mandibular length, the distance between the third cervical vertebra and the hyoid bone and cranial base angle.

Hyoid bone position has been described to be of vital importance because it partly determines the shape, size, and posture of the tongue (Tangugsorn et al., 1995). This study found a more inferiorly positioned hyoid bone in patients with OSA, level of cervical vertebrae being at C4-C6, compared with C3-C4 in controls (Tangugsorn et al., 1995). Riley et al. (1983) found an increased soft palate length, a lower hyoid bone position and a decreased posterior airway space were significant cephalometric parameters in patients with the condition. Partinen et al. (1988) showed similar findings. When the
hyoid bone to mandibular plane distance was greater than 24mm and the posterior airway space equal to or less than 5mm, the RDI was increased.

Computed tomographic studies by Lowe et al. (1986) have shown that the upper airway is narrower in patients with obstructive sleep apnea. They also observed that obese patients had larger tongues and soft palates and suggested a link between obesity and the abnormal upper airway observed in patients with OSA. Fat deposition may be contributing to an enlargement of the soft palate and tongue in obese patients with OSA.

Most of the cephalometric data tend to support the concept of both craniofacial and soft tissue abnormalities in subjects with OSA, but the results are inconsistent. These findings must be viewed in the knowledge that the cephalometric view provides a limited two dimensional picture. Radiographic examination of the face and airway has been historically used and is efficient, cheap and readily available. Woodson and Naganuma (1999) suggested the use of supine endoscopy for upper airway evaluation in OSA as they found it correlates significantly with objective measures of apnea severity and posterior airway space as viewed on cephalograms.

2.3.2.3 Functional Aspects

Head posture, body position and muscle function have been investigated for their effect on OSA. Deviated head posture and an increased cranio-cervical angle have been described in subjects with OSA by Ozbek et al. (1998), Tangugsorn et al. (1995) and Solow et al. in 1993 and again in 1996. It was suggested that the large cranio-cervical angle is a physiological adaptation to maintain airway adequacy.

Other soft tissue changes with different body positions have been investigated by Pae et al. (1994) who showed that the thickness of the soft palate increases and the oropharyngeal cross-sectional area decreases when a patient changes from an upright to supine position. However, they stated soft palate size may be less important than vertical and anteroposterior
position of the tongue. Pae et al. (1998) then used statistical analysis of coordinate data to show tongue shape changes, hyoid bone and fourth vertebra movement as well as inferior movement of the submental area with a change from upright to supine. The study speculated that a large neck circumference in OSA patients may be caused by, not only fat deposition in the neck, but also by repositioning of the hyoid bone and vertebra. Upright versus supine cephalometric analysis has been debated with one group, (Yildirim et al., 1991) advocating supine cephalometry and another group (Pracharktam et al., 1994) concluding that this technique does not add any more information.

Vertical mandibular posture is also thought to be related to narrowing of the upper airway (Lavie, 1987). Mouth opening has been found to be associated with an inferior-posterior movement of the mandible and the tongue, which decreases the pharyngeal diameter (Kuna and Remmers, 1985). Although this may explain the demonstrated relation between an open mouth posture and upper airway collapse (Suratt et al., 1983) the mechanisms that may be responsible for these inter-relations remain controversial.

Muscle function investigated by Adachi et al. (1993) suggested that genioglossus function in individuals with OSA may be impaired allowing the prolapse of the tongue against the posterior pharyngeal wall with inspiratory effort during sleep. Evidence also suggests that a general hypotonia of the pharyngeal dilating muscles of the upper airway can also be involved in allowing an obstruction of the upper airway (Nasser and Rees, 1992). Mezzanotte et al. (1992) reported increased activation of the upper airway muscles in patients with OSA when awake indicating a greater need for muscular control of airway patency. This was supported with findings that use of continuous positive airway pressure (CPAP) in OSA patients significantly reduced genioglossus electromyographic (EMG) activity.

The aetiology and pathophysiology of OSA are clearly complex and multifactorial, resulting from a combination of predisposing, anatomical and functional factors.
2.3.3 Clinical Features

2.3.3.1 Symptoms of OSA
The most common manifestations of OSA are snoring and excessive daytime sleepiness (Guilleminault and Dement, 1978). The most common nocturnal symptom is loud snoring which has usually been present for years before other symptoms develop. The snoring takes on a temporal character, being interrupted periodically by episodes of silence corresponding to the occlusive events (Lugaresi et al., 1984). The termination of each apnea episode is usually marked by a very loud snoring gasp, accompanied by body movement. The patient may be unaware of these events but often complains of a disturbed or restless sleep, as does the bed partner (Flemons and Remmers, 1996). Occasionally, the patient awakens fully and may complain of choking, air hunger, or insomnia. Patients can complain of an unrefreshing sleep, non alert mental state, disorientation, morning headache and a sore and/or dry throat (Battagel and L'Estrange, 1996).

Daytime sleepiness develops under passive conditions such as watching television and reading. This may progress to daily activities such as driving as car or operating machinery in severe cases. Tests of driving ability reveal significant impairment in patients with OSA, and rates of motor vehicle accidents among these patients are considerably increased compared with control subjects (George et al., 1987; Findley et al., 1989). Signs of intellectual impairment, memory loss, poor judgment, and personality changes are also associated with OSA (Strohl et al., 1984).

2.3.3.2 Clinical Consequences of OSA
Sleep-disordered breathing has been hypothesized to have a close relationship with hypertension. The relationship between hypertension and three forms of sleep-disordered breathing (chronic snoring, breathing pauses and OSA) was found to be an independent risk factor for hypertension in 13,057 individuals aged 15-100 years (Ohayon et al., 2000). In addition, Hung et al., (1990) showed a highly significant association between sleep
apnea and myocardial infarction even after control for known coronary risk factors (Hung et al., 1990).

2.3.4 Diagnosis of OSA

2.3.4.1 Polysomnography
Polysomnography is the current standard for the evaluation of sleep-disordered breathing (Martin et al., 1985). It provides detailed data on abdominal and thoracic respiratory effort, nasal and oral airflow, blood oxygen saturation, sleep stage, and neurophysiological recordings (Ryan et al., 1995). Body position, leg and body movements and sounds can also be recorded (Battagel and L'Estrange, 1996). However, this recording is time consuming and has a low cost efficiency. Therefore other reliable diagnostic methods have been developed, such as blood oxygen saturation measurement in combination with monitoring of the breathing pattern and body position (Svanborg et al., 1990). Portable sleep studies conducted at home have been shown to provide reasonable negative predictive values in the diagnosis of OSA in symptomatic patients (Portier et al., 2000; Schafer et al., 1997).

2.3.4.2 Clinical Examination
A comprehensive history and clinical examination are unreliable in diagnosing OSA (Viner et al., 1991). Nevertheless, a complete history and examination accompanied by the sleeping partner is necessary. Wiggins et al. (1990) suggested that information obtained from a bed partner may significantly improve the accuracy of responses to questions about sleep and sleepiness. The clinical examination should include an ear, nose and throat assessment, so that any physical obstructions to breathing may be identified. Height and weight recordings are recommended to calculate the subject's body mass index (BMI) (Battagel and L'Estrange, 1996). This is calculated as the weight in kilograms divided by the square of height in metres. The normal range for BMI is considered to be between 20 and 25. Subjects with a BMI of greater
than 25 are overweight, whilst those whose index is greater than 30, are obese (Hoffstein et al., 1993).

2.3.4.3 Questionnaires
Different types of questionnaires have been used in different centres to assess symptoms of OSA and for use in epidemiological studies. Kump et al. (1994) evaluated the ability of a questionnaire in 465 participants in an epidemiologic study of OSA. Subjects and their room-mates each completed a questionnaire and underwent home sleep studies. Five main factors; functional impact of sleepiness, self-reported breathing disturbances, roommate-observed breathing disturbances, driving impairment and insomnia explained 67 percent of the total variance in the questionnaire data. Use of symptoms with data on gender and BMI improved predictive ability by 10 percent. The study confirmed that questionnaire data provide a valid means of characterizing symptoms in epidemiologic surveys of OSA. Predictive ability was not significantly improved with multiple questions or a separate room-mate questionnaire, but was improved with consideration of data on BMI and gender.

Excessive daytime sleepiness can be evaluated with both subjective and objective methods. Of the objective methods, the multiple sleep latency test (MSLT) is generally considered the gold standard (Carskadon and Dement, 1985). This test is based on the principle that degrees of sleepiness can be measured by how quickly one falls asleep (sleep latency) when given the opportunity to do so. However, the multiple sleep latency test is cumbersome and costly taking a day for both patient and polysomnographer.

The Epworth Sleepiness Scale (ESS) is a subjective questionnaire consisting of eight questions and yields a score of 0 to 24 (Johns, 1991). It is easy to administer and is currently the most widely used subjective test for sleepiness (Benbadis et al., 1999). Whilst polysomnography provides a definitive diagnosis, the ESS is merely a suggestive indicator of OSA. The sleepiness score is determined from a questionnaire designed to assess how likely a person would be to doze off in the following eight specific situations: 1) Sitting
and reading. 2) Watching TV. 3) Sitting inactive in a public place (e.g. in a lecture). 4) As a passenger sitting in a car for an hour. 5) Lying down to rest in the afternoon. 6) Sitting and talking to someone. 7) Sitting quietly after lunch, without having consumed any alcohol. 8) As the driver of a car, stopped for a few minutes in traffic. Scores of more than 12 (that is over 50 percent) suggest that the subject is sleepier than would be considered normal. The mean ESS score for normal subjects is 6 ± 2. OSA is rated at a score of 11.7 ± 4.6 (Johns, 1991). The reliability of ESS item scores for a particular subject has been reported to be high whether repeated by the same subject at different times or by another person such as a spouse. This reliability is only slightly less than that for measurements of sleep latency on the same day in the MSLT. The reliability of total ESS scores is higher again than for the separate item scores (Johns, 1994).

2.4 Management of OSA

Current treatment for patients with OSA can be categorised into conservative, medical, surgical and dental. The goals of treatment are to establish normal nocturnal oxygen saturation levels and to eliminate snoring and disruption of sleep due to upper airway resistance (ASDA, 1996).

2.4.1 Conservative Treatment

Eliminating risk factors for OSA is an important first step in treatment. Weight loss of ten percent has been shown to reduce the number of apneas by approximately 50 percent (Hudgel, 1996). Weight reduction in patients without anatomical risk factors can often eliminate OSA (Grunstein and Wilcox, 1994). Weight loss may also decrease the severity of apnea in part by reducing the size of the tongue and soft palate (Lowe et al., 1986) and the collapsibility of the upper airway (Suratt et al., 1983). Alcohol and sedatives should be avoided in the early evening (Liistro et al., 1995) as it prolongs apnea by delaying arousal (Berry et al., 1991). Training patients to sleep in a lateral position may be beneficial in mild forms of sleep apnea (Robertson, 1996). Coexisting chronic obstructive airways disease, asthma and
hypothyroidism are medical conditions known to exacerbate OSA. Adequate therapeutic control of these is therefore important (Strohl et al., 1984).

2.4.2 Medical Treatment

Medical treatment involves the use of CPAP. This treatment is regarded by most as the first line of treatment in patients with moderate to severe obstructive sleep apnea (Sullivan et al., 1981). This generally means patients with more than 20 episodes of apnea or hypopnea per hour with associated oxygen desaturations (Grunstein and Wilcox, 1994). CPAP provides dramatic relief of symptoms, by keeping the airway patent during sleep (Sullivan et al., 1981). A continuous stream of air under pressure is filtered and delivered to the pharynx, via a nasal mask. This constant flow is sufficient to prevent the airway from collapsing regardless of the position of the subject, but not enough to prevent periodic expiration. To be effective, the mask must fit firmly round the nose and be secured with straps and be in place for 6 hours per night, 7 days a week (Battagel and L'Estrange, 1996).

The advantages of CPAP are immediate beneficial effects, including improvements in nocturnal oxygenation, reductions in the frequency of arousals, consolidation of sleep and augmentation of cardiac output (Malone et al., 1991). In addition, long term treatment with CPAP results in marked improvements in left ventricular function during daytime (Malone et al., 1991). The disadvantages are noisiness from the pump and the anti social and intrusive nature of the mask. Dryness of the throat, burning and congestion of nasal mucosa and problems with the fit of the mask are also common problems (Teschler and Berthon-Jones, 1998; Hudgel, 1996). Compliance with CPAP varies from 60 to 85 percent with long term use closer to 50 percent (Hoffstein et al., 1992; Waldhorn et al., 1990). Covert monitoring revealed compliance of less than half the night and less affected patients were more likely to discontinue CPAP use (Engelman et al., 1994).
2.4.3 Surgical Treatment

Surgical methods to eliminate OSA aim to either enlarge or bypass the upper airway. The surgical procedures include reduction of the inferior turbinate bones, adenoidectomy (nasopharyngeal involvement), uvulopalatopharyngoplasty (UPPP), uvulopalatopharyngoglossoplasty (UPPG), tonsillectomy (oropharyngeal involvement), genioglossal and hyoid advancement (hypopharyngeal involvement), bimaxillary advancement, and tracheotomy (ASDA, 1996).

Classical surgical techniques such as nasal-septal reconstructions, cauterization and tonsillectomy are less favoured due to frequent failures (Sher et al., 1996). UPPP and UPPG enlarge the retropalatal airway through excision of the tonsils and trimming and reorienting the anterior and posterior tonsillar pillars in conjunction with excision of the uvula and posterior part of the palate. Patients with a narrow oropharynx relative to tongue size have been reported to have a good response to UPPP (Fleetham et al., 1992). However, the technique was found to be curative in less than 50 percent of patients (Shepard and Olsen, 1990). Complications included velopharyngeal incompetency, post operative bleeding, nasopharyngeal stenosis and voice change (Sher et al, 1996).

Surgical treatment involving bimaxillary advancement is recommended for patients with a retrognathic dolichofacial type combined with pharyngeal narrowing (Riley et al., 1993; Riley et al., 1989). Surgical advancement of the maxilla and mandible of at least 10mm has been suggested to ensure success (Hochban et al., 1995). Other major surgical techniques designed to advance the hyoid and associated structures include inferior sagittal mandibular osteotomy and genioglossal advancement with hyoid myotomy and suspension (Riley et al., 1989). Treatment success was varied depending on patient selection and measurement criteria and ranged from 20 to 100 percent (ASDA, 1996). It was suggested that successful surgical treatment for OSA requires judicious selection of the available techniques with regard to the regional analysis of the individual patient’s pattern of airway compromise (ASDA, 1996).
In severe instances of OSA that are life-threatening the ultimate treatment is tracheotomy (Sher et al., 1996). This procedure completely bypasses the upper airway and thus all upper airway obstruction. The procedure is, however, usually used as the last option in the treatment of OSA. Cohen et al. (1998) described mandibular distraction osteogenesis in the treatment of children with craniofacial deformities and OSA as another emerging surgical procedure in severe cases.

2.4.4 Dental Treatment

For treatment of OSA patients and patients with socially handicapping snoring, there is a need for a treatment modality that benefits patients who are not suitable for surgery or CPAP treatment, who are poor surgical risks, reject surgery or CPAP, or have not responded adequately to surgical or CPAP procedures.

Dental appliances in the treatment of OSA are of three classes (Lowe, 1994). One type attempts to lift the soft palate with a distal extension from a palatal plate (Marklund et al., 1996). This has not found wide acceptance due to gagging and the uncertainty of maintaining hypopharyngeal width during sleep. The second class of appliances is designed to act directly on the tongue by holding it forward by means of negative pressure from an anterior suction bulb or proprioceptive reminder. The tongue retaining device (TRD) is the most successful of these appliances, especially in the elimination of snoring (Cartwright and Samelson, 1982).

The third group of appliances repositions the mandible in a more protrusive position and is generally accepted as being the most effective design in the elimination of both snoring and obstructive sleep apnea (Lowe, 1994). This category of appliances has numerous terminology including mandibular advancement appliances (MAA) (Schmidt-Nowara et al., 1995), mandibular advancement splints (MAS) (O'Sullivan et al., 1995) and anterior mandibular positioning appliances (AMP) (Clark et al., 1993). For the purpose of this paper, oral appliances that advance the mandible will be termed mandibular
advancement splints (MAS).

Loube and Strauss (1997), in their survey of 355 dental practitioners found 25 different types of oral appliances were being used with appliances that advance the mandible making up 93 percent. Custom fit MAS were most frequently used (45 percent) followed by adjustable appliances (34 percent), pre-fit appliances (14 percent) and TRD (7 percent). The following sections will refer to oral appliances that advance the mandible.

2.5 Mandibular Advancement Splint Therapy

2.5.1 Background

The role of dental appliances in the management of upper airway obstruction was recognised as early as 1902 (Robin, 1902). In 1984, Meier-Kwert et al., (1984) reported on a MAS and in 1985, Soll and George (1985) showed improvement in apnea index and oxygen saturation with a one piece MAS. The initial success of this treatment (reduction in RDI from 48 to 9 and disappearance of snoring) contributed much to the subsequent interest in MAS.

By 1995, there was sufficient evidence for the efficacy of oral appliances that the American Sleep Disorders Association (ASDA) issued practice guidelines which state that OA therapy is indicated for simple snoring, for mild OSA, and for moderate to severe OSA if CPAP is not accepted or if surgery is not appropriate (ASDA, 1995). The guidelines are based on a review of 21 papers which showed reduction of snoring in a high proportion of patients and correction of OSA in most, although not all, of the patients (Schmidt-Nowara et al., 1995). This further increased interest in OA and resulted in additional evaluation of this treatment modality.

The presence or absence of OSA must be determined before initiating treatment with MAS to identify those patients at risk due to complications of sleep apnea and to provide a baseline to establish the effectiveness of
subsequent treatment (ASDA, 1995).

2.5.2 Appliance Design

Despite the wide variety of appliances described in the literature, most study groups have been small and there are few indications as to which design features maybe important for success (Johal and Battagel, 1999). Johal and Battagel (2001) suggest that appliances be designed with regard to the following factors:

1. A healthy dentition and supporting periodontium.

2. Sufficient protrusion to maintain airway patency. Whilst the degree of forward protrusion attainable will vary from individual to individual, Johal and Battagel (2001) aim to achieve the maximum comfortable protrusion. This was reported to be between 50 percent and 75 percent of the subjects' maximum protrusion. They advocated MAS designs that allow incremental advancement, for example, the removable Herbst design.

Mehta et al. (2001) advanced the mandible an average 7.5mm representing 78 percent of maximum protrusion. Clark et al. (1993) suggested that greater than 75 percent of maximum protrusion is needed for a satisfactory effect, however cautioned against unnecessarily large mandibular advancements because the long-term negative side effects on occlusion and the TMJs are unknown. Marklund et al. (1998) found satisfactory treatment results were found at mandibular advancements ranging from 41 percent to 88 percent of maximum protrusion. Success rate was higher in advancements of at least 5mm. The results also suggested that larger mandibular advancements are needed for treatment success in the more severe cases. Pantin et al. (1999) reported advancements as much as 16mm.

3. Minimal vertical opening. A MAS which promotes mandibular opening results in a downward and backward rotation of the mandible with a concomitant posterior movement of both tongue and soft palate. This can
negate the benefits to the airway from protrusion, resulting in the further narrowing of the pharyngeal airway, particularly at the level of the hypopharynx. The authors suggest that excessive vertical opening may explain why the 40 percent of subjects in the ASDA report (ASDA, 1995) were left with significantly elevated RDI levels. They did not provide specific measurements for this parameter.

Lamont et al. (1998) studied two different designs of MAS providing maximum advancement and 3mm to 4mm interincisal opening versus 70 percent advancement and 6mm to 9mm of opening. They found evidence of a slight benefit in the MAS with the greater vertical opening. Measurements of appliance advancement and opening in other studies is summarised in Figure 2. Further research is required to fully explain these variables.

4. Johal and Battagel (2001) suggested full occlusal coverage to prevent any unwanted changes in the occlusion resulting from over-eruption of unopposed teeth. No studies were cited to show vertical movements of molar teeth in either the short or the long term.

5. Good retention. It is important to ensure the splint is well retained by the dentition, in order to prevent disengagement and thereby loss of the desired antero-posterior opening of the airway achieved through forward posturing of the mandible (Johal and Battagel, 2001). The use of short intermaxillary elastics were suggested to help prevent mouth opening during sleep (Clark et al., 1996).

6. Incorporation of an anterior opening in the splint to allow easier airflow for mouth breathers. This is suggested as particularly helpful with vacuum formed one-piece appliances, however a two-piece design is preferred. Most research appears to be with one piece appliances or Herbst style. Mehta et al. (2001) used a two piece appliance allowing free mandibular opening movement and acrylic guide planes to hold protrusion.
Figure 2. Sagittal and vertical activations of MAS.

<table>
<thead>
<tr>
<th>First Author</th>
<th>Sagittal Activation</th>
<th>Vertical Activation</th>
<th>Appliance Design</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nakazawa et al., 1992</td>
<td>3 – 5mm</td>
<td>4 mm</td>
<td>One piece</td>
</tr>
<tr>
<td>Clark et al., 1993</td>
<td>75%</td>
<td>5 – 7mm</td>
<td>Herbst</td>
</tr>
<tr>
<td>Lowe AA., 1994</td>
<td></td>
<td>15mm</td>
<td></td>
</tr>
<tr>
<td>Yoshida, 1994</td>
<td>7mm</td>
<td>15mm</td>
<td>One piece</td>
</tr>
<tr>
<td>O'Sullivan et al., 1995</td>
<td>75%</td>
<td>10mm</td>
<td>One piece</td>
</tr>
<tr>
<td>Clark et al., 1996</td>
<td>65%</td>
<td></td>
<td>Herbst</td>
</tr>
<tr>
<td>Ferguson et al., 1996</td>
<td>3mm posterior to maximum</td>
<td>7mm</td>
<td></td>
</tr>
<tr>
<td>Menn et al., 1996</td>
<td>7 – 10mm</td>
<td>5 – 7mm</td>
<td>One piece</td>
</tr>
<tr>
<td>Lamont et al., 1998</td>
<td>70%</td>
<td>6 – 9mm</td>
<td>One piece</td>
</tr>
<tr>
<td>Marklund et al., 1998</td>
<td>4 – 6mm</td>
<td>5mm</td>
<td>One piece</td>
</tr>
<tr>
<td>Bondemark, 1999</td>
<td>5 – 8mm</td>
<td>5mm</td>
<td>One piece</td>
</tr>
<tr>
<td>Johal, Battagel 1999</td>
<td>75%</td>
<td>7mm</td>
<td>Herbst</td>
</tr>
<tr>
<td>Pantin et al., 1999</td>
<td>3 – 16mm</td>
<td></td>
<td>One piece</td>
</tr>
<tr>
<td>Ryan et al., 1999</td>
<td>Up to 11mm</td>
<td>1 – 5mm</td>
<td>One piece</td>
</tr>
<tr>
<td>Bondemark, Lindman, 2000</td>
<td>50 – 70%</td>
<td>5mm</td>
<td>One piece</td>
</tr>
<tr>
<td>Tegelberg et al., 1999</td>
<td>4 – 6mm</td>
<td>5mm</td>
<td>One piece</td>
</tr>
<tr>
<td>Liu et al., 2000</td>
<td>75%</td>
<td>7mm</td>
<td>One piece</td>
</tr>
<tr>
<td>Shadaba et al., 2000</td>
<td>Maximum comfortable</td>
<td>Minimum</td>
<td>Herbst</td>
</tr>
<tr>
<td>Marklund et al., 2001</td>
<td>4 – 6mm</td>
<td>5mm</td>
<td>One piece</td>
</tr>
<tr>
<td>Mehta et al., 2001</td>
<td>7mm</td>
<td></td>
<td>Two piece</td>
</tr>
<tr>
<td>Robertson C, 2001</td>
<td>3 – 14mm</td>
<td></td>
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</tr>
</tbody>
</table>

2.5.3 Mechanism of Action

The proposed mode of action of the MAS in the treatment of OSA is that it increases upper airway size and influences tongue muscle activity thereby improving airflow to the lungs (Lowe et al., 1990). Whether this is achieved by increasing the vertical dimension or via antero-posterior changes in the mandibular posture or a combination of both, remains to be confirmed.

Several studies have demonstrated an increase in the anteroposterior diameter of the upper airway following insertion of a MAS (Eveloff et al., 1994; Ferguson et al., 1997; Johal and Battagel, 1999). Whether these changes
were associated with an increase in the cross sectional area of the upper airway is unclear. Ferguson et al. (1997) have shown that voluntary mandibular and tongue protrusion increases the cross sectional area of the upper airway and alters its shape in patients with OSA and non-apnoeic control subjects. Assuming this effect on upper airway calibre is not eliminated by sleep, MAS may reduce the severity of OSA by maintaining patency of the velopharynx, particularly in its lateral dimension (Bonham et al., 1988).

Other mechanisms of action that have been proposed include increased upper airway calibre, decreased upper airway compliance, or activation of upper airway dilator muscles (Eveloff et al., 1994). MAS were believed to exert their effects predominantly in the oropharynx and hypopharynx but some studies have suggested an effect on the retropalatal airway also (Johal and Battagel, 1999).

2.5.4 Efficacy

Limited follow up data are available comparing the risk to benefit ratio associated with the use of MAS (Schmidt-Nowara et al., 1995). Large variability between different studies is undoubtedly caused by differences in apnea severity, facial morphology of patients, type of appliances, criteria for treatment success and methods of end point assessment (Pancer et al., 1999). Despite this and considerable variation in the design of MAS, the ASDA (ASDA, 1995) found the desired effects to be "remarkably consistent".

Snoring was reduced and often eliminated in almost all patients who used oral appliances and OSA was found to improve in the majority of patients (Schmidt-Nowara et al., 1995). Studies by Bonham et al. (1988), Lowe et al. (1994), Schmidt-Nowara et al. (1995), O'Sullivan et al. (1995), Eveloff et al. (1994), Marklund et al. (1998), Tegelberg et al. (1999), Ryan et al (1999) and Liu et al. (2000), have all reported significant improvement in RDI and blood oxygen saturations. Schmidt-Nowara et al. (1995) showed a reduction of mean RDI levels from 47 to 19 with approximately half of the treated patients
achieving RDI levels less than 10. However, as many as 40 percent of those treated were left with significantly elevated RDI levels.

The ASDA (ASDA, 1995) have indicated that treatment success with MAS is higher in those with mild to moderate rather than severe OSA. In a recent randomised, controlled study, Mehta et al. (2001) showed significant improvements in RDI, blood oxygen saturation and episodes of arousal. They concluded MAS to be effective in some patients with moderate or severe OSA. The effectiveness in treating OSA, however, is less with MAS therapy than with CPAP in randomised cross over studies (Ferguson et al. 1997; Clark et al., 1996)

Polygraphic assessments of sleep before and during MAS treatment have shown a reduction in stage 1 sleep, an increase in slow wave and REM sleep with a reduction in sleep fragmentation, mid sleep wake time and arousals. Most, but not all, patients reported an improvement in daytime sleepiness. (Schmidt-Nowara et al., 1995).

2.5.5 Compliance

Lowe et al. (2000) have tested a covert compliance monitor in a MAS in eight patients with OSA. They found an average 6.8 hours use per night. Kribbs et al. (1993) showed that 54 percent of OSA patients with CPAP therapy were considered irregular users with less than four hours of CPAP per night. Although not as effective as CPAP in the reduction of RDI, the study by Lowe et al. (2000) indicates MAS can be well tolerated throughout the night which emphasises its' long term beneficial effects. Other long term compliance studies with MAS have shown a range from 100 percent after following patients for up to 21 months (Ichioka et al., 1991) to 75 percent after seven months (Schmidt-Nowara et al., 1991) to 52 percent after 3 years (Clark et al., 1993).
2.6 Side Effects of MAS

As many consider that the treatment of OSA with MAS will be lifelong, knowledge of the long term sequelae are important (ASDA, 1995). Until recently, reports were either anecdotal, subjective or vague and often in small sample sizes (Schmidt-Nowara et al., 1995). Recent studies have investigated quantitatively the side effects and dental and skeletal changes with long term MAS use.

2.6.1 General Side Effects

Pantin et al. (1999) reviewed 106 patients treated over a five year period with mandibular advancement for OSA. Although 81 percent of their sample reported some side effects, namely excessive salivation (30 percent), xerostomia (23 percent), TMJ pain (26 percent), and myofacial pain (25 percent), these side effects were mostly minor and decreased with continuing use of the appliance.

Pancer et al. (1999) evaluated 121 patients following MAS treatment (65 of whom had worn the appliance for more than one year) and found that 45 percent of their patients complained of some side effects. The most frequent side effect was excessive salivation (32 percent) coupled with teeth and jaw discomfort (26 percent).

Tegelberg et al. (1999) followed up 49 dental appliance patients (37 of whom used the appliance for 12 months). They found the most frequent adverse events occurred in the initial part of the study and consisted of dryness of the lips, mouth and throat (affecting 43 percent of patients after three months falling to 13 percent after 12 months) which they attributed to open mouth breathing during sleep. Headache was the most commonly reported pre-treatment symptom with a frequency of at least once a week. An increased prevalence of morning headaches has been found previously among patients with heavy snoring and OSA (Aldrich and Chauncey, 1990). No patient discontinued MAS use because of these factors. Tiredness and stiffness of jaw function tripled from pre-treatment levels but halved between two weeks
and three months after MAS issue. Tooth tenderness diminished from affecting 19 percent of patients over the initial six months to 8 percent after 12 months.

Sleep bruxism is an oral habit characterized by a rhythmic activity of the temporomandibular muscles that causes a forced contact between dental surfaces during sleep (Macaluso et al., 1998). It is accompanied by tooth clenching or grinding that can be loud enough to be heard by the bed partner. Sleep bruxism has been linked to craniomandibular disorders including headaches, TMJ discomfort and muscle aches, premature loss of teeth due to excessive attrition and mobility, and sleep disruption of the individual as well as of the bed partner (Hartmann et al., 1987). Snoring, breathing pauses during sleep, and OSA have been reported to be significantly more frequent in sleep bruxism. Ohayon et al. (2001) suggest screening OSA patients for bruxism due to the risks of stomatognathic side effects when prescribing MAS.

Other follow up data indicate that oral discomfort is a common but tolerable side effect and seen by the majority of patients as being minor and not influencing their compliance. (Schmidt-Nowara et al., 1995; Pancer et al., 1999).

2.6.2 Dental Side Effects

Changes in the dentition have been reported with functional orthopaedic appliances in growing patients (Heather et al., 1998; Panchez et al., 1989). In general, proclination of lower incisors and retroclination of upper incisors occur because the pressure on the mandibular teeth is directed forward and the maxillary pressures are directed backward when the appliance is in place. However, the literature is limited on dental changes with protrusive appliances worn at night only in adults. McNamara (1984) showed very small dental changes with a Frankel appliance in a series of five case reports with treatment times from one to three years.
Robertson (2001) evaluated 100 consecutively treated OSA patients using lateral cephalograms. He found dental changes varied from six to 30 months with MAS use. No dental changes were observed after six months. However, at the 18 month review period a retroclination of the maxillary incisors, along with significant reductions in both overbite (0.9mm) and overjet (1.06mm) were observed. Following 24 months of treatment, the first changes in the mandibular incisor position were observed, with a proclination of these teeth of 2.20°. The most significant dental changes were, however, observed following 30 months of treatment with a 4.9° proclination of the mandibular incisors and a reduction in overbite of 1.82mm. One patient developed a 5mm posterior open bite that resulted in anterior tooth wear and the necessity of fixed appliances. It was concluded that changes in the angulation of the maxillary and mandibular incisors occur with increasing length of MAS use and that these changes are distinguishable from natural dental changes associated with aging.

Bishara and Jakobsen (1994) investigated the facial and dental changes in adulthood in an untreated normal sample. Overbite and overjet changed by less than 0.5mm for both males and females aged from 25 years to 46 years. Interincisal angle increased by 2.5° and lower incisors uprighted by less than 0.5° in females and 1° in males relative to the mandibular plane. Upper incisors also uprighted by 2.7° in females and by 1.5° in males compared to the sella-nasion line.

Marklund et al. (2001) measured a treatment induced change in overjet of $-0.4 \pm 0.8$mm and a change in overbite of $-0.4 \pm 0.7$mm in 47 patients after 2.5 ± 0.5 years. They also found smaller orthodontic side effects with increased mandibular opening with hard acrylic appliances. This led them to recommend the use of soft elastomeric devices because in these appliances orthodontic side effects were not related to degree of opening. Subjectively, 54 percent reported "no observed effect on the dentition" by the appliance. 40 percent reported "the occlusion changes in the morning after a night of MAS use but the occlusion becomes normal during the day", 5 percent reported a permanent change and one percent "didn't know".
In a pilot study, Bondemark (1999) investigated cephalometrically the effects of two years treatment with MAS in 30 patients diagnosed with habitual snoring and or OSA. He found no dental changes and attributed this to full arch coverage of the MAS. None of his patients reported any permanent sense of altered occlusion, and the anteroposterior distance between centric occlusion and centric relation did not exceed 1.0mm in any of the patients either before or after the treatment.

Pantin et al. (1999) reported occlusal changes resulting in an overjet decrease of 1mm to 3mm in 14 percent of their sample. Overall, 10 patients discontinued appliance wear due to adverse dental side effects. Occlusal changes were not investigated by Pancer et al. (1999) and neither Pantin et al. (1999) nor Pancer et al. (1999) undertook a cephalometric analysis.

2.6.3 Skeletal Side Effects

Bondemark (1999) observed small forward and downward changes in mandibular position accomplished by a minor increase in mandibular length and resulting in a decrease in both overjet (-0.4 ± 0.53mm) and overbite (-0.1 ± 0.26mm). A change in mandibular posture was observed in 17 patients, while 13 patients showed no change. He found the change in mandibular position might be the result of a condylar and/or glenoid fossa remodelling or a condylar positional change within the fossa as a compensatory reaction to MAS therapy.

He concluded that both skeletal and dento-alveolar changes may be associated with long-term mandibular advancement. The change in mandibular position, up to 2.0mm, that took place during the treatment period of 2 years in the study was significantly more pronounced compared with the small magnitude, between 0.05mm and 0.1mm per year, of natural changes in the dentofacial complex found over time in adulthood (Bishara et al., 1994; Forsberg et al., 1991). He stated that the statistically significant increase in mandibular length is less likely to be a functional adaptation than a structural change due to the lack of a centric occlusion/centric relation discrepancy.
Robertson (2001) found an increase in vertical face height, which he attributed to a repositioning of the head of the mandibular condyle in the glenoid fossa. Changes in condyle vertical position were observed at the first review period (six months), along with changes in vertical face height. No changes were observed, however, in the horizontal position of the condyle. He found no changes attributable to growth in any of the cephalometric variables measured. No studies were cited that investigated vertical movement of the molars as a result of MAS therapy and the possible effects that this type of orthodontic movement may have on the dentofacial complex.

It would appear that MAS therapy can affect the position of the dentition and the mandible although the changes largely go unnoticed by the patients. Prior disclosure to the patient regarding potential changes and regular dental reviews are recommended procedures (Robertson, 2001; ASDA, 1995; Schmidt-Nowara, 1995).

2.6.4 Effects on TMJ

Episodes of severe TMJ problems are generally limited to small numbers of patients in the studies available. Bonham et al (1988) reported no TMJ discomfort or soreness in a sample of 12 with OSA. Two patients from a sample size of 37 patients in the study by Tegelberg (1999) developed severe TMJ pain on palpation. The ASDA (1995) reports TMJ discomfort and changes in occlusal alignment are relatively uncommon occurrences but the long term risk is not well defined.

Bondemark and Lindman (2000) evaluated the status and function of the TMJ and masticatory system in patients with habitual snoring and OSA after two years nocturnal treatment with a MAS. They found no clinically recordable adverse effects on craniomandibular status and function.

Data reported in latter study was in the form Helkimo has called "dysfunction scores" (Helkimo, 1974a; Helkimo, 1974b; Helkimo, 1974c). Each patient's
Helkimo score summarizes the findings from the prescribed set of muscle and joint palpations, observations of sounds and smoothness of jaw movement, and measurements of mandibular mobility. Resulting scores may range from a low of zero, if the patient is normal and has no symptoms, to as high as 25 if the patient has severely impaired mobility, locking or luxation of a joint, tenderness to palpation in 4 or more of the 12 muscle palpation sites, tenderness to palpation to the posterior aspect of a joint, and pain on two or more of four mandibular movements.

In a previous study, Bernhold and Bondemark (1998) found six months treatment of snoring and OSA patients with a two piece magnetic MAS caused negligible changes of the anamnestic and clinical pain dysfunction index (Helkimo, 1974a). They concluded that MAS therapy for snoring and OSA seems to be well tolerated by the stomatognathic system in subjects with healthy TMJs and muscles at the start of treatment. However, the observation period was rather short, 6 months, and the mandibular advancements minimal.
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3 Manuscript

Long Term Follow Up of Mandibular Advancement Splint Therapy in Obstructive Sleep Apnea

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This study was approved by the South Eastern Sydney Area Health Service Research Ethics Committee (Southern Section)
Abstract

Mandibular advancement splints (MAS) are a recognized therapeutic option in the treatment of obstructive sleep apnea (OSA). This study aimed to investigate side effects and possible long term changes on the dentofacial complex with long term MAS use. The sample consisted of 112 patients with OSA. Questionnaires were completed by 78 patients (70 percent), 12 patients (10.5 percent) had been advised to stop MAS use and 22 patients (19.5 percent) were unavailable for follow up. MAS use was an average 25.1 ± 11.8 months (range 10.7 to 64.5 months) in 64 patients (82 percent) who completed the questionnaire and 14 patients (18 percent) had discontinued treatment. The most commonly reported side effects were jaw discomfort, tooth tenderness, excessive salivation and dry mouth. Subjective snoring improved in 69 patients (88 percent) and daytime sleepiness (Epworth Sleepiness Scale, ESS) scores significantly decreased from pretreatment to follow up appointment (11.4 to 7.1, p <0.001). Small subjective occlusal changes were experienced by 8 patients (12.5 percent). A reduction in overbite (-0.3 ± 0.08mm; p <0.01) and overjet (-0.2 ± 0.06mm; p < 0.05) was found and cephalometric analysis revealed statistically significant but clinically insignificant changes limited to anterior movement of the mandibular incisors (0.5 ± 0.12mm; p <0.001). Side effects of long term MAS use are common but mild and well tolerated by the majority of patients and dentofacial changes with long term MAS use are negligible.

Key words: Obstructive sleep apnea, mandibular advancement splint, side effects.
Introduction

Mandibular advancement splints (MAS) have been reported to be an effective treatment option for common upper airway disorders such as snoring and obstructive sleep apnea (OSA) \(^1\)-\(^3\). Snoring is a sign of a partially obstructed upper airway during sleep and affects 20 percent of middle-aged men and 10 percent of middle-aged women \(^4\),\(^5\). OSA is a common and chronic disorder estimated to affect 2 percent of women and 4 percent of men in the middle-aged work force \(^6\). It manifests as repeated partial or complete collapse of the upper airway during sleep affecting blood oxygen levels and sleep quality and quantity \(^7\). Consequently, a variety of health problems have been associated with OSA while snoring can be a major social problem \(^8\).

MAS therapy has gained increasing popularity over other treatment methods such as continuous positive airway pressure (CPAP) and surgical procedures due to its' easy acceptance, non-invasiveness, cost effectiveness and increased patient compliance \(^9\),\(^10\). Efficacy of oral appliances on OSA has been demonstrated by a reduction of sleep apnea parameters, such as apnea frequency and duration \(^1\),\(^11\)-\(^13\). However, little is known about the exact mechanism of their effect. The shared design feature of oral appliances is that they hold the mandible in a forward position changing the dimensions of the upper airway, including the hypopharynx, oropharynx and nasopharynx. This action improves upper airway patency and reduces its' collapsibility \(^14\)-\(^17\). It has been suggested that MAS therapy increases the passive muscle tension in the pharyngeal wall thereby reducing the vibration of the soft tissues and the turbulent airflow \(^10\).

There are many MAS designs with variable efficacies \(^18\). Most MAS are modified functional appliances used routinely in orthopedic treatment for growth modification in growing individuals. The usual method of securing the MAS to provide the mandibular protrusion is full arch occlusal coverage with acrylic. Appliances may be one-piece or two-piece and may include clasps
for retention 9,10,13,19,20. Some designs enable adjustments and the activation ranges from opening 2mm to 9mm vertically and advancing 3mm to 16mm sagittally 17,20-22. This equates to 50 percent to 75 percent of maximal protrusion 9,12,23.

Dental and bony changes with the use of functional appliances in growing patients are well documented 24-28. Mandibular unit length increases, restriction of forward maxillary growth, retroclination of upper incisors and proclination of lower incisors have been found with appliances resembling MAS designed for full time wear during growth 29,30. However, MAS are normally prescribed to adults for use during sleep only, and dental and skeletal changes would be considered undesirable. Considering the chronic nature of OSA and consequent length of time of MAS wear, there are limited published data on the dentofacial changes and side effects associated with long term MAS use.

Excessive salivation and transient discomfort for a brief time after awakening are commonly reported with initial use and may prevent early acceptance of an oral appliance 10,31. Temporomandibular joint (TMJ) discomfort, occlusal changes, dryness of the mouth, gum irritation and headaches are other side effects that have been investigated 11,19,21,32-34. Dental and skeletal changes associated with MAS use have been evaluated using cephalometric analysis in 100 patients by Robertson (2001)35 and in 30 patients by Bondemark (2000)34. Both studies found significant changes in the dental relationships and the position and/or size of the mandible. The aim of this study was to investigate the long-term effects of the use of MAS for the treatment of OSA on the dentition and bony structures and other side effects affecting MAS use.

Materials and Methods

The sample consisted of 112 patients (88 males and 24 females) recruited from the patient data bank at a multidisciplinary Sleep Disorders Clinic in a
university hospital. All patients had been assessed by a respiratory physician and an orthodontist associated with the sleep disorders clinic and had been prescribed MAS for their OSA. The selection criteria was the issue of a MAS at least six months prior to commencement of the study. Overnight polysomnography was undertaken prior to treatment with a MAS (T1) to confirm the degree of severity of the OSA and again after acclimatisation (T2) to assess MAS efficacy. Patients were sent a questionnaire and subsequently contacted as soon as was practicable for a clinical and radiographic examination. The study was approved by the institutional ethics committee and written informed consent was obtained from all patients.

**Appliance**

All patients used the same appliance which was a two-piece acrylic design described by Mehta et al., (2001)\(^1\), with full occlusal coverage and a unique screw device to titrate the advancement. The upper and lower appliances were independent of each other and allowed a hinge opening movement of the mandible to occur. Retention was obtained by occlusal and minimal buccal coverage with ball clasps interproximally at the premolars. The mandible was held in a protruded position via acrylic flanges rising superiorly from the lower appliance and resting against an acrylic block on the upper appliance. A screw mechanism was embedded in this acrylic block allowing sagittal titration of the mandible forward. The efficacy of this appliance was demonstrated by Mehta et al., (2001)\(^1\) in a randomized controlled study with rigorous criteria for treatment success. Significant improvements in sleep parameters were found compared with controls. The respiratory disturbance index (RDI, number of apneas and hypopneas per hour of sleep) more than halved on average and minimum oxyhaemoglobin saturation (minSaO\(_2\)) levels increased on average from 87 percent to 91 percent.

**Questionnaire**

A custom questionnaire was mailed to each patient to obtain subjective ratings of overall satisfaction with the MAS, treatment compliance, appliance fracture and the severity and frequency of a range of side effects associated
with use of the MAS. The severity of a side effect was rated as ‘mild’, ‘moderate’ or ‘severe’. The frequency of a side effect was rated as ‘rarely’, ‘sometimes’ or ‘often’ (Table 1). A table was included to rate the change in selected symptoms over the time frame of MAS use (Table 2). In addition, the questionnaire attempted to quantify subjective snoring frequency and intensity, headache frequency and severity, quality of sleep and daytime sleepiness. The latter was assessed by the validated Epworth Sleepiness Scale (ESS) 36. Patients were asked to consult with their bed partner when responding to the questionnaire.

Clinical Examination

The questionnaire was collected and followed by a clinical examination (T3), consisting of a medical and dental history, anthropometric measurements, an extraoral and intraoral examination as well as an evaluation of mandibular and TMJ function. Anthropometric measurements included neck circumference at the cricothyroid cartilage, and height and weight measurements to calculate body mass index (BMI). These measurements were compared to data taken at T1. The extraoral examination was carried out by one operator (RH) assessing the temporomandibular joint (TMJ) as outlined by Zarb and Carlsson, (1988)37. A history of pain and joint noises was included followed by palpation of the joint and the muscles of mastication according to Okeson, (1998)38. The intraoral examination included measurement of centric relation to centric occlusion (CR/CO) discrepancy, maximal opening, protrusion and left and right lateral movements 39. The patient’s functional evaluation was graded using the clinical dysfunction (D₈) and anamnestic dysfunction (A₈) indices and the index for occlusal state (O₈) according to Helkimo, (1974)40. In addition, a measure was taken of the vertical opening of the incisal edges and the overjet with the MAS in place using a dial caliper, closest to 0.05mm (Mitutoyo Corporation, Japan). The caliper was also used to measure the mandibular protrusion (MAS Pro) and maximal protrusion (Mx MAS Pro) with the MAS in place. Alginate impressions and a centric relation wax bite were taken to produce orthodontic study dental study models.
Dental measurements obtained from the study dental study models were compared with T1 measurements in a subset of patients whose records from T1 were available. These measurements included overjet (OJ), overbite (OB), upper arch length (UAL) and lower arch length (LAL). OB was defined as the vertical distance in millimetres between the incisal edges in CR of the upper and lower incisors and OJ was defined as the sagittal distance between the labial surfaces of the most prominent upper and lower incisor in CR. Arch length was measured as the perpendicular distance from the midpoint of the incisal edges of the central incisors to a line drawn between the mesial anatomic contact points of the first molars $^{41,42}$. The vertical distance from CR was calculated as the sum of the OB and the vertical distance between the incisors with the MAS in place. The percentage of maximal protrusion from CR (% Max Pro) provided by the MAS was calculated by the following formula:

$$\frac{\text{MAS Pro} + \text{OJ}}{\text{MxMASPro} + \text{OJ}} \times 100$$

**Radiographic Evaluation**

Lateral cephalometric radiographs were taken for each patient in centric occlusion both at T1 and T3 in natural head position with a barium mouthwash with a Phillips Orthoralix L.D. All radiographs were traced by one operator (RH) then digitised using Quick Ceph Image Pro/Quick Ceph 2000 (Quick Ceph™ Systems).

The cephalometric analysis was based on the methods described previously by Pancherz, (1982)$^{25}$ and Forsberg, (1991)$^{43}$. For sagittal measurements, the occlusal line (OL) and the occlusal line perpendicular (OLp) through sella from the first lateral cephalogram were used as a reference axis (Figure 1). This was transferred to the subsequent cephalometric tracing by superimposing on the nasion-sella line (NSL) and on the anterior contour of
the sella turcica 44. Sagittal changes were measured parallel to OL. For vertical measurements, the NSL-7° 42,45 and a line perpendicular to NSL-7° (NSL-7°p) through sella from the first cephalogram were used as a reference axis (Figure 2). This axis was transferred to subsequent cephalograms in a similar way as for the sagittal axis. Vertical changes were measured parallel to NSL-7°p. Soft tissue and angular measurements are detailed in Figure 3.

Sleep Studies

Overnight polysomnographs were recorded for all patients at T1 and for 95 percent of patients at T2. Variables were recorded continuously on a 20-channel computerized sleep monitoring system (Compumedics, Vic, Australia). Calculated respiratory variables were RDI and minSaO₂. Follow up sleep studies (T3) were performed on a smaller subset of patients with a portable sleep study machine (Autoset Portable II™, ResMed Inc, Ryde, Australia). Air pressure through a nasal cannula and percutaneous oxygen saturation levels through a non-invasive finger probe were measured and RDI and minSaO₂ calculated. A portable machine was utilized due to its' cost and time efficiency compared to overnight polysomnograph.

Statistical Procedures

Data were compiled and analysed using SPSS Software (Version 8; SPSS Inc., Chicago, Il, USA). Data are expressed as the mean and standard deviation (SD) at pretreatment and mean and standard error (SE) of the differences at follow up. Pretreatment versus recall values for the anthropometric, dental study model analysis and cephalometric analysis measures were compared using paired t tests. A repeated measures analysis of variance (ANOVA) for each of RDI, minSaO₂ and ESS, with the three time points being the repeated values, was carried out to compare changes in the sleep parameters in the three time points taken as a whole. Within-subject contrasts were carried out to compare T2 against T1 and T3 against T1. Differences with probabilities of less than five percent (p<0.05) were considered to be statistically significant.
Method Error

Twenty cephalograms of 10 randomly selected patients were digitized on two separate occasions. Mean absolute percentage error (MAPE) was calculated for all cephalometric values and found to be less than five percent for sagittal, vertical and angular measurements. MAPE was above five percent for the following soft tissue measurements: H-MP, PhW-spt, MinPhW and PAS indicating the difficulty in landmark identification for these points.

Results

Of the 112 patients, 17 (15 percent) were unable to be contacted and five (4.5 percent) did not wish to participate. Of the latter, the MAS had broken in one patient and three patients were using their appliance on occasions with one alternating with CPAP. Twelve patients (10.5 percent) were advised by their respiratory physician to stop MAS therapy after follow up polysomnographs revealed no improvement in their sleep parameters. The average time of MAS use for this group was between three and six months and all were advised to return to CPAP therapy. Four of these 12 patients reported satisfaction with the MAS despite polysomnography results and expressed disappointment with the advice to use CPAP.

The remaining 78 patients (70 percent) completed the questionnaire with 57 patients of this group (73 percent) undergoing a clinical examination. Sufficient records at T1 and T3 were available to complete study dental study model analysis in 45 patients (58 percent) and radiographic analysis in 46 patients (59 percent).

Fourteen patients (18 percent; 11 males) completing the questionnaire discontinued MAS therapy for the following reasons: general discomfort and jaw discomfort in three patients (4 percent); persistent snoring in two patients (2.5 percent); presumed ineffectiveness and a preference for CPAP in two patients (2.5 percent); concern for tooth damage and joint noises in two patients (2.5 percent); MAS breakage, recurring mouth ulcers and social
problems in one subject (1.3 percent) each; and a combination of side effects in two patients (2.5 percent). The average time of MAS use for this group was 2.9 months (SD 1.33) and 57 percent of the subset stopped within six months of receiving their appliance.

The results of the questionnaire based on the 64 patients (82 percent; 50 males) still using the MAS are summarized in Figures 4, 5 and Table 1 and 2. Eight patients (12.5 percent; 6 males) experienced none of the side effects listed in Figure 5. Bed partners assisted in answering the questionnaire in 46 patients (72 percent). Nine patients (14 percent) were not assisted by their partners and in the same number partners were not applicable.

Snoring was subjectively reported to be "better" in 56 patients (88 percent) of the subset continuing MAS use while six patients (9 percent) reported "no change". The remaining two patients (three percent) rated their snoring as "never a problem". However, 40 patients (63 percent) answered "yes" to snoring despite a frequency of MAS use greater than five nights per week (Figure 4). Snoring intensity was rated as "medium" or "low" by 60 patients (95 percent) of this group. Daytime sleepiness was subjectively "better" in 45 patients (70 percent) of the respondents using MAS while 14 patients (22 percent) found "no change" and in eight percent it was "never a problem". Correspondingly, ESS scores decreased significantly (p<0.001) from T1 to T3. Within-subjects contrasts showed the average ESS score drop from 11.4 at T1 to 6.9 at T2 then increase slightly but not significantly (p=0.83) to 7.1 at T3.

Replacement of the MAS was required in 16 patients (25 percent) with four (6 percent) needing replacement twice and one subject (1.6 percent) three times. Breakages and unsatisfactory fit of the MAS accounted for 11 (17 percent) and five (8 percent) replacements respectively.

The changes in variables measured on the 57 patients (44 males and 13 females) at the clinical examination and the dental study model analysis completed for 45 patients (33 males and 12 females) are detailed in Table 3.
The cephalometric analysis completed on 46 patients (34 males and 12 females) is summarized in Table 4. The average length of time of MAS use was 25.1 ± 11.8 months with a minimum of 10.7 months and a maximum of 64.5 months. The percentage of maximal protrusion provided by the MAS was on average 72.9 ± 11.9 percent with a range from 12.5 percent to 92.3 percent. The average vertical distance from CR with the MAS in place was 7.1 ± 2.2 mm with a range from 2.7 mm to 12.7 mm.

Clinical TMJ assessment at recall showed that 16 patients were clinically symptom free (D1 0=28 percent); 38 patients were experiencing mild symptoms (D1 I=67 percent); and three patients were experiencing moderate symptoms (D1 II=5 percent). No patients were assessed as having severe symptoms (D1 III=0). The patient’s subjective assessment of their TMJ status at recall showed that 35 patients were symptom free (A1 0=61 percent); 20 patients were experiencing mild symptoms (A1 I=35 percent); and two patients were experiencing severe symptoms (A1 II=4 percent). Assessment of the occlusal status showed that at recall one subject had no occlusal disturbances (O1 0=2 percent); 13 patients had moderate occlusal disturbances (O1 I=23 percent); and 43 patients had severe occlusal disturbances (O1 II=75 percent).

Subjective occlusal and jaw functional changes were reported in eight patients (12.5 percent) and two patients (3 percent) respectively. The occlusal changes were slight and non-specific except in three patients where they were described in terms of inability to chew nails, cut cotton thread or open packets with incisors since using MAS. The jaw functional changes were described by the two patients as a more forward habitual posture and a decrease in jaw muscle power.

RDI and minSaO2 data at T1 were available for all 57 patients (100 percent) that were examined clinically. At T1, the average RDI was 25.3 ± 17.74 per hour with a range from 3.0 to 81.0 per hour and the average minSaO2 was 86.7 ± 6.9 percent with a range from 54.0 to 96.0 percent. Data at T2 were available for 54 patients (95 percent) and at T3 for 16 patients (28 percent).
The smaller sample size at T3 was due to a failure of subjects to collect the machines despite making appointments and, in three cases, failure of the machine in recording the data.

There were significant differences for both RDI \((p=0.001)\) and \(\text{minSaO}_2\) \((p=0.003)\). Within-subjects contrasts for RDI showed significant differences for both T2 versus T1 \((p=0.001)\) and T3 versus T1 \((p=0.012)\). The mean RDI from T1 through to T3 was 25.4 to 7.8 to 13.7 per hour. Within-subjects contrasts for \(\text{minSaO}_2\) showed a significant difference for T2 versus T1 \((p=0.002)\) however, T3 versus T1 was not significant \((p=0.59)\). The mean \(\text{minSaO}_2\) from T1 through to T3 was 86.7 to 90.9 to 83.1 percent.

**Discussion**

The increasing popularity of MAS as a therapeutic option in the treatment of snoring and sleep related breathing disorders has necessitated more detailed investigation of their side effects and possible long term effects on the dentofacial complex\(^9\). This study has found that MAS therapy is subjectively effective and well tolerated despite the side effects indicated by a high proportion (87.5 percent) of patients. Of the contacted patients still using the MAS (82 percent), the majority (97 percent) found control of OSA symptoms to be satisfactory or better after an average 25.1 ± 11.8 months. Sleep quality was reported as ‘moderately’ or ‘very’ refreshing in 83 percent of patients and the subjective improvement in snoring and daytime sleepiness agrees with data in other studies \(^1,10,46\). Although the questionnaire was not validated, the reduction in subjective daytime sleepiness was supported by the significant decrease in ESS scores. A small study of covertly measured compliance has shown an average 6.8 hours MAS use per night \(^2\). Compliance of 5 nights per week or greater were reported by 83 percent of our sample. Furthermore, 97 percent tolerated the MAS greater than 5 hours per night. This is a relatively high level of compliance compared to recent long term studies \(^21,47\).
The most commonly reported side effects were jaw discomfort, tooth tenderness, excessive salivation and/or dry mouth. Patients reported that side effects were temporary or transient especially during the early stages of MAS use, however, these side effects were responsible for discontinuation of MAS use in 8 patients (10 percent). The proportion of the sample (less than 45 percent) experiencing these side effects was similar to that found in other studies 1,10,21,48,49. In general, the side effects were rated as mild with the exception of joint noises and headaches, which are symptoms along with craniomandibular problems such as feeling of fatigue of the jaws and facial pain that have been shown to increase with age in the general population 39. Increases in joint noises and an improvement in headache suffering with MAS use have been reported previously 34. Our data showed joint noises and headaches subjectively affected a small proportion of the sample at recall (12 percent and 16 percent respectively) and that the change in these symptoms during MAS use was rated as “better” in 9 percent and 13 percent respectively. These symptoms were “never a problem” or did not change for greater than 82 percent of patients during MAS use. Further study is needed on the effect of MAS therapy on the periodontal support and gingival health.

Sleep bruxism has been shown to occur at least weekly in over 8 percent of the general population and higher in patients with OSA and to have a significant effect on quality of life 50. The study found bruxism to have significant consequences (i.e., muscular discomfort on awakening, disturbing tooth grinding, or necessity of dental work) in half of the patients. We found only two patients to be sufficiently concerned about tooth damage to stop using MAS and 22 percent to indicate bruxing as a noted side effect with three percent rating it as worsening during MAS use. This information further supports findings that MAS therapy does not appear to have a major adverse effect on the stomatognathic system in the long term 19,21,34.

Little attention in the literature has been focused on maintenance of the appliance, which was found to be an important clinical factor with 25 percent of patients using MAS requiring a replacement. Adams clasps have been
reported to be an area of appliance weakness and both acrylic and metal components can break. Appliance design both for efficacy and durability is important. In this study, breakages tended to be at the screw mechanism. Further research on optimal mandibular advancement and opening may allow elimination of this weak spot.

Dental study model measurements revealed a statistically significant reduction in both overbite (-0.3 ± 0.08mm; \( p<0.01 \)) and overjet (-0.2 ± 0.06mm; \( p<0.05 \)) with MAS use although the changes were clinically insignificant. This finding was supported in the cephalometric analysis where dental changes were limited to the lower anterior teeth and lower molars moving anteriorly a statistically significant but clinically insignificant amount. Specifically, the lower anterior teeth proclined an average 0.96 ± 0.30 degrees \( (p<0.01) \) and the incisal edge moved anteriorly 0.52 ± 0.12mm \( (p<0.001) \). The mean absolute percentage error for these two measurements was 2.19 percent and 0.90 percent respectively indicating accurate reproducibility of the cephalometric analysis. This contrasts the findings of Robertson, (2001) where as much as 4.9 degrees proclination of the lower incisors occurred after 30 months of appliance wear. Their sample included only patients that stated compliance of five to six hours per night and seven nights per week appliance wear and this, in conjunction with the treatment time, may account for the markedly different result compared to the present study. Full occlusal coverage with acrylic may account for the lower molar anterior movement of 0.3 ± 0.77mm \( (p<0.05) \). There was no significant effect on the maxillary dentition, mandibular length (Co-Pg) or mandibular position (Pg-OLp) indicating that neither an orthopaedic effect nor a functional adaptation occurred. Similar results have been shown in a series of case reports of tissue borne functional appliances in adults by McNamara, (1984). However, Bondemark, (1999) found a small but statistically significant increase in mandibular length and/or position following 2 years’ nocturnal treatment with a MAS and speculated the cause to be condylar or glenoid fossa remodelling. Similarly, Robertson, (2001) showed a vertical repositioning of the condyle relative to cranial base as early as six months
into MAS use. Neither study investigated vertical movements of the molar
teeth as a factor in the change in mandibular position. We found no
significant vertical changes either dentally or skeletally but the anterior teeth
movements were similar to those found in other studies 19,21,34,47. No soft
tissue measurements showed a statistically significant change. Therefore,
long term MAS use does not appear to affect pharyngeal shape or hyoid
position.

Little subjective awareness of occlusal change has been shown in different
studies. Awareness of occlusal changes was found in only eight out of 15
patients with a measurable change in a sample size of 106 patients by Pantin,
(1999) 21; no patients in a sample size of 30 by Bondemark, (1999) 19; and
in 3 patients from a sample size of 69 by Marklund, (2001) 47. We found
awareness of a permanent occlusal change in 8 patients (12.5 percent) but in
no patients did it cause a discontinuation of MAS use. The 20 percent of
patients indicating a change in “the way [their] teeth bite together “ to be
“worse” reported the condition to be transient and limited normally to the 2
hours at most following MAS removal. The findings of the cephalometric and
study dental study model analysis are that the dental and skeletal changes
are minimal when measured both objectively and subjectively.

A limitation of the clinical data is the lack of a standardized pretreatment TMJ
assessment. Nevertheless, examined by one operator (RH) at T3, there was
a greater percentage of the sample that was symptom free with less severe
occlusal disturbances than that found in a general population sample studied
by Helkimo, (1974), in the development of the index 52. In addition, the 61
percent of symptom free patients (A\textsubscript{i} 0) at T3 in our study compares well with
the 69 percent of symptom free patients found by Bondemark and Lindman,
(2000) 34 after two years of nocturnal MAS use. The latter study also
showed similar results in the clinical dysfunction index with D\textsubscript{0}/D\textsubscript{1} I and D\textsubscript{1} II in
84 percent and 9 percent respectively compared to 95 percent and 5 percent
in our sample. Although TMJ problems are considered to be a risk in long
term MAS use 9, our findings show that jaw joint pain and joint noises are
indicated by less than a quarter of the sample to be a long term problem with severity rated as mild and frequency low in the majority of cases. Furthermore, the Helkimo scores suggest that symptoms of TMJ problems are less than or equivalent to those found in the general population and therefore, that long term MAS use is not detrimental to TMJ health and function.

Evaluation of the changes in polysomnographic variables was limited by the small number of patients undergoing T3 sleep tests and the different methods of polysomnograph recordings. Measures of RDI and minSaO₂ have been shown to vary in comparison with overnight polysomnography 53,54. Nevertheless, comparison of the results indicated a significant improvement in RDI and minSaO₂ from T1 to T2 followed by a trend toward T1 levels for both parameters at T3. Mean RDI values were almost halved from T1 to T3 however, mean minSaO₂ at T3 decreased to below T1 levels. MAS effectiveness may diminish and investigations are needed to understand the reported impressive subjective responses and compliance that contrast the objective responses of some sleep parameters ¹. Negligible improvement in minSaO₂ has been shown previously with MAS therapy 10,23,49,55. Further investigations with follow up overnight polysomnographs are needed to more accurately examine the long term effectiveness of MAS on sleep parameters.

Conclusion

MAS therapy in the treatment of OSA has been found to provide subjective and objective benefits with minimal dental and skeletal side effects. Compliance levels found in this study attest to the satisfaction of patients with MAS therapy despite a range of minor side effects associated with its' use by a large proportion of the sample. Management by a multidisciplinary team is essential to adequately assess the different facets of treatment progress and address problems specific to the different operator's skills. A recall program is recommended to monitor symptoms and the effects of the MAS on both the stomatognathic and respiratory systems.
Acknowledgements
This research was supported by The ASO Foundation for Research and Education Inc. We thank J.Qian, E.Noakes and G.Hughes for their assistance in collection of the data.
References


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Legends

**Figure 1.** Sagittal landmarks and measurements. Orientation lines are the registration line nasion sella line, (NSL), line joining sella (S), the midpoint of sella turcica and nasion (N), the most anterior point of the frontonasal suture; reference grid: occlusal line (OL), line joining midpoint between upper and lower molars and midpoint between upper and lower first premolars; occlusal line perpendicular (OLp), line perpendicular to OL intersecting S. **Anatomical Landmarks:** ss, (A point) deepest midline point on the maxillary alveolus between the anterior nasal spine and the maxillary alveolar crest; Pg, pogonion, the most anterior point on the outline of the bony chin; Co, condyion, the most superior, posterior point on the condyle of the mandible; is, incision superius, the incisal point on the most prominent maxillary incisor; ii' incision inferius, the incisal point on the most prominent mandibular incisor; ms, molar superius, the distal contact point on the maxillary molar, the midpoint was used if there was double projection; mi, molar inferius, the distal contact point on the mandibular molar, the midpoint was used if there was double projection; Measurements: ss-OLp, position of the maxilla; Pg-OLp, position of the mandible; Co-Pg, mandibular length; is-OLp, position of the most prominent maxillary incisor; ii-OLp, position of the most prominent mandibular incisor; ms-OLp, position of the maxillary molar; mi-OLp, position of the mandibular molar.

**Figure 2.** Vertical landmarks and measurements. Orientation lines are the registration line nasion sella line minus 7 degrees (NSL-7°) and a line perpendicular to NSL-7° through sella (NSL-7°p). **Anatomical landmarks:** indicates landmark is projected perpendicularly to NSL-7°p. Me', Menton, the most inferior point on the lower border of the mandibular symphysis; Go', the midpoint of the angle of the mandible. Measurements: n'-Me', anterior face height; S-Go', posterior face height; n'-is', position of the maxillary incisor; ii'-Me', position of the mandibular incisor; n'-ms', position of the maxillary molar; mi'-Me', position of the lower molar.
**Figure 3.** Soft tissue and angular measurements. **Anatomical landmarks:** ANS, anterior nasal spine, tip of the median sharp bony process of the palatine bone in the hard palate; B point, the deepest midline point between the mandibular alveolar crest and pogonion (Pg); C2, the tangent point on the dorsal surface of the second cervical vertebra to a line from C4, C4, the postero-inferior point of the fourth cervical vertebra; Eb, base of the epiglottis, the deepest point of the epiglottis; H, hyoidale, the most superior anterior point on the body of the hyoid bone; Ht, the most superior point on the tongue in relation to the line from Eb to T; MP, mandibular plane, line joining menton and gonion; P, tip of the soft palate; PP, palatal plane, line joining ANS and PNS; PNS, posterior nasal spine, tip of the posterior spine of the palatine bone in the hard palate; PhW, posterior pharyngeal wall, a point on the posterior pharyngeal wall at the same horizontal level as spt; pm, pterygomaxillare, the intersection between the nasal floor and the posterior contour of the maxilla; spt, the tangent point on a line parallel to pm-P on the dorsal surface of the soft palate at the maximum width; T, tongue tip, the most anterior point of the tongue which touches the lingual surface of the mandibular incisor. **Angular measurements:** SNA, sella, nasion, A point angle; SNB, sella, nasion, B point angle; ANB, A point, nasion, B point angle; MP/SN, mandibular plane angle to SN line; is/SN, maxillary incisor angle to SN line; is/PP, maxillary incisor angle to palatal plane; ii/MP, mandibular incisor angle to mandibular plane; ii/is, interincisal angle; ii/OL, mandibular incisal angle to occlusal plane. **Soft tissue measurements:** H-MP, perpendicular distance from the mandibular plane to hyoidale; C2C4-SN, craniocervical angle formed by a line from C2 to C4 and SN line, PhW-spt, width of the pharynx where soft palate is thickest, MinPhW, minimum pharyngeal width, pm-P, length of soft palate, PAS, posterior airway space, pharyngeal width at the base of the tongue parallel to a line intersecting Go and B point; spt, soft palate thickness; Tongue length; measured from T to Eb; Tongue height, measured perpendicularly from Ht to line from Eb to T.

**Figure 4.** Compliance and subjective satisfaction and effects of MAS use.
**Figure 5.** Side effects in the stomatognathic system experienced by patients using MAS. Figures are the percentages of the sample affected by the side effects.

**Table 1.** Rating of severity of symptoms by the patients who experienced side effects. Figures are the percentages of the affected patients with severity rated as mild, moderate or severe and frequency rated as rarely, sometimes or often.

**Table 2.** Changes in side effects of MAS use. Figures are the percentages of the sample.

**Table 3.** Patient variables and dental changes in patients attending follow up clinical examination.

**Table 4.** Effect of MAS on cephalometric measurements.
Figures and Tables

Figure 1. Sagittal landmarks and measurements.
Figure 2. Vertical landmarks and measurements.
Figure 3. Soft tissue and angular measurements.
**Figure 4.** Compliance, subjective satisfaction and the effects of MAS use on OSA symptoms.
**Figure 5.** Subjective side effects in the stomatognathic system experienced by patients using MAS.
Table 1. Rating of severity of symptoms by the patients who experienced side effects.

<table>
<thead>
<tr>
<th>Side Effect</th>
<th>Severity</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mild</td>
<td>Mod</td>
</tr>
<tr>
<td>Jaw Discomfort</td>
<td>75</td>
<td>21</td>
</tr>
<tr>
<td>Jaw Joint Pain</td>
<td>67</td>
<td>20</td>
</tr>
<tr>
<td>Tooth Tenderness</td>
<td>79</td>
<td>14</td>
</tr>
<tr>
<td>Excessive Salivation</td>
<td>69</td>
<td>31</td>
</tr>
<tr>
<td>Dry Mouth</td>
<td>66</td>
<td>30</td>
</tr>
<tr>
<td>Grinding of Teeth</td>
<td>60</td>
<td>40</td>
</tr>
<tr>
<td>Gum Irritation</td>
<td>80</td>
<td>10</td>
</tr>
<tr>
<td>Jaw Joint Noises</td>
<td>37</td>
<td>63</td>
</tr>
<tr>
<td>Headaches</td>
<td>50</td>
<td>40</td>
</tr>
</tbody>
</table>

Percentage of Patients

Severity: Mild, Moderate (Mod), Severe (Sev)
Frequency: Rare, Sometimes (Some), Often
Table 2. Subjective long term side effects of MAS use.

<table>
<thead>
<tr>
<th>Changes Affecting</th>
<th>Better</th>
<th>No Change</th>
<th>Worse</th>
<th>Never A Problem</th>
</tr>
</thead>
<tbody>
<tr>
<td>Way Your Teeth Bite Together</td>
<td>10</td>
<td>53</td>
<td>20</td>
<td>17</td>
</tr>
<tr>
<td>Jaw Discomfort</td>
<td>11</td>
<td>34</td>
<td>22</td>
<td>33</td>
</tr>
<tr>
<td>Pain in the Jaw Joint Area</td>
<td>9</td>
<td>38</td>
<td>15</td>
<td>38</td>
</tr>
<tr>
<td>Tooth Tenderness</td>
<td>9</td>
<td>30</td>
<td>23</td>
<td>38</td>
</tr>
<tr>
<td>Excess Salivation</td>
<td>13</td>
<td>30</td>
<td>25</td>
<td>32</td>
</tr>
<tr>
<td>Dry Mouth</td>
<td>22</td>
<td>34</td>
<td>17</td>
<td>27</td>
</tr>
<tr>
<td>Grinding of Teeth at Night</td>
<td>19</td>
<td>36</td>
<td>3</td>
<td>42</td>
</tr>
<tr>
<td>Gum Irritation</td>
<td>6</td>
<td>28</td>
<td>6</td>
<td>60</td>
</tr>
<tr>
<td>Jaw Joint Noises</td>
<td>9</td>
<td>25</td>
<td>2</td>
<td>64</td>
</tr>
<tr>
<td>Headaches</td>
<td>13</td>
<td>27</td>
<td>5</td>
<td>55</td>
</tr>
<tr>
<td>Fit of the Dental Splint</td>
<td>17</td>
<td>28</td>
<td>16</td>
<td>39</td>
</tr>
</tbody>
</table>

Percentage of Patients
**Table 3.** Anthropomorphic and dental changes in patients attending follow up clinical examination.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Before Treatment</th>
<th>Changes at Follow Up Appointment</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean ± SD</td>
<td>Range</td>
<td>Mean ± SE</td>
</tr>
<tr>
<td>Age (Years)</td>
<td>4.94 (9.82)</td>
<td>29.4 – 72.5</td>
<td>2.1 (0.13)</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>29.1 (4.99)</td>
<td>19.9 – 42.4</td>
<td>0.2 (0.23)</td>
</tr>
<tr>
<td>Neck Circumference (mm)</td>
<td>39.9 (3.18)</td>
<td>34.5 – 48.0</td>
<td>1.1 (0.23)</td>
</tr>
<tr>
<td>Upper Arch Length (mm)</td>
<td>25.2 (3.71)</td>
<td>17.3 – 35.6</td>
<td>0.0 (0.10)</td>
</tr>
<tr>
<td>Lower Arch Length (mm)</td>
<td>21.5 (3.76)</td>
<td>13.5 – 31.7</td>
<td>-0.2 (0.11)</td>
</tr>
<tr>
<td>Overbite (mm)</td>
<td>2.8 (2.28)</td>
<td>-3.6 – 9.8</td>
<td>-0.3 (0.08)</td>
</tr>
<tr>
<td>Overjet (mm)</td>
<td>2.9 (1.32)</td>
<td>0.0 – 6.1</td>
<td>-0.2 (0.06)</td>
</tr>
</tbody>
</table>

NS = Not Significant
Table 4. Effect of long term MAS use on cephalometric measurements.

<table>
<thead>
<tr>
<th>Measurements</th>
<th>Before Treatment</th>
<th>Changes at Follow Up Appointment</th>
<th>Mean Absolute % Error (MAPE)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean ± SD</td>
<td>Mean ± SE</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sagittal (mm)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ss-OLp</td>
<td>80.4 (5.68)</td>
<td>-0.09 (0.10)</td>
<td>0.64</td>
<td>NS</td>
</tr>
<tr>
<td>Pg-OLp</td>
<td>87.9 (7.83)</td>
<td>0.05 (0.15)</td>
<td>0.94</td>
<td>NS</td>
</tr>
<tr>
<td>Co-Pg</td>
<td>121.7 (7.65)</td>
<td>0.38 (0.23)</td>
<td>1.05</td>
<td>NS</td>
</tr>
<tr>
<td>Is-OLp</td>
<td>89.0 (6.46)</td>
<td>-0.12 (0.13)</td>
<td>0.77</td>
<td>NS</td>
</tr>
<tr>
<td>ii-OLp</td>
<td>85.1 (6.13)</td>
<td>0.52 (0.12)</td>
<td>0.90</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>ms-OLp</td>
<td>50.6 (5.88)</td>
<td>0.01 (0.12)</td>
<td>1.07</td>
<td>NS</td>
</tr>
<tr>
<td>mi-OLp</td>
<td>51.5 (6.58)</td>
<td>0.26 (0.11)</td>
<td>1.74</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Vertical (mm)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>'n-Me'</td>
<td>126.2 (7.47)</td>
<td>0.10 (0.22)</td>
<td>0.93</td>
<td>NS</td>
</tr>
<tr>
<td>S-Go'</td>
<td>85.2 (7.86)</td>
<td>0.15 (0.19)</td>
<td>1.18</td>
<td>NS</td>
</tr>
<tr>
<td>n'-is'</td>
<td>86.2 (5.22)</td>
<td>-0.12 (0.08)</td>
<td>0.67</td>
<td>NS</td>
</tr>
<tr>
<td>ii'-ms'</td>
<td>42.8 (3.76)</td>
<td>-0.12 (0.15)</td>
<td>2.08</td>
<td>NS</td>
</tr>
<tr>
<td>n'-ms'</td>
<td>76.8 (5.38)</td>
<td>-0.14 (0.13)</td>
<td>1.02</td>
<td>NS</td>
</tr>
<tr>
<td>mi'-Mo'</td>
<td>43.8 (3.74)</td>
<td>0.01 (0.12)</td>
<td>1.48</td>
<td>NS</td>
</tr>
<tr>
<td>Angles (degrees)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SNA</td>
<td>81.3 (3.84)</td>
<td>0.08 (0.10)</td>
<td>0.66</td>
<td>NS</td>
</tr>
<tr>
<td>SNB</td>
<td>78.5 (3.69)</td>
<td>0.17 (0.10)</td>
<td>0.69</td>
<td>NS</td>
</tr>
<tr>
<td>ANB</td>
<td>2.8 (3.10)</td>
<td>-0.09 (0.10)</td>
<td>1.46</td>
<td>NS</td>
</tr>
<tr>
<td>MP/SN</td>
<td>32.8 (5.44)</td>
<td>0.01 (0.19)</td>
<td>3.33</td>
<td>NS</td>
</tr>
<tr>
<td>is/SN</td>
<td>99.9 (9.46)</td>
<td>0.06 (0.20)</td>
<td>1.15</td>
<td>NS</td>
</tr>
<tr>
<td>is/PP</td>
<td>106.8 (10.51)</td>
<td>-0.22 (0.20)</td>
<td>1.09</td>
<td>NS</td>
</tr>
<tr>
<td>ii/MP</td>
<td>92.4 (9.07)</td>
<td>0.96 (0.30)</td>
<td>2.19</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>ii/is</td>
<td>134.7 (15.73)</td>
<td>-1.69 (0.58)</td>
<td>2.68</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>ii/OL</td>
<td>73.5 (9.05)</td>
<td>-1.02 (0.34)</td>
<td>3.15</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Soft Tissues (mm)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>H-MP</td>
<td>21.3 (5.96)</td>
<td>0.62 (0.57)</td>
<td>6.63</td>
<td>NS</td>
</tr>
<tr>
<td>C2C4-SN</td>
<td>108.6 (6.84)</td>
<td>0.71 (0.77)</td>
<td>0.50</td>
<td>NS</td>
</tr>
<tr>
<td>PhW-spt</td>
<td>11.3 (3.13)</td>
<td>0.44 (0.35)</td>
<td>7.70</td>
<td>NS</td>
</tr>
<tr>
<td>MinPhW</td>
<td>9.6 (3.05)</td>
<td>-0.01 (0.40)</td>
<td>6.81</td>
<td>NS</td>
</tr>
<tr>
<td>pm-P</td>
<td>42.0 (4.85)</td>
<td>0.05 (0.46)</td>
<td>1.84</td>
<td>NS</td>
</tr>
</tbody>
</table>

NS = Not Significant
4 Recommendations for Future Research
As the treatment of snoring and OSAS patients is considered to be lifelong, the benefit of treatment with MAS must be further evaluated in the light of all possible side effects. Therefore, the change in mandibular forward position has to be carefully controlled and followed up for analysis. To visualize and analyze such possible changes in detail, additional studies using lateral tomography of the TMJs or magnetic resonance imaging would be beneficial. There are no studies of the periodontal factors involved in long term MAS therapy. A study of the gingival and bony support would coordinate well with a study of other intraoral and extraoral parameters.
5 Appendices

5.1 Information Sheet and Consent Form

CENTRE FOR SLEEP DISORDERS AND RESPIRATORY FAILURE

Ground Floor
Prince William Wing
St George Hospital
KOGARAH NSW 2217
Ph: (02) 9350 2696
Fax: 9350 2570

Ethics Approval No. 00/134

ST. GEORGE HOSPITAL, THE UNIVERSITY OF NEW SOUTH WALES
AND THE UNITED DENTAL HOSPITAL, UNIVERSITY OF SYDNEY

Subject Information Statement and Consent Form

LONG TERM FOLLOW UP OF MANDIBULAR ADVANCEMENT (DENTAL) SPLINT THERAPY IN PATIENTS WITH OBSTRUCTIVE SLEEP APNEA

Information: You are invited to participate in a study of the long term effects of wearing a dental splint to treat obstructive sleep apnea. We aim to investigate the ability to wear a dental splint over an extended time frame and to study its' effect on your sleep apnea, snoring and sleep quality. Specifically, we wish to monitor tooth movement, jaw bone and jaw joint changes, gum condition, bite changes and other potential side effects such as excess salivation, dry mouth or discomfort. You have been selected as a possible participant in this study because you have been previously fitted with a dental splint for treatment of obstructive sleep apnea.
**Investigations:** If you decide to participate, we will ask you to fill out a questionnaire and to attend the Centre for Sleep Disorders and Respiratory Failure at St. George Hospital for a dental and radiographic examination. This will be similar to a regular dental check up and will include impressions for study models. You will also have routine dental x-rays. **An appointment will be made to collect all necessary information at the single visit to St. George Hospital. This is estimated to take 1-2 hours.**

**Risks:** All these procedures are safe and carry minimal risk to you.

**Benefits:** Apart from the benefits of a check up of your teeth and dental splint, you will be contributing to our knowledge of the effectiveness of dental splints in the treatment of obstructive sleep apnea. This will improve the delivery of care for all patients suffering the disease.

**Confidentiality:** Any information that is obtained in connection with this study and that can be identified with you will remain confidential. A report of the study may be submitted to medical or dental journals for publication but individuals will not be identifiable.

**Contacts:** Any questions regarding the study can be directed to either Dr. Peter Cistulli at the Sleep Disorders Centre (Ph: 9350 2696) or Dr. Roger Hammond at the United Dental Hospital (Ph: 9293 3388). Your decision whether or not to participate will not prejudice your future relations with the Centre for Sleep Disorders and Respiratory Failure, St. George Hospital. If you decide to participate, you are free to withdraw your consent and to discontinue participation at any time without prejudice.

**Complaints:** Any concerns or complaints may be directed to the Ethics Secretariat, South Eastern Sydney Area Health Service Research Ethics Committee (Southern Section), St. George Hospital, Gray St., Kogarah 2217. Telephone: 9350 2986. Fax: 9350 2988. Email: nhcn@ozemail.com.au

You are making a decision whether or not to participate in this study. Your signature indicates that you have decided to participate, having read the information provided.

Signature of subject: ........................................... ........................................... Date:
Signature of witness: ........................................... ........................................... Date:
Signature of investigator: ........................................... ........................................... Date:
5.2 Patient Questionnaire

CENTRE FOR SLEEP DISORDERS AND RESPIRATORY FAILURE
ST GEORGE HOSPITAL

Long Term Follow up of Mandibular Advancement Splint Therapy on Obstructive Sleep Apnea

SLEEP QUESTIONNAIRE

1  How to answer questionnaire
   • For each question please tick one box only (unless otherwise requested).
   • Some tick boxes have ‘Go to’ instructions that look like ‘Go to 6’. By following the ‘Go to’
     you will skip questions that are not relevant to you.
   • If you have a bed partner, please ask her/him to assist you in answering these questions.
   • If you require any assistance in completing this form, please ask for Dr Roger Hammond at the
     Sleep Disorders Centre, St George Hospital.

Date: ..................................

1  Do you ever wear the dental splint received from the St George Sleep Disorders Centre?
   Yes ............... □ 1
   No ............... □ 2 Go to 22

If Yes: How long have you been using the dental splint?
   6 – 12 months .................................. □ 1
   1 – 2 years .................................... □ 2
   2 – 3 years .................................... □ 3
   More than 3 years ............................. □ 4

2  On average, how many nights each week do you wear the dental splint during sleep?
   1-2 nights .................................... □ 1
   3-4 nights .................................... □ 2
   5-6 nights .................................... □ 3
   7 nights ...................................... □ 4

3  On average, how many hours each night do you wear the dental splint during sleep?
   Less than 2 hours ............................. □ 1
   3-4 hours ..................................... □ 2
   5-6 hours ..................................... □ 3
   More than 6 hours ........................... □ 4

4  Do you ever wake up at night when wearing the dental splint during sleep?
   Yes ............... □ 1
   No ............... □ 2 Go to 7

If Yes: On average, how many nights each week do you wake up when wearing the dental splint during sleep?
   1-2 nights .................................... □ 1
   3-4 nights .................................... □ 2
   5-6 nights .................................... □ 3
   7 nights ...................................... □ 4
5 On average, how many times do you wake up each night when wearing the dental splint during sleep?
   1-2 times .......................................... □ 1
   3-4 times ........................................... □ 2
   More than 4 times .................................. □ 3

6 In your opinion, what has been the MAIN reason for your waking up at night when wearing the dental splint during sleep?
   Going to toilet .................................. □ 1
   Jaw discomfort ................................... □ 2
   Pain in the jaw joint .............................. □ 3
   Tooth tenderness ................................. □ 4
   Excess salivation ................................. □ 5
   Dry mouth ......................................... □ 6
   Grinding of teeth at night ...................... □ 7
   Gum irritation .................................... □ 8
   Loose dental splint .............................. □ 9
   Other (please specify) ...........................  □ 10

7 Do you ever stop breathing at night when wearing the dental splint during sleep?
   Yes ............. □ 1
   No ............... □ 2
   Don't know □ 3

8 Do you ever experience choking at night when wearing the dental splint during sleep?
   Yes ............. □ 1
   No ............... □ 2

9 Do you ever snore at night when wearing the dental splint during sleep?
   Yes ............. □ 1
   No ............... □ 2 ➔ Go to 11
   Don't know □ 3 ➔ Go to 11

If Yes: On average, how many nights each week do you snore when wearing the dental splint during sleep?
   1-2 nights ...................................... □ 1
   3-4 nights ...................................... □ 2
   5-6 nights ...................................... □ 3
   7 nights ........................................ □ 4

10 Overall, how would you rate the intensity of your snoring at night when wearing the dental splint during sleep?
   High intensity .................................. □ 1
   Medium intensity ............................... □ 2
   Low intensity ................................... □ 3

11 Overall, how would you rate the quality of your sleep at night when wearing the dental splint during sleep?
   Very refreshing .................................. □ 1
   Moderately refreshing ........................ □ 2
   Slightly refreshing ............................. □ 3
   Not refreshing ................................... □ 4
12 Overall, how would you rate your level of tiredness during the day after wearing the dental splint at night during sleep?

Very tired ........................................... □ 1
Moderately tired .................................... □ 2
Slightly tired □ 3
Not tired .................................................. □ 4

13 How likely are you to doze off or fall asleep in the following situations, in contrast to feeling just tired?

-... This refers to your usual way of life in recent times
-... Even if you haven't done some of these things recently, try to work out how they would have affected you

-... Use the following scale to choose the **most appropriate number** for each situation:

0 = never doze
1 = slight chance of dozing
2 = moderate chance of dozing
3 = high chance of dozing

*It is important that you put a number (0 to 3) in each of the 8 boxes*

<table>
<thead>
<tr>
<th>Situation</th>
<th>Chance of dozing (0-3)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sitting and reading</td>
<td>□</td>
</tr>
<tr>
<td>Watching television</td>
<td>□</td>
</tr>
<tr>
<td>Sitting, inactive in a public place (eg in a theatre or a meeting)</td>
<td>□</td>
</tr>
<tr>
<td>As a passenger in a car for an hour without a break</td>
<td>□</td>
</tr>
<tr>
<td>Lying down to rest in the afternoon when circumstances permit</td>
<td>□</td>
</tr>
<tr>
<td>Sitting and talking to someone</td>
<td>□</td>
</tr>
<tr>
<td>Sitting quietly after lunch without alcohol</td>
<td>□</td>
</tr>
<tr>
<td>In a car, while stopped for a few minutes in the traffic</td>
<td>□</td>
</tr>
</tbody>
</table>
14 Which of the following side effects do you experience when wearing the dental splint during sleep?

<table>
<thead>
<tr>
<th>Side Effect</th>
<th>Severity</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>(a) Jaw discomfort</em></td>
<td>If yes, specify severity</td>
<td>If yes, specify frequency</td>
</tr>
<tr>
<td>Yes ☐ 1</td>
<td>Mild.............☐ 1</td>
<td>Rarely.........☐ 1</td>
</tr>
<tr>
<td>No ☐ 2 (If No go to (b))</td>
<td>Moderate........☐ 2</td>
<td>Sometimes.....☐ 2</td>
</tr>
<tr>
<td></td>
<td>Severe...........☐ 3</td>
<td>Often..........☐ 3</td>
</tr>
<tr>
<td><em>(b) Pain in the jaw joint area</em></td>
<td>If yes, specify severity</td>
<td>If yes, specify frequency</td>
</tr>
<tr>
<td>Yes ☐ 1</td>
<td>Mild.............☐ 1</td>
<td>Rarely.........☐ 1</td>
</tr>
<tr>
<td>No ☐ 2 (If No go to (c))</td>
<td>Moderate........☐ 2</td>
<td>Sometimes.....☐ 2</td>
</tr>
<tr>
<td></td>
<td>Severe...........☐ 3</td>
<td>Often..........☐ 3</td>
</tr>
<tr>
<td><em>(c) Tooth tenderness</em></td>
<td>If yes, specify severity</td>
<td>If yes, specify frequency</td>
</tr>
<tr>
<td>Yes ☐ 1</td>
<td>Mild.............☐ 1</td>
<td>Rarely.........☐ 1</td>
</tr>
<tr>
<td>No ☐ 2 (If No go to (d))</td>
<td>Moderate........☐ 2</td>
<td>Sometimes.....☐ 2</td>
</tr>
<tr>
<td></td>
<td>Severe...........☐ 3</td>
<td>Often..........☐ 3</td>
</tr>
<tr>
<td><em>(d) Excess salivation</em></td>
<td>If yes, specify severity</td>
<td>If yes, specify frequency</td>
</tr>
<tr>
<td>Yes ☐ 1</td>
<td>Mild.............☐ 1</td>
<td>Rarely.........☐ 1</td>
</tr>
<tr>
<td>No ☐ 2 (If No go to (e))</td>
<td>Moderate........☐ 2</td>
<td>Sometimes.....☐ 2</td>
</tr>
<tr>
<td></td>
<td>Severe...........☐ 3</td>
<td>Often..........☐ 3</td>
</tr>
<tr>
<td><em>(e) Dry mouth</em></td>
<td>If yes, specify severity</td>
<td>If yes, specify frequency</td>
</tr>
<tr>
<td>Yes ☐ 1</td>
<td>Mild.............☐ 1</td>
<td>Rarely.........☐ 1</td>
</tr>
<tr>
<td>No ☐ 2 (If No go to (f))</td>
<td>Moderate........☐ 2</td>
<td>Sometimes.....☐ 2</td>
</tr>
<tr>
<td></td>
<td>Severe...........☐ 3</td>
<td>Often..........☐ 3</td>
</tr>
<tr>
<td><em>(f) Grinding of teeth at night</em></td>
<td>If yes, specify severity</td>
<td>If yes, specify frequency</td>
</tr>
<tr>
<td>Yes ☐ 1</td>
<td>Mild.............☐ 1</td>
<td>Rarely.........☐ 1</td>
</tr>
<tr>
<td>No ☐ 2 (If No go to (g))</td>
<td>Moderate........☐ 2</td>
<td>Sometimes.....☐ 2</td>
</tr>
<tr>
<td></td>
<td>Severe...........☐ 3</td>
<td>Often..........☐ 3</td>
</tr>
<tr>
<td><em>(g) Gum irritation</em></td>
<td>If yes, specify severity</td>
<td>If yes, specify frequency</td>
</tr>
<tr>
<td>Yes ☐ 1</td>
<td>Mild.............☐ 1</td>
<td>Rarely.........☐ 1</td>
</tr>
<tr>
<td>No ☐ 2 (If No go to (h))</td>
<td>Moderate........☐ 2</td>
<td>Sometimes.....☐ 2</td>
</tr>
<tr>
<td></td>
<td>Severe...........☐ 3</td>
<td>Often..........☐ 3</td>
</tr>
<tr>
<td><em>(h) Jaw joint noises</em></td>
<td>If yes, specify severity</td>
<td>If yes, specify frequency</td>
</tr>
<tr>
<td>Yes ☐ 1</td>
<td>Mild.............☐ 1</td>
<td>Rarely.........☐ 1</td>
</tr>
<tr>
<td>No ☐ 2 (If No go to (i))</td>
<td>Moderate........☐ 2</td>
<td>Sometimes.....☐ 2</td>
</tr>
<tr>
<td></td>
<td>Severe...........☐ 3</td>
<td>Often..........☐ 3</td>
</tr>
<tr>
<td><em>(j) Headaches</em></td>
<td>If yes, specify severity</td>
<td>If yes, specify frequency</td>
</tr>
<tr>
<td>Yes ☐ 1</td>
<td>Mild.............☐ 1</td>
<td>Rarely.........☐ 1</td>
</tr>
<tr>
<td>No ☐ 2 (If No go to 16)</td>
<td>Moderate........☐ 2</td>
<td>Sometimes.....☐ 2</td>
</tr>
<tr>
<td></td>
<td>Severe...........☐ 3</td>
<td>Often..........☐ 3</td>
</tr>
</tbody>
</table>

Please list any other side effects (if any) not mentioned above:

..................................................................................
15 Over the period that you have been wearing the dental splint, on average, how would you rate the CHANGE in the following (Please tick appropriate column):

<table>
<thead>
<tr>
<th>Changes Affecting</th>
<th>Better</th>
<th>No Change</th>
<th>Worse</th>
<th>Never a Problem</th>
</tr>
</thead>
<tbody>
<tr>
<td>Snoring</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Daytime sleepiness</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>The way your teeth bite together</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Jaw discomfort</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pain in the jaw joint area</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tooth tenderness</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Excess salivation</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dry mouth</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Grinding of teeth at night</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gum irritation</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Jaw joint noises</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Headaches</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fit of the dental splint</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

16 During the time that you have been wearing the dental splint, has it required replacement?
Yes ................. □ 1
No ................ □ 2 ➔ Go to 18

If yes: How often has it required replacement?
Once ................................................ □ 1
Twice .............................................. □ 2
3 times ......................................... □ 3
More than 3 times ............................. □ 4

17 Which of the following was the reason for the replacement of the dental splint?
Broken ............................................ □ 1
Lost ................................................. □ 2
Fit of the dental splint ..................... □ 3
Other (please specify) □ 4
........................................................................................................

18 Do you and your bed partner sleep in the same room when wearing the dental splint at night during sleep?
Yes ................................ □ 1 ➔ Go to 19
No ........................................ □ 2
Not Applicable ............................... □ 3 ➔ Go to 19

If No: What is the main reason for not sleeping in the same room?
Snoring ........................................... □ 1
Other than snoring ............................ □ 2

19 Overall, would you rate the dental splint as being very satisfactory, satisfactory or not satisfactory in controlling your sleep apnea symptoms?
Very satisfactory ................................ □ 1
Satisfactory ...................................... □ 2
Not satisfactory .............................. □ 3

20 Will you continue to wear the dental splint at night during sleep?
Yes ................. □ 1
No ................ □ 2

If No: Please specify reasons:
21 Did your bed partner assist you in answering these questions?
   Yes ........................................... □ 1
   No................................................ □ 2
   Not Applicable ....................... □ 3

If you HAVE been wearing the dental splint you received from the St George Sleep Disorders Centre, you DO NOT need to answer any further questions

22 How long after being issued the dental splint did you stop wearing it?
   Less than 1 month.......................... □ 1
   Between 1 and 3 months ................. □ 2
   Between 3 and 6 months ................. □ 3
   Between 6 and 12 months .............. □ 4
   More than 12 months .................. □ 5

23 In your opinion, what has been the MAIN reason for not wearing the dental splint at night during sleep?
   Jaw discomfort ............................... □ 1
   Pain in the jaw joint ...................... □ 2
   Tooth tenderness ........................... □ 3
   Excess salivation .......................... □ 4
   Dry mouth... ................................. □ 5
   Grinding of teeth at night............ □ 6
   Gum irritation ............................... □ 7
   Loose appliance ............................ □ 8
   Lost appliance .............................. □ 9
   Broken appliance ......................... □ 10
   Other (please specify) ............. □ 11

THANK YOU FOR COMPLETING THIS QUESTIONNAIRE
5.3 Orthodontic History and Examination Sheet

**General:**
Date : ____________  
Patient No : ____________

Name : ____________  
Weight : ____________

Date of Birth : ____________  
Height : ____________

Neck Circumference: ____________  
BMI : ____________

**Medical History:**
Asthma : ____________  
Tonsils/Adenoids: ____________

Medication : ____________  
Allergies : ____________

Respiratory Mode:  
Mouth .  
Nose .

Previous/Current OSA Tx: 


**Dental History:**
Any appliance treatment prior to current splint:  
No .  
Yes .

If yes, for how long and its' success: ____________________________

Jaw surgery:  
No .  
Yes .

Bruxism:  
No .  
Yes .

Last dental visit and treatment: ____________________________
**Extra-Oral Examination:**

Frontal Profile  
Symmetry and shape: Skeletal Base:  
Vertical Thirds: Vertical Thirds:  
Lips: Lips:  
Nose: Nose:  

**Intra-Oral Examination:**

<table>
<thead>
<tr>
<th>Oral Hygiene:</th>
<th>Teeth present:</th>
<th>87654321 / 12345678</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>87654321 / 12345678</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>CPITN:</th>
<th>Caries:</th>
<th>87654321 / 12345678</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>87654321 / 12345678</td>
<td></td>
</tr>
</tbody>
</table>

Soft Tissue Abnormalities: Restorations:  
Periodontium: Tongue:  

<table>
<thead>
<tr>
<th>Periodontium:</th>
<th>Tongue:</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>RHS</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Pocketing:</th>
<th>Molars</th>
<th>:</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>Overbite:</th>
<th>Canines</th>
<th>:</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>Crossbites:</th>
<th>Overjet:</th>
</tr>
</thead>
</table>

Headaches before splint treatment: No . Yes .  
Headaches during splint treatment: No . Yes .  
Headaches after splint treatment: No . Yes .  
Range of motion: Maximum opening: _____________ mm  
Right excursion: _____________ mm  
Left excursion: _____________ mm  
Protrusion: _____________ mm  
Deviation on opening:  
CR/CO discrepancy: _____________ mm  
Exaggerated Gag reflex: No . Yes .
**Splint Evaluation:**

Evidence of wear: No . Yes .

Fit of splint: ____________________________

With splint in place:

- MAS Pro: ________ mm
- Mx MAS Pro: ________ mm
- Vertical opening: ________ mm

**SLEEP ASSESSMENT**

<table>
<thead>
<tr>
<th></th>
<th>T1</th>
<th>T2</th>
<th>T3</th>
</tr>
</thead>
<tbody>
<tr>
<td>RDI</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MinSaO₂</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
5.4 TMJ Assessment Sheet  (Helkimo, 1974a)

Clinical dysfunction index $D_i$:

**Impaired range of movement**

<table>
<thead>
<tr>
<th>Description</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal range of movement</td>
<td>0</td>
</tr>
<tr>
<td>Slightly impaired mobility</td>
<td>1</td>
</tr>
<tr>
<td>Severely impaired mobility</td>
<td>5</td>
</tr>
</tbody>
</table>

**Impaired TMJ function**

<table>
<thead>
<tr>
<th>Description</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Smooth movement, no sound, deviation $\leq$ 2mm</td>
<td>0</td>
</tr>
<tr>
<td>TMJ sounds in 1 or 2 joints, deviation $\geq$ 2mm</td>
<td>1</td>
</tr>
<tr>
<td>Locking or luxation of TMJ</td>
<td>5</td>
</tr>
</tbody>
</table>

**Muscle pain**

<table>
<thead>
<tr>
<th>Description</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>No tenderness to palpation in masticatory muscles</td>
<td>0</td>
</tr>
<tr>
<td>Tenderness to palpation in 1 – 3 palpation sites</td>
<td>1</td>
</tr>
<tr>
<td>Tenderness to palpation in 4 or more palpation sites</td>
<td>5</td>
</tr>
</tbody>
</table>

**TMJ pain**

<table>
<thead>
<tr>
<th>Description</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>No tenderness to palpation</td>
<td>0</td>
</tr>
<tr>
<td>Tenderness to palpation laterally</td>
<td>1</td>
</tr>
<tr>
<td>Tenderness to palpation posteriorly</td>
<td>5</td>
</tr>
</tbody>
</table>

**Pain on movement of the mandible**

<table>
<thead>
<tr>
<th>Description</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>No pain on movement</td>
<td>0</td>
</tr>
<tr>
<td>Pain on 1 movement</td>
<td>1</td>
</tr>
<tr>
<td>Pain on 2 or more movements</td>
<td>5</td>
</tr>
</tbody>
</table>

---

**Clinical Dysfunction Index Score**  = 

<table>
<thead>
<tr>
<th>Code</th>
<th>Description</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>clinically symptom free</td>
<td>$D_{i0}$</td>
</tr>
<tr>
<td>1–4</td>
<td>mild dysfunction</td>
<td>$D_{iI}$</td>
</tr>
<tr>
<td>5–9</td>
<td>moderate dysfunction</td>
<td>$D_{iII}$</td>
</tr>
<tr>
<td>10–25</td>
<td>severe dysfunction</td>
<td>$D_{iIII}$</td>
</tr>
</tbody>
</table>
Anamnestic dysfunction index $A_i$:

$A_{i0} = $ symptom free

$A_{i1} = $ mild symptoms—TMJ sounds, jaw fatigue, stiffness on waking or movements

$A_{iII} = $ severe symptoms—locking, luxation, pain on movement, in TMJ, in muscles

Anamnestic dysfunction index $= \underline{\hspace{2cm}}$
Index for occlusal state $O_i$:

**Number of teeth**

- 28 – 32 teeth: 0
- 20 – 27 teeth: 1
- < 20 teeth: 5

**Number of occluding teeth**

- 24 – 32 occluding teeth: 0
- 16 – 23 occluding teeth: 1
- 2 – 15 occluding teeth: 5

**Occlusal interferences between CR and CO**

- No interferences: CR to CO < 2mm: 0
- Mild interferences:
  - unilateral contact in CR (CR – CO < 2mm) and/or lateral deviation < 0.5mm during CR to CO: 1
- Severe interferences:
  - lateral deviation > 0.5mm during CR to CO
  - Cr to CO > 2mm: 5

**Articulation interferences**

- No disturbances in articulation: 0
- Mild interferences:
  - laterotrusive interferences distally to 3 and/or unilateral contact on protrusion: 1
- Severe interferences:
  - Uni or bilateral mediotrusive interferences: 5

---

Index for occlusal state score = __________

**Code:**

- 0 = no occlusal disturbance = $O_{i0}$
- 1 – 4 = moderate occlusal disturbance = $O_{iI}$
- 5 – 20 = severe occlusal disturbance = $O_{iII}$