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MAXILLARY MORPHOLOGY
IN PATIENTS WITH OBSTRUCTIVE
SLEEP APNOEA

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

by

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August, 1996
This thesis is dedicated to my family.

Kin, my loving wife, for her unfailing love, support and encouragement.

Wei-Chien and Wei-Lyn, my children, for their understanding and patience.
ACKNOWLEDGEMENTS

When the seeds for the concept of this thesis were sown, it was clear that the “journey” would not be plain sailing. The culmination of this thesis would not have been possible without expert guidance, unfailing support, stimulating discussion and sometimes even gentle prodding along the way.

I am grateful to my immediate supervisor, Dr. Peter Cistulli, Director of the Sleep Disorders Centre at St. George Hospital for his expert guidance, encouragement and advice right through this project. He gave me an opportunity to glimpse into the world of scientific research. The stimulating discussions we had have broadened my knowledge and made me think more laterally.

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SIGNED STATEMENT

This thesis contains no material which has been accepted for the award of any other degree or diploma in any university and that, to the best of the candidate's knowledge and belief, the thesis contains no material previously published or written by another person, except where due reference is made in the text of the thesis.

..............................................................

Boon Hong Seto
August 1996
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<th>Definition</th>
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</thead>
<tbody>
<tr>
<td>ADA</td>
<td>American dental association</td>
</tr>
<tr>
<td>AHI</td>
<td>Apnoea hypopnoea index</td>
</tr>
<tr>
<td>BMI</td>
<td>Body mass index</td>
</tr>
<tr>
<td>CPAP</td>
<td>Continuous positive airway pressure</td>
</tr>
<tr>
<td>ECG</td>
<td>Electrocardiogram</td>
</tr>
<tr>
<td>EDS</td>
<td>Excessive daytime sleepiness</td>
</tr>
<tr>
<td>EEG</td>
<td>Electroencephalogram</td>
</tr>
<tr>
<td>EMG</td>
<td>Electromyogram</td>
</tr>
<tr>
<td>EOG</td>
<td>Electro-oculogram</td>
</tr>
<tr>
<td>ESS</td>
<td>Epworth sleepiness scale</td>
</tr>
<tr>
<td>GAHM</td>
<td>Genioglossal advancement with hyoid myotomy and suspension</td>
</tr>
<tr>
<td>ICD</td>
<td>Inter-canine distance</td>
</tr>
<tr>
<td>IMD</td>
<td>Inter-molar distance</td>
</tr>
<tr>
<td>IPD</td>
<td>Inter-premolar distance</td>
</tr>
<tr>
<td>MinSaO₂</td>
<td>Minimum saturation of oxygen</td>
</tr>
<tr>
<td>OSA</td>
<td>Obstructive sleep apnoea</td>
</tr>
<tr>
<td>PAS</td>
<td>Posterior airway space</td>
</tr>
<tr>
<td>RDI</td>
<td>Respiratory disturbance index</td>
</tr>
<tr>
<td>UPPP</td>
<td>Uvuloplatopharyngoplasty</td>
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</tbody>
</table>
ABSTRACT

The purpose of this study was to test the hypothesis that patients with obstructive sleep apnoea (OSA) have different maxillary morphology compared to non-snoring and non-apnoeic controls, and that the shape and dimension of the maxilla is important in the development of OSA. The sample consisted of 40 randomly chosen adult OSA patients confirmed by polysomnography and 21 adult control subjects. These control subjects were selected with validated questionnaires to exclude the two most common symptoms of OSA, namely heavy snoring and excessive day time sleepiness. Patients and controls underwent orthodontic assessment, anthropometric measurements, dental impressions, and lateral and postero-anterior cephalograms. Inter-tooth distances, palate heights, and maxillary depths were measured directly from the dental casts. All cephalograms were hand traced and analysed by the author. Maxillary archforms were described using 4th order polynomial equations. Measurement errors, checked by double determination, were insignificant.

The results showed that 50% of the OSA patients had posterior crossbites in contrast to 5% of the controls. These patients also had a significantly shorter (p<0.05), and narrower (p<0.005) maxillary skeletal base. In addition, patients’ archforms were more tapered and significantly narrower at the canine (p<0.005), premolar (p<0.005) and molar (p<0.05) regions. There were no significant differences in the palate heights at the above regions, but the palatal indices at the 2nd premolar (p<0.05) and molar (p<0.05) regions were significantly different, suggesting a difference in shape in the posterior part of the palate. Univariate regression analysis showed significant correlations between the arch dimensions and posterior airway space at the canine (r=0.37, p=0.04), premolar (r=0.41, p=0.02) and molar (r=0.35, p=0.04) regions in the OSA patients. These correlations suggest a possible link between maxillary constriction, tongue posture and posterior airway space. No correlation was found between maxillary arch dimensions and apnoea severity in this study. Nevertheless, the investigation clearly demonstrated that maxillary skeletal constriction is a common feature in OSA patients, and that patients have differently shaped palates with narrower, shorter and more tapered archforms than controls. The relationship between maxillary morphology and OSA is clearly complex. The exact role of maxillary constriction and its interaction with tongue posture and nasal resistance in the pathogenesis of OSA requires further exploration.
1 LITERATURE REVIEW

1.1 INTRODUCTION

Obstructive sleep apnoea (OSA) is increasingly being recognised as a major public health problem (Phillipson, 1993). It is a common disorder affecting approximately 4% of men and 2% of women in their middle age (Young et al., 1993). The condition is characterised by repetitive, complete, or partial upper airway collapse during sleep, resulting in oxygen desaturation and sleep fragmentation. The range of symptoms and sequela are wide and varied and are a source of significant morbidity and mortality (Partinen and Guilleminault, 1990; Klitzman and Miller, 1994).

The precise cause of OSA and its precursor, snoring, is still not known. Numerous studies have demonstrated that, as a group, patients with OSA have abnormally narrow upper airways compared to control subjects (Rivlin et al., 1984; Bradley et al., 1986; Schwab et al., 1993). It is thought that this narrowing and the normal physiological loss of muscle tone during the onset of sleep are important factors in the development of snoring and OSA. In addition, many cephalometric studies have revealed that patients with OSA have a range of craniofacial abnormalities and that several of these measurements correlate with the degree of OSA (Riley et al., 1983; Partinen et al., 1988; Pepin et al., 1992). The correlation between these craniofacial abnormalities and apnoea severity further suggests that structural abnormalities are likely to play a role in the pathophysiology of OSA.

Recent work indicated that maxillary constriction could be one such abnormality. In a study of Marfan's syndrome (Cistulli, 1994), the findings demonstrated that maxillary constriction, which is part of the syndrome, may account for the reported high prevalence of OSA in this group of patients. In addition, a recent pilot study (Palmisano et al., 1994) suggested that maxillary constriction may be a common feature in the "typical" OSA population. Therefore,
the purpose of this thesis was to test the hypothesis that patients with OSA have different maxillary morphology compared to non-snoring, non-apnoeic controls and that maxillary constriction is an important factor in the development of OSA.

This literature review provides the background knowledge of OSA, its pathophysiology and its relationship to craniofacial abnormalities. Upper airway obstruction and facial development was thought to be relevant because some studies have suggested a genetic basis for OSA (Strohl et al., 1978; Wittig et al., 1988; Redline et al., 1992) with the inference that OSA may evolve during childhood before becoming clinically obvious later in life (Guilleminault et al., 1989). If so, the possibility of preventive treatment is there to be explored and developed as there is currently no curative treatment for OSA.

1.2 AN OVERVIEW OF SLEEP APNOEA

1.2.1 An historical perspective

Although most of the manifestations of sleep apnoea have been described for many years, a wider recognition of the pathophysiology and clinical features has occurred only in the last decade. Two main reasons for overlooking the sleep apnoea syndrome for so long have been the misdiagnosis of patients with the syndrome and scepticism regarding the validity of excessive somnolence as a clinical sign (Lavie, 1984).

It is generally believed that the first description of sleep apnoea was made by Charles Dickens (1837) in his novel the “Posthumous Papers of the Pickwick Club”. He described an extremely fat boy named Joe who suffered from hypsomnolence; hence the term “Pickwickian” Syndrome (Kryger 1983; Lavie 1984).
In 1889, William Hill also recognised this syndrome and wrote "The stupid looking lazy kid who frequently suffers from headaches at school, breathes through his mouth instead of his nose, snores and is restless at night, wakes up with a dry mouth in the morning, is well worthy of the attention of the school medical officer."

William Osler (1906) made the syndrome more famous when he stated "An extraordinary phenomenon in excessively fat young persons is an uncontrollable tendency to sleep like the fat boy in Pickwick." However, no link between excessive somnolence to nocturnal sleep disturbance was appreciated.

Gastaut and associates (1965) were the first contemporary investigators to show repeated apnoeas in the sleep of Pickwickian patients. This disturbed sleep pattern was hypothesised to be responsible for the daytime hypersomnolence. Sleep apnoea, as a syndrome, was first described by Guilleminault, Eldrige and Dement (1973). They were among the first investigators to recognise the full importance and frequency of these apnoeas during sleep.

Prior to 1981, tracheostomy was the only effective treatment. Then in 1981, Fujita et al. introduced uvulopalatopharyngoplasty (UPPP) for the treatment of snoring and OSA. This surgical approach has not enjoyed success, and there are data suggesting an appreciable morbidity and mortality rate in patients treated with UPPP (Larson et al., 1991; Rodenstein, 1992). During the same year, an Australian and his co-workers, Sullivan et al. (1981) developed a highly effective non-surgical treatment for obstructive sleep apnoea. This procedure, which remains the gold standard of treatment today, involved the production of a continuous stream of positive air pressure applied through the nose during sleep. The positive pressure acts as a splint maintaining the patency of the airway and hence preventing it from closing. This treatment is known as nasal continuous positive airway pressure (nasal CPAP).
1.2.2 Definition and diagnosis of sleep apnoea

An Apnoea is defined as the cessation of airflow lasting at least ten seconds. Obstructive sleep apnoea has been defined as the presence of at least 30 apnoeic episodes observed in both rapid eye movement, or non-rapid eye movement sleep, during seven hours of nocturnal sleep (Guilleminault and Dement, 1978).

The number of episodes of apnoea per hour of sleep is termed the Apnoeic Index (Guilleminault and Dement, 1978). These authors also proposed that an Apnoeic Index ≥ 5 as a more sensitive threshold for the diagnosis of OSA.

Hypopnoea, or reduction in breathing, is when a 50% or greater reduction in airflow occurs simultaneously with a 4% or greater reduction in blood oxygen saturation (Wynne et al., 1979; Guilleminault, Cummiskey and Motta, 1980). The number of episodes of apnoea and hypopnoea per hour is called the Apnoea Hypopnoea index (AHI) or the Respiratory Disturbance Index (RDI).

There are three forms of sleep apnoea; central, obstructive and mixed. Central apnoea is characterised by the cessation of both airflow and respiratory movements. In obstructive sleep apnoea, there is breathing effort and movements with the absence of air passage. A mixture of these two conditions is described as mixed sleep apnoea (Guilleminault and Dement, 1978).

A precise definition of sleep apnoea has not yet been established. Opinion has moved away from the early rigid definitions which, although initially useful for research in the field, excluded many patients with disabling symptoms (Royal College of Physicians of London, 1993). In the same report, it was pointed out that there is uncertainty over the type of abnormality of nocturnal breathing which must be present for the daytime features to be manifest. Complete apnoea was recognised as the event that led to arousal. Subsequently, it was shown that hypopnoea alone could cause arousal (Gould, Whyte and Rhind, 1988), and
recently heavy snoring has also been found to produce sleep disruption even in the absence of apnoea, hypopnoea or hypoxaemia (Guilleminault, Stooohs and Duncan, 1991; Stradling, Crosby and Payne, 1991).

Guilleminault et al. (1992) described patients who do not snore but present with clinical symptoms of daytime sleepiness and fatigue. He called this "Upper airway resistance syndrome". Sometimes excessive daytime sleepiness may not occur. Instead, the clinical picture may mimic an anxiety state, especially in women (Ambrogetti, Olsen and Saunders, 1991).

Table 1.1 Symptoms of obstructive sleep apnoea

<table>
<thead>
<tr>
<th>Major symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Loud snoring</td>
</tr>
<tr>
<td>Excessive daytime sleepiness</td>
</tr>
<tr>
<td>Restless sleep</td>
</tr>
<tr>
<td>Unrefreshing sleep</td>
</tr>
<tr>
<td>Nocturia</td>
</tr>
<tr>
<td>Apparent personality changes</td>
</tr>
<tr>
<td>Witnessed apnoeas</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Minor symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Choking or shortness of breath sensations at night</td>
</tr>
<tr>
<td>Reduced libido</td>
</tr>
<tr>
<td>Nocturnal sweating</td>
</tr>
<tr>
<td>Morning headaches</td>
</tr>
</tbody>
</table>


The definition of abnormal breathing in children is more difficult. Clinical apnoea in infants has been defined as a pause in breathing of more than 20 seconds duration or a briefer pause associated with bradycardia (Henderson-Smart and Cohen, 1986). Adult criteria for OSA may not be appropriate for children (Rosen, D’Andrea, and Haddad, 1992). Norms for a healthy elderly population are scant and the associated risks and survival rates of having a
particular Apnoea Hypopnoea Index are not well characterised. For example, the individual with one or two apnoeas per hour, oxygen desaturation to 60-70%, and impaired arousal reflexes due to autonomic neuropathy, is far more vulnerable to the consequences of their OSA than a healthy, asymptomatic 75 year old with 15 apnoeas per hour (Guilleminault et al., 1977). It is important then that the clinical definition of OSA should not be too rigid. Currently, under ideal circumstances, a full sleep study is the most appropriate investigation for assessing and diagnosing OSA.

1.2.3 Prevalence

Epidemiological studies on OSA are conducted in three ways:

1) Questionnaire data about habitual snoring, or a history of witnessed apnoeas, or both.

2) Studies in which questionnaires are validated by full polysomnographic sleep studies or nocturnal respiration monitoring in a random or selected subpopulation.

3) Studies where all or most patients undergo full sleep studies or nocturnal respiratory monitoring.

In the past five years, studies of the latter two types have shown that OSA occurs commonly, but the reported prevalences have a wide range, varying from 1% to 9% (Gislason et al., 1988; Cirignotta et al., 1989; Bearpark et al., 1991; Young, Zaccaro and Leder, 1991; Jennum and Soul, 1992). A recent study (Young et al., 1993), suggests a prevalence of approximately 4% in middle-aged men and 2% in women based on the criteria of the Apnoea Hypopnoea index (AHI) > 5 with symptom of excessive day time sleepiness (Appendix 1).

The range of differences in the prevalence of OSA is probably due to the disparity in the methodologies, diagnostic criteria and the specific subgroups of patients studied. Further variables to be considered include population differences in obesity and alcohol consumption, or even genetic variability. For example, the percentage of the population with heavy snoring, doubles when the bed partner contributes to the questionnaire (Stradling and Crosby, 1991). Berry et al. (1984)
found that the false-positive rates (Apnoea Index > 5 in controls) are not very high in younger healthy subjects. However, a dramatic increase in false-positives takes place in samples using large numbers of subjects past 60 years of age.

Ancoli-Israel et al. in 1986 reported that approximately 25% of persons over 65 years of age have an Apnoea Index > 5, but concluded that it does not mean that 25% have OSA syndrome.

Hence, although OSA is undoubtedly common, its exact prevalence in the general population remains uncertain. This is due to the lack of population based epidemiological research with many of the previous studies focusing on specific subgroups. Furthermore, assessment of symptoms is often made difficult by the fact that the patient with OSA is often the last person to realise the extent of the effects of the disorder.

1.2.4 Pathophysiology of Sleep Apnoea

Predisposing factors of OSA such as male sex, middle age, familial tendency, alcohol consumption, obesity, endocrine and metabolic disorders will not be described here as they are outside the scope of this thesis.

The characteristic event in OSA is repetitive occlusion of the airway during sleep. The patency of the airway at any time is influenced by the balance of the forces generated by the dilator muscles of the airway on the one hand and the forces of inspiration that tend to occlude the airway on the other. This “Balance of forces” concept (Cistulli and Sullivan, 1994) is depicted in Figure 1.1.

Normally the upper airway remains patent because the dilating force exerted by the upper airway muscles exceeds the subatmospheric intraluminal pressure generated during inspiration (Remmers et al., 1978; Surrat et al., 1985; Howell et al., 1989). The patency of the airway is also affected by the lack of co-ordination between the dilator muscles and respiratory pump muscles, particularly the diaphragm. Usually, the activation of the dilators precedes that of the respiratory
pump muscles thereby counterbalancing the collapsing force exerted by the respiratory pump muscles.

Figure 1.1, shows that the narrower the airway, the more the predisposition to closure, and the greater the dependence on the upper airway dilator muscle activity to maintain patency. This is because greater subatmospheric inspiratory pressure is generated in an attempt to achieve adequate airflow through the narrow airway (Cistulli and Sullivan, 1994).

**Figure 1.1** Balance of forces that sustain upper airway patency. The two major forces are airway suction pressure and upper airway dilator tone, and these are in turn influenced by other factors. Adapted from Cistulli PA, Sullivan CE, Pathophysiology of sleep apnoea, In: Saunders NA, Sullivan CE (Ed). Sleep and Breathing. Marcel Dekker Inc, New York, 1994, pp. 405-488.

Some studies have suggested that the narrowing is due to fat deposition around and particularly lateral to the upper airways (Honer et al., 1989; Shelton et al., 1993a). Others have proposed micrognathia and retrognathia (Conway, Bower...
and Barnes, 1977), adenotonsillar hypertrophy (Orr and Martin, 1981),
macroglossia associated with hypothyroidism (Rajagopal et al., 1984) and
acromegaly (Perks et al., 1980) as the underlying structural abnormalities.

Studies using sophisticated imaging techniques such as fluoroscopy
(Suratt et al., 1983), acoustic reflection (Rilvin et al., 1984), cephalometric
radiographs (Riley et al., 1983), computed tomograms (Haponick et al., 1983; Lowe
et al., 1986) and magnetic resonance imaging (Abbey et al., 1989) have identified
more subtle structural abnormalities of the upper airway. It was found that patients
with OSA as a group, have a narrower upper airway when compared to normal
controls (Rivlin et al., 1984; Bradley et al., 1986; Pepin et al., 1992; Schwab et al.,
1993). However, one must be cautious in interpreting these results because most
of these studies were performed during wakefulness and in the erect rather than
supine posture. A study by Yildirim et al. (1991) has shown that posture is an
important determinant of upper airway dimensions.

Patients with OSA also have increased activation of their upper airway
muscles when awake (Mezzanotte, Tangel and White, 1992) which may reflect the
need to keep their narrowed upper airway patent. This is clearly demonstrated
when it is reported that genioglossus EMG activity during wakefulness reduces
significantly in OSA patients following the application of 5 cm H₂O of continuous
positive airway pressure, whereas in normal controls there is only a small reduction
(White and Mezzanotte 1993). Similarly, White and Ballard (1990) found that
patients with sleep apnoea had considerable phasic inspiratory-linked
electromyographic activity in the genioglossus, both awake and asleep, whereas
little or no activity could be found in normal subjects.

These studies support the theory that the underlying cause of sleep apnoea
is airway narrowing, and when awake, these patients must have more activation of
the upper airway dilator muscles simply to keep the airway open. Hence, the
occurrence of upper airway obstruction during sleep is the result of a normal
physiological loss of muscle tone with sleep onset and the dominant pathological
element is a mechanical narrowing of the upper airway.
Furthermore, any narrowing of the nose or nasopharynx would also require extra effort to breathe and, consequently, increase the negative upper airway pressure during inspiration, predisposing to upper airway collapse (Kuna and Remmers, 1985).

In summary, the development of OSA is multifactorial. It involves two major components. The first relates to the physical properties and spatial relationships of the pharyngeal airway. The second factor relates to both the normal sleep related decrease in action of the muscles which usually open the upper airway, but also includes abnormalities in that muscle action and its neurological control.

This thesis will examine the potential role of maxillary morphology in the development of OSA. A model, whereby the morphology of the maxilla could influence nasal resistance, is proposed as a possible factor in the development of OSA.

1.2.5 Management of obstructive sleep apnoea

Management strategies of OSA can be divided into three categories; namely,

(1) General conservative measures,
(2) Medical treatment, and
(3) Surgical treatment.

1.2.5.1 Conservative measures

These measures include advice on sleep posture, weight loss and alcohol consumption.

The earliest therapy for snoring and sleep apnoea may arguably have been an elbow nudge to the ribs of the snorer in an effort to stop the snoring or to induce a change of sleep position, usually from a supine posture to a sideways posture.
Many studies have investigated the effect of sleep posture and sleep apnoea severity (Cartwright, 1984; Cartwright et al., 1985, 1991; McEvoy et al., 1986; George et al., 1988). However, the beneficial effects of a lateral sleeping position appeared to be limited to patients with minor obesity and mild forms of sleep apnoea syndromes (Cartwright, 1984; George et al., 1988). Patients with marked obesity and hypoxemia seem to benefit by sleeping in a more upright position at a 60° angle (McEvoy et al., 1986).

Obesity is a well recognised aggravating factor of upper airway obstruction during sleep and is present in the majority of patients with sleep apnoea (Peiser et al., 1984). Weight loss may improve breathing during sleep both by increasing lung volumes and resting arterial blood gas tensions (Thomas et al., 1989), and by decreasing nasopharyngeal collapsibility (Suratt et al., 1987; Rubinstein et al., 1988; Schwartz et al., 1991).

Patients with known OSA are observed to have an increased frequency and duration of obstructive apnoeic episodes as well as a lower nadir of haemoglobin oxygen saturation during sleep after alcohol ingestion (Guilleminault and Rosekind, 1981; Issa and Sullivan, 1982). These authors also noted that alcohol evoked obstructive apnoea in heavy snorers who did not otherwise manifest apnoeas. Alcohol ingestion also decreased genioglossus activity in normal individuals (Krol et al., 1984) and increased upper airway collapsibility (Issa and Sullivan, 1982) in non-snorers and snorers. Therefore, patients who are known to have sleep disordered breathing, obese individuals and heavy snorers should all be encouraged to abstain from alcohol consumption.

1.2.5.2 Medical treatment

This therapy includes the use of CPAP (Sullivan et al. 1981) and oral appliances (Lowe, 1993, 1994).

Nasal CPAP treatment is becoming part of the routine expertise of sleep disorder centres. It secures the patency of anatomically narrowed and collapsible
upper airways and restores unobstructed breathing during sleep. An adequate pressure level is usually set at a range from 5 to 20 cm of H₂O. Nasal CPAP treatment is usually offered on a trial basis after a positive diagnostic sleep study. It has few contraindications and does not produce serious immediate side effects. However, it is not curative and a recent report has raised doubts about long term compliance (Kribbs et al., 1993).

With the growing recognition of the role of orthodontic abnormalities in the development of OSA, the use of oral appliances is gaining popularity. They offer an alternative treatment modality that may be attractive for patients who are dissatisfied with nasal CPAP or unwilling to accept surgical treatment. The oral appliances are effective to varying degrees and appear to work due to an increase in airway space. This change is achieved by the provision of a stable anterior position of the mandible, the advancement of the tongue and/or the soft palate, and possibly by a change in genioglossus muscle activity (Lowe, 1993).

The American Sleep Disorders Association guidelines for the use of oral appliance therapy for OSA requires patients to be diagnosed with polysomnography. The severity of the sleep-related respiratory problems can then be established and decisions for the appropriate treatment implemented. In addition, the sleep data can be used as a baseline to establish the effectiveness of subsequent treatment (American Sleep Disorders Association and Sleep Research Society, 1995).

Currently, there are about thirty or so patented oral appliances on the market. They fall into two major classes, those that advance the mandible and those that are designed to keep the tongue in an anterior position during sleep (Lowe, 1994). A recent review of three hundred and twenty patients treated with oral appliances for snoring and obstructive sleep apnoea was carried out by Schmit-Nowara et al. (1995). Snoring is improved in almost all patients and is often eliminated. The mean results of studies demonstrated that obstructive sleep apnoea improves in the majority of patients, but as many as 40% are left with a notably elevated apnoea hypopnoea index. Limited follow-up data indicated that
oral discomfort is a common but tolerable side effect, that dental and mandibular complications appear to be uncommon, and that long term compliance varies from 50% to 100% of patients.

Nevertheless, a comparison of the risks and benefits of oral appliances presents a useful alternative, especially for patients with simple snoring and others with moderate OSA who cannot tolerate nasal CPAP (Schmit-Nowara et al., 1995).

1.2.5.3 Surgical treatment

Upper airway surgery is considered when there are anatomical abnormalities such as nasal septal deviation, excessive palatal soft tissues, enlarged tonsils, retrognathia or severe craniofacial abnormalities. A recent review paper by Sher et al. (1996) included surgical techniques such as uvulopalatopharyngoplasty (UPPP), uvulopalatopharyngoglossoplasty, laser midline glossectomy, lingualplasty, inferior sagittal mandibular osteotomy and genioglosssal advancement with hyoid myotomy and suspension (GAHM), maxillo-mandibular advancement, and tracheostomy. Tracheostomy has been successfully used in sleep apnoea for several years (Guilleminault et al., 1981). However, with the advent of nasal CPAP, it has become the last surgical choice.

In 1964, UPPP was developed by Ikematsu for the treatment of heavy snoring. This surgical technique was then introduced as a treatment for OSA in North America in 1981. Accumulated data from a decade of studies suggest the need for extreme caution in performing this form of surgery for OSA (Harmon, Morgan and Chaudhary, 1989; Larson, Carlsson-Nordlander and Svanborg, 1991; Rodenstein, 1992).

Maxillo-mandibular surgery is usually proposed when patients are unable to tolerate nasal CPAP (Powell, Riley, and Guilleminault, 1990) unless there is gross craniofacial discrepancy as in syndromic patients. Orthognathic surgery has been cited by some in the treatment of OSA (Alvarez, Lessin and Gross, 1987; Waite et al., 1989; Riley and Powell, 1990; Riley et al., 1990). In theory, the advancement of
the maxilla increased the airway patency at the level of the soft palate and advancement of the mandible increased the airway posterior to the tongue base.

Riley et al. (1990) reported a 97% success rate on 40 patients who had maxillo-mandibular osteotomies with hyoid advancement. Criteria for success were based on a RDI of less than, or equal to, 20 in addition to a 50% or greater reduction in the RDI. Three patients, after a 36 month follow up, had no symptoms of apnoea confirmed by polysomnography.

In summary, nasal CPAP therapy is still the treatment of choice for OSA even though there is a compliance problem. Surgical treatment such as maxillofacial surgery and UPPP are dictated by the appropriate anatomical abnormalities present. Oral appliances are effective in treating snoring and moderate OSA, and are an attractive alternative to the other therapies, but the results are unpredictable. Therefore, there is still a need to search for a more definitive treatment and/or preventive strategy for OSA.

1.3 CRANIOFACIAL ABNORMALITIES AND OSA

This thesis is concerned with the anatomical component in the pathophysiology of OSA. It is a well established fact that OSA occurs in patients with gross and obvious craniofacial deformities such as severe mandibular micrognathia or retrognathia (Valero and Alroy, 1965; Tammeling et al., 1972; Coccagna et al., 1978). With the increasing use of cephalometry, more subtle craniofacial abnormalities have been found to occur commonly in patients with OSA, and this association has recently been reviewed (Cistulli, 1996a). Figure 1.2 presents a schematic summary of the morphological differences between a "typical" OSA patient and a normal control.
1.3.1 Cephalometric abnormalities in OSA

The first reports of cephalometric abnormalities in OSA patients were in the early 1980s. The study by Riley et al. (1983) drew attention to a narrow posterior air space (PAS), retrognathia, an inferiorly positioned hyoid bone relative to the mandibular plane, and elongation of the soft palate in OSA patients. These same findings were also reported by numerous other studies (Guilleminault et al., 1984; Jamieson et al., 1986; Lowe et al., 1986; deBerry-Borowiecki et al., 1988; Strelzow et al., 1988; Lyberg, Krogstad and Djupesland, 1989). In a recent investigation,
Maltais et al. (1991), found no significant difference in the PAS. However, patients with OSA tended to have a smaller PAS than non-apnoeic snorers who, in turn, had a smaller PAS than normal controls.

Lowe et al. (1986) documented several alterations in the craniofacial morphology of 25 males with OSA. These alterations included a posteriorly positioned maxilla and mandible, a steep mandibular angle, high upper and lower facial heights, overerupted maxillary and mandibular teeth, and proclined incisors. An elongation of the soft palate, and a retruded maxilla and mandible, were found to be the major discriminating variables which distinguished OSA patients from normals.

An increased lower facial height and an increase in the length and width of the soft palate were also most commonly reported (deBerry-Borowiecki et al., 1988; Bacon et al., 1990; Maltais et al., 1991; Yildrim et al., 1991). A retrognathic mandible has been noted in OSA patients by some authors and believed to be involved in occluding the airway at the level of the base of the tongue (Jamieson et al., 1986; Lowe et al., 1986; Strelzow et al., 1988). Other recent studies showed that there is a backward rotation of the mandible (Lyberg et al., 1989; Bacon et al., 1990). Some authors have reported a retropositioned maxilla and a more elongated upper face (Lowe et al., 1986; deBerry-Borowiecki et al., 1988; Strelzow et al., 1988).

A more acute cranial base angle (Na-S-Ba) was recorded by Jamieson et al. (1986), Steinberg and Fraser, (1995), and Battagel (1995). Battagel also found a significant reduction in the anterior cranial base length in patients with OSA. Similarly, Bacon et al. (1990) found that the anterior cranial base was shortened in OSA patients and so was the length of the midface when measured from the center of the condyle to subspinale (A point). The distance from basion to posterior nasal spine (Ba-PNS) was also significantly reduced.
Similarly, Tangugsorn et al. (1995) also found that OSA patients had a significantly shorter cranial base and maxillary length in their sample of 100 OSA patients and 36 controls. Other significant findings were similar with most other studies, and these included mandibular retrognathia, reduced size of the bony pharynx, an inferiorly positioned hyoid bone, increased anterior lower face height and mandibular plane, and a deviated posture with a larger cranio-cervical angle.

Calculations have been made of the area of the naso-, oro- and hypopharyngeal airways and a significant decrease has been recorded in patients with OSA (deBerry-Borrowiecki et al., 1988; Strelzow et al., 1988).

Some authors found that tongue length and area were significantly larger in obstructive sleep apnoea patients and thought to be associated with a reduced airway due to the posterior positioning of the tongue base (Lowe et al., 1986; deBerry-Borrowiecki et al., 1988; Strelzow et al., 1988). This condition was found to be associated with a backward rotated mandible, increased upper and lower facial height, a posteriorly positioned maxilla and mandible, a steep occlusal plane, extruded teeth, proclined incisors and open bites (Lowe et al., 1986; Bacon et al., 1990).

In a recent report by Pracharktam et al. (1994), the palatal plane was also rotated counterclockwise which contributed to a short effective dimension of the maxilla in OSA patients. Furthermore, the maxilla was also retropositioned with respect to the angle of the posterior wall of the maxilla and cranial base. He concluded that the cranial base, the position of the maxilla, ramus width, intermaxillary relation and hyoid bone position were the skeletal boundaries for the soft tissues of the upper airway passage. A discrepancy among any, or all, of these structures could contribute to an anatomical predisposition to a great number of apnoeas.

In summary, it is quite clear that OSA patients had craniofacial features that were different from normal controls. The more commonly reported features were
an increased mandibular plane angle, an increased mandibular plane to hyoid bone distance, a decreased mandibular body length and an increased soft palate length. Less commonly reported features were a shorter anterior cranial base with a more acute cranial base flexure, a shorter maxillary length, a reduced bony pharynx, and maxillo-mandibular retrognathia.

All of these studies ignored the transverse dimension and described only the features in the sagittal plane. The airway is a three dimensional conduit and the transverse dimension is just as important as the sagittal and hence needs to be assessed as well. This thesis aims to explore the potential role of the maxilla with an emphasis on its transverse dimension and its relationship to the development of OSA.

1.3.2 Relationship of cephalometric variables to apnoea severity

Many studies have found that various cephalometric measurements correlate with the degree of OSA (Partinen et al., 1988; Davies and Stradling, 1990; Tsuchiya et al., 1992; Ferguson et al., 1995). Davies and Stradling (1990) reported significant univariate correlations between oxygen “saturation dip rate” and hyoid position, soft palate length and hard palate to spine angle. Using a stepwise regression analysis Davies and Stradling, also found that neck circumference and retroglossal space were the only significant independent correlates.

Others have reported that an increased mandibular plane angle, measured erect (Partinen, et al., 1988) or supine (Yildrim et al., 1991), and a decreased posterior airway space (Partinen et al., 1988) were significant predictors of apnoea. Apnoea frequency has also been found to correlate with the logarithm of tongue volume, the position of the mandible (SNB), the maxilla/mandible relationship (ANB), the overbite, and BMI when using a stepwise regression analysis (Lowe, 1994).
Ferguson et al. (1995) in their study of the relationship between obesity and craniofacial structure concluded that there is a spectrum of upper airway soft tissues and craniofacial abnormalities among patients with OSA. The OSA patients can be categorised into 3 groups:

1. Obese patients with increased upper airway soft tissue structures,
2. Non obese patients with abnormal craniofacial structures, and
3. An intermediate group of patients with abnormalities in both craniofacial structure and upper airway soft tissue structures.

In the same vein, Tsuchiya et al. (1992), classified OSA patients on the basis of their apnoea index and BMI. When BMI was taken into account, three subgroups of OSA patients appeared to emerge; namely, morbidly obese individuals with few cephalometric abnormalities, individuals who were not too obese but had marked cephalometric abnormalities, and a large group with combined obesity and cephalometric abnormalities. These studies suggested that it is the combined effect of obesity and abnormal craniofacial structure that determines the development of OSA. This viewpoint gives support to a model of OSA whereby the degree of craniofacial abnormality in a patient determines the degree of obesity required to cause OSA.

This concept was supported to a degree by Cistulli (1994) in his study of OSA in patients with Marfan's syndrome. These patients were not obese and were found to have significant cephalometric abnormalities. However, as these patients had a disorder in their connective tissues, it appears likely that laxity of these tissues played a role in the development of OSA in this group of patients (Cistulli et al., 1995).
1.4 UPPER AIRWAY OBSTRUCTION AND FACIAL DEVELOPMENT

The influence of airway obstruction on facial development has been reported in the orthodontic literature as early as the 1860s (Meyers, 1870). Recent surveys of this literature have been given by O'Ryan et al. (1982), Cooper (1989), Tourne (1990) and Warren and Spalding (1991).

One current explanation is that nasal airway inadequacy, resulting in oral breathing produced postural changes which could lead to altered dentofacial growth (McNamara, 1973). In addition, a study by Solow and Kreiborg (1977) has proposed that impaired nasal respiratory function leads to modification of head posture, which may have influenced dentofacial development. The change in posture was thought to be necessary to counteract the obstruction by extending the head backwards in an effort to increase the airway. This change in head position and increase in the cranio-cervical angle has also been observed in studies by Solow and Greve, (1979), Woodside and Linder-Aronson (1979) and Wenzel et al. (1985). Of interest is the recent finding by Solow et al. (1991) that this postural sequel to airway obstruction is also present in patients with OSA.

Other than an increase in the cranio-cervical angle, other dentofacial features observed in association with nasal obstruction were a decrease in mandibular size, retrognathia, an increase in lower anterior face height and a steep inclination of the mandibular plane (Linder-Aronson, 1970; Solow, Siersbaek-Nielsen and Greve, 1984). These features, which were similar to those identified in adult OSA patients, led to the concept that OSA may have evolved during childhood before becoming clinically obvious in adult life. This theory is supported by Guilleminault and co-workers (1989) who showed that cephalometric abnormalities accounted for persisting snoring and upper airway obstruction in adolescents who had undergone tonsillectomy and adenoidectomy for upper airway obstruction prior to puberty.
Animal studies support the important link between airway obstruction and craniofacial development. Harvold et al. (1981) occluded the nasal passages of nine growing infant rhesus monkeys, forcing them to breathe orally, and compared their growth radiographically to that of a group of nine control monkeys. The occluded groups showed significantly increased facial height, decreased maxillary dental arch length, decreased maxillary and mandibular intercanine distance, and alteration of tongue and mandibular morphology.

The neuromuscular changes associated with these morphologic changes were explored in electromyographic studies by Miller et al. (1978, 1980, 1982, 1984). They found new neuromuscular patterns with rhythmic recruitment of specific craniofacial muscles. When the obstruction was removed at a sufficiently early age, there was a partial regression of the anatomical changes, but the altered neuromuscular activity persisted (Vargervik et al., 1984; Miller et al., 1984).

The above findings are of interest as they provide a possible explanation for the evolution of OSA in humans. Most individuals who snore, and patients with OSA syndrome, are mouth breathers during sleep (Sher, 1990). Therefore, the question arises, "Could it not be possible that over time, mouth breathing in OSA can influence the shape of the maxilla as it develops?" This thesis aims to test the hypothesis that OSA patients have different maxillary morphology when compared with normal controls.

1.4.1 Maxillary morphology and nasal obstruction

Several authors have suggested that abnormal oral and nasal pressures during breathing hinder palatal descent causing abnormal maxillary growth. (Kantorowicz, 1916; Wustrow, 1917; Norlund, 1918; Linder-Aronson and Backstrom, 1960). These researchers have suggested that oral breathing leads to increased negative air pressure in the nasal cavity, thereby preventing palatal descent. Alternatively, they have proposed that oral air pressure against the palate is sufficient to prevent normal palatal function. Norlund (1918, cited in
Bushey, 1979) indicated that nasal obstruction due to enlarged adenoids could cause the nasal cavity to undergo disuse atrophy, resulting in a deep palate from the lack of downward palatal vault growth and continued alveolar process growth.

Solow (1992) explained that the extension of the head in relation to the cervical column which accompanied upper airway obstruction also lifted the head, and the maxillary dental arch away from the tongue. If the concept is accepted that the form of the dental arches is shaped by the balancing forces of the tongue, the lips and the cheeks, then, the lower relative position of the tongue caused by an increase in the cranio-cervical angulation would explain the constriction of the maxilla and the occurrence of posterior crossbites. Certain types of malocclusion, such as a high palatal vault and constricted maxilla, do seem to be associated with nasal airway impairment (Warren, 1990).

1.4.2 Maxillary morphology and OSA

Marfan’s syndrome, is a genetic connective tissue disorder with abnormalities of the musculoskeletal, cardiovascular and ocular systems (Hirst and Gore, 1973). Dentally, these patients characteristically, have a high-arched palate associated with maxillary arch constriction. In addition, 70% of these patients suffer from OSA (Cistulli, 1993).

A series of studies of Marfan’s syndrome patients has shed some light on the potential importance of maxillary morphology in the development of OSA (Cistulli, 1994; Cistulli et al., in press). The hard palate not only forms the roof of the mouth, but also the floor of the nasal cavity. Therefore, a high-arched palate may narrow the nasal airway, resulting in a higher resistance. With this concept, Cistulli evaluated the role of maxillary arch constriction and palatal height in determining nasal airway resistance and the severity of OSA in 13 patients with Marfan’s syndrome. Nasal airway resistance was measured by posterior rhinomanometry. Maxillary constriction was assessed by measuring inter-cuspid distance, inter-premolar distance (distal fossae) and inter-molar distance.
(central fossae) from dental casts. The maximum height of the palate and the maxillary basal arch length were also recorded.

Highly significant correlations were found between the degree of maxillary constriction and nasal airway resistance after decongestant \( r=0.9, p<0.005 \), and OSA severity \( r=0.63, p<0.05 \). These findings strongly suggest that the shape and dimensions of the maxilla are important determinants of nasal airway resistance and OSA severity in patients with Marfan's syndrome. Whilst these findings appear to be relevant to OSA in Marfan's syndrome patients, could maxillary constriction be important in the development of "idiopathic" OSA?

A pilot study conducted by Palmisano, Sullivan and Cistulli (1994) specifically looked at orthodontic abnormalities in 35 consecutive patients presenting for nocturnal polysomnography. All subjects had varying degrees of OSA. There were thirty males (mean age of 48 years, range 14-72), and five females (mean age 55 years, range 34-68).

Examination included analysis of tooth size to basal bone size, analysis of dental and skeletal relationships, postural analysis and auscultation, and palpation of the temporomandibular joints through their range of movement in function. Photographic records, study models and cephalometric radiographs were also taken for some patients.

The study by Palmisano et al. (1994) showed that 55% of the patients had Class I malocclusion, 11% had Class II malocclusion, 33% had Class III malocclusion, 64% had evidence of bruxism and 62% had maxillary constriction. Their data indicated a very high proportion of Class III malocclusion. Maxillary constriction was diagnosed on clinical evidence of dental crossbite. No measurements were made. It appeared that a high proportion of these patients had maxillary constriction. The authors believed that some of these abnormalities were the result of sleep disorder breathing and suggested that orthodontic treatment aimed at correcting these abnormalities might be of therapeutic benefit for OSA patients.
Guilleminault et al. (1995) have recently reported a high odds-ratio for the presence of a high narrow hard palate in the immediate family relatives of index cases with OSA. A recent abstract from the Stanford Sleep Disorders Clinic and Research Center in California (Kushida et al., 1996) reported the use of the height of the palate, maxillary intermolar distance and mandibular intermolar distance as part of a morphometric model for clinical prediction of OSA. These investigators suggested that the transverse dimensions of the maxilla and mandible were important parameters to consider and may have a role to play in the development of OSA.

Clearly, these findings, and the results of the pilot study, have led to the hypothesis that the maxillary morphology of OSA patients is different from normal controls and that the shape and size of the maxilla could be involved in the development of OSA.

1.5 MAXILLARY CONSTRICTION

1.5.1 Skeletal constriction

Maxillary constriction could be due to a dental arch constriction, a skeletal base constriction, or a combination of both. Skeletal maxillary constriction is not well defined in the orthodontic literature. Proffit (1993) stated that skeletal maxillary constriction was distinguished by a narrow palatal vault. He compared the width of the palatal vault to the inter-tooth distance of the mandible. He considered the width inadequate if there was a large discrepancy between the width of the palatal vault and the inter-tooth distance (Figure 1.3). However, it is not clear at what level these measurements were taken. As there was no mention of the height of the palatal vault, it is also assumed that the vertical dimension had no influence in the assessment of skeletal maxillary constriction.
Figure 1.3  Skeletal crossbite. Distance CD is considerably larger than distance AB. Adapted from Contemporary Orthodontics. (Proffit 1993).

 Clinically it is difficult to measure the widths of the maxillary and mandibular bases. Hence, transverse discrepancies are traditionally assessed by the presence of dental crossbites. If a crossbite is present, the next step is to determine whether the crossbite is of dental or skeletal origin. A purely dental crossbite implies normal width of the palate, with the teeth tilted toward the midline. The presence of a purely skeletal crossbite implies that the palatal width is inadequate, causing the teeth to compensate by tilting out more than normal but, nevertheless, remaining in crossbite (Proffit and Ackerman, 1994).

 On the other hand, the absence of crossbite does not necessarily mean that the maxilla is not constricted. Dento-alveolar compensation (Solow, 1980) can mask a transverse discrepancy by buccal tilting of the upper teeth and upper alveolar arches and by lingual tilting of the lower teeth.

 Recently, McNamara (1994) used inter-molar distance as an indicator of maxillary bony base development. He proposed a simple rule of thumb, indicating an ideal average inter-molar width in males of 37.4mm (SD 1.7mm) and in females of 36.2mm (SD 1.92mm). These measurements were made at the intersection of the lingual groove with the gingival margin of the maxillary first permanent molars. However, these data were based on the work of Howe and co-workers (1983) who compared arch widths of two subjectively selected groups of dental casts. Such methods are open to selection bias and may not be representative of the
population at large. Hence, measuring the inter-molar width only, and not considering other features, is an inaccurate way to assess skeletal maxillary constriction.

Skeletal maxillary constriction is defined by the present author as a combination of the following features:

a) a narrow and high palatal vault,
b) a corresponding narrow archform,
c) unilateral or bilateral buccal tilting of maxillary alveolar arches, and
d) unilateral or bilateral buccally inclined maxillary posterior teeth in crossbite or in an edge to edge relationship, with the lower teeth.

1.6 MAXILLARY ARCHFORM

1.6.1. Arch dimensions

The maxillary denture is housed by the alveolar process which in turn sits on the basal bone or apical base of the maxilla. The size, form and boundaries of the apical base were difficult to determine. Downs (1944) demonstrated that it was possible to delineate both dental arch, and what was assumed to be the maxillary apical base, by x-raying the plaster model from directly above with the teeth resting on an x-ray film.

Richardson and Brodie (1964) refined and validated the Downs’ technique. They conducted a pilot study on 25 patients by positioning the x-ray tube above and in front of the head with the x-ray axis directed at right angles to the film between the teeth. These x-rays were found to be comparable to the x-rays of the models of the same individuals. Even so, it was difficult to establish the normal relationship between the dental arch and the apical base. However, they found
that the correlation between the area of the apical base and that of the dental arch was statistically significant.

Tooth position provides a stable reference basis as indicated by the usual stability of inter-molar and inter-canine width dimension (Proffit and Ackerman, 1994). In the past, dental arch width dimensions have been measured by using certain cusp tips or points at the lingual cervical margins of the teeth as reference points. However, cusp tips wear off and vary in location. Points at the lingual cervical margins are affected by the buccolingual width of a tooth and the level of eruption especially during the transition of primary teeth to their permanent successors (Moyers et al., 1976).

Moyers et al. (1976) illustrated the construction of the 'centroid' of the tooth as a reference point for the measurement of arch dimensions as follows:

![Diagram](image)

**Figure 1.4** Schematic illustration of the determination of the midpoints and centroid of a tooth. (Moyers et al., 1976)

A = midpoint between the approximal midpoints
B = points half way between the buccal and lingual points, and
C = the centroid, halfway between A and B.

In the majority of teeth studied, the centroid and the midpoint between the approximal midpoints coincided, or were very close to each other.
Methods of measurement of arch widths have ranged from the use of simple vernier callipers, or a Bole gauge, to the sophisticated Optacon machine (Moyers et al., 1976) or stereophotogrammetry (Berkowitz, 1971).

Arch depth measurement varied in different studies. In the study by Moyers et al. (1976), arch depth was measured from the midpoint of the most labial points of the central incisors to the maxillary first molars at the distal midpoints. Moyers’ method of recording inter-tooth distance was adopted for the present study.

1.6.2 Geometric representation of maxillary arch form

Various descriptions of archform by different authors were cited in an article by Biggerstaff (1972). The dental arches were qualitatively described as semi-ellipsoid (Black 1894), paraboloid (Angle, 1907), U-shaped (Martin, 1914), horseshoe-shaped (Hrdlicka, 1920) and catenary (Mac-Conail and Scher, 1949). Recently, another mathematic model of dental arch form was advocated by Brader (1972). It was based on a trifocal ellipse. The anterior segment of the trifocal ellipse closely approximated the anterior segment of the catenary curve, but the trifocal ellipse gradually constricted posteriorly in a way that the catenary curve does not.

Recently, the computer has been used to mathematically fit the curvature of the dental arches by exponential function (Hayashi, 1957), Fourier series (Lu, 1965) and fourth degree polynomial equations (Lu, 1966). Of these methods, the fourth, fifth, or sixth order least squares polynomial regression equations give the most accurate and reproducible likeness of arch form and arch length (Sanin et al., 1970). The application of fourth order polynomials of the form:

\[ y = a + bx + cx^2 + dx^3 + ex^4 \]

has a number of advantages, the most significant being that the coefficients can be easily interpreted (Lu, 1966).
The second ($x^2$, or quadratic) and the fourth ($x^4$, or quartic) coefficients described the arch shape while the first ($x$, or linear) and the third ($x^3$, or cubic) coefficients described asymmetry.

The larger the magnitude of the quadratic coefficients ($x^2$) the more the arch assumed the form of a parabola. Hence, we may say it was measuring the “taperedness” of the arch. If ($x^2$) is small, it implies that ($x^4$) is relatively large, and the consequence of a small ($x^2$) in physical terms would tend to give the arch a more square-like appearance. Therefore, ($x^4$) is a measure of “squared-ness” (Lu, 1966). Combinations of positive and negative linear ($x$) and cubic ($x^3$) terms described different types of arch asymmetry (Richards et al., 1990). For example, large positive linear coefficients ($x$) described lingual displacement of the left teeth and buccal displacement of the teeth on the right side. Large positive cubic terms ($x^3$) described similar shapes as the linear coefficients ($x$) and, in addition, were associated with a tendency for the mid-line to be displaced to the right, the left teeth to be displaced labially and the anterior right teeth to be linguually placed. In each case negative coefficients reflected the opposite effect.

$$y = 1E-05x^4 + 0.0006x^3 + 0.0359x^2 - 0.0646x - 0.675$$

**Figure 1.5**  A fourth degree polynomial equation generated by best fit of coordinates of the maxillary arch
1.7 AIMS OF THE STUDY

Recent work has demonstrated that maxillary morphology may be one important craniofacial abnormality in the development of OSA in patients with Marfan’s syndrome (Cistulli, 1994). In addition, an uncontrolled pilot study (Palmisano et al., 1994) suggested that maxillary constriction may be a common feature in the “typical” OSA population. These results, led to the hypothesis that patients with OSA have different maxillary morphology when compare with normal controls and that maxillary constriction is an important factor in the development of OSA. Therefore, the main purpose of this study was to test this hypothesis with the following aims.

1. To examine the prevalence of maxillary constriction in a random sample of OSA patients compared with normal controls.
2. To compare the maxillary inter-canine, inter-premolar and inter-molar distances between the two groups.
3. To compare the palatal indices at the above regions.
4. To represent the maxillary archforms with fourth order polynomial equations and to compare the quadratic (\(x^2\)) and quartic (\(x^4\)) coefficients between the two groups.
5. To examine the relationships between maxillary arch dimensions and posterior airway space (PAS).
6. To derive statistical correlations between maxillary and cephalometric dimensions and apnoea severity.
2 MATERIALS AND METHODS

2.1 SUBJECT SELECTION

2.1.1 Patients

The patient sample consisted of 40 randomly selected patients who presented for overnight polysomnography investigations at the St. George Hospital Sleep Disorders Centre. Edentulous patients were excluded because of the inability to assess their maxillary arch dimensions without dental landmarks. The author had no prior knowledge of the patient to be seen and there was no regular pattern of the days they were seen. These patients had all been assessed clinically by a sleep physician prior to polysomnography and were deemed to have a high likelihood of OSA. Data were collected in a time span of approximately 10 months.

2.1.2 Control subjects

The controls were recruited from the staff of the United Dental Hospital, Sydney. Subjects with a history of major jaw surgery or orthodontic treatment were excluded. All volunteers were screened by the use of a questionnaire to exclude heavy snoring, and significant excessive daytime sleepiness (EDS) which are the major symptoms of OSA. Assistance from bed partners was sought where possible. EDS was assessed with The Epworth Sleepiness Scale, a subjective measure of daytime sleepiness. These questionnaires have been previously shown to be valid and reliable (Johns, 1991, 1992; Kump et al., 1994). An attempt was made to match controls and patients for age, weight and height. A total of 35 subjects was recruited, but only 21 were accepted as controls based on the absence of snoring and significant daytime sleepiness.
2.1.3 Consent and ethics

The research protocols described in this thesis were all approved by the Ethics Review committee of the Central Sydney Health Service as well as the Ethics Review committee of the United Dental Hospital. All control subjects gave their written consent under witness prior to participation in the study.

2.1.4 Anthropomorphic measurements

The weight, height, neck circumference and waist measurements of each patient and control were recorded. Neck circumference was measured at the level of the cricothyroid. Obesity was expressed as a Body Mass Index (BMI).

\[ \text{BMI} = \frac{\text{Weight in kilograms}}{\text{Height in metres}^2} \]

2.2. SLEEP-RELATED MEASUREMENTS

2.2.1 Overnight polysomnography

The following is a description of a typical overnight sleep study where many variables were recorded. For the purpose of this study, only the variables, minSaO₂ and the number of apnoeas and hypopneas were utilised.

Polysomnograms were performed between 9pm and 7am. All variables were recorded continuously on a 16-channel polygraph (Grass Instrument, Quincey, Mass). The sleep data were relayed and displayed on monitor screens in the monitoring room next to the sleeping room. Table 2.1 summarises the polysomnographic information. Figure 2.1 and 2.2 show the patient "set up" ready for the sleep study.
### Table 2.1 Polysomnographic information

<table>
<thead>
<tr>
<th>Signal</th>
<th>Information obtained</th>
</tr>
</thead>
<tbody>
<tr>
<td>EEG</td>
<td>Gross sleep architecture, sleep staging</td>
</tr>
<tr>
<td>EOG</td>
<td></td>
</tr>
<tr>
<td>EMG</td>
<td>Apnoea-hypopnoea</td>
</tr>
<tr>
<td>Oronasal airflow</td>
<td></td>
</tr>
<tr>
<td>Rib-cage and abdominal movements</td>
<td>Respiratory effort. Paradox may suggest upper airway</td>
</tr>
<tr>
<td>movements</td>
<td>obstruction</td>
</tr>
<tr>
<td>Calibrated rib-cage and abdominal</td>
<td>Apnoea-hypopnoea</td>
</tr>
<tr>
<td>movements ( rarely accurate )</td>
<td></td>
</tr>
<tr>
<td>ECG</td>
<td>Cardioacceleration of arousal</td>
</tr>
<tr>
<td></td>
<td>Bradycardia of apnoea</td>
</tr>
<tr>
<td></td>
<td>Arrhythmias</td>
</tr>
<tr>
<td>Snoring</td>
<td>Upper airway obstruction</td>
</tr>
<tr>
<td>Oxygen saturation</td>
<td>Respiratory adequacy ( indirectly, apnoeas hypopnoeas )</td>
</tr>
<tr>
<td>Posture</td>
<td>Links between posture and degree of upper airway</td>
</tr>
<tr>
<td></td>
<td>obstruction</td>
</tr>
<tr>
<td>Leg movements</td>
<td>Detect periodic movements of the legs during sleep</td>
</tr>
</tbody>
</table>

The polysomnographic variables recorded were

1) **Electroencephalogram (EEG).**
   The 4 points used were C3, A1, A2 and O2. Surface electrodes were attached to the scalp with the aid of a special skin glue. The EEG gave information on the different stages of sleep and their duration right through the night. It also recorded the total awake and sleep time. The latter included rapid eye movement sleep and non-rapid eye movement sleep.

2) **Electromyogram (EMG).**
   These surface electrodes monitored the electrical activity movements of the mentalis muscle, the supra-hyoid muscles, the anterior tibialis muscle of the leg and the diaphragm. Three electrodes were used for the mentalis and supra-hyoid muscles. Two electrodes were used for the leg muscle and only one electrode was positioned between the 7th and 8th intercostal space to monitor the movement of the diaphragm.

3) **Electrocardiogram (ECG).**
   A modified electrocardiogram using only 2 leads was used to monitor the heart.
4) Electro-oculogram (EOG).
A right and left oculogram was set up to monitor the eye movements. One electrode was placed on the right outer canthus approximately 1 cm above the eye. The other was placed 1 cm below the left outer canthus. Movements were discerned by the shift in electric polarity between the retina and skin surface.

5) Nasal and Oral airflow (Grass volumetric unit, Grass Instrument, Quincey, Mass.). A thermistor was attached either via a nasal cannula or nasal prong. It measured the temperature of the oral and nasal breathing.

6) Minimum Oxygen Saturation (Ohmeda Biox 3700e, Louisville, CO).
Minimum oxygen saturation was calibrated by pulse oximetry on the finger.

7) Abdominal and Thoracic movements (Respitrace, Ambulatory Monitoring Inc., Ardsley, New York). These were monitored by special bands that were strapped over the respective area. Movements were translated as ‘make or break’ of an electric circuit in the band and this information was transferred graphically, as a curve, to the computer monitor.

8) Transcutaneous Carbon dioxide.
This was monitored by an electrode maintained at 41°C near a surface artery.

9) Position monitor.
This was attached to the front of the chest and worked on the same principle as a spirit level.

10) Sound and video recording.
The sound of the snoring was recorded via a microphone over the bed and an overhead video camera was also used.

2.2.2 Scoring of sleep studies

Sleep recordings were scored in 30-second epochs and staged according to standard criteria (Rechtschaffen and Kales, 1968). Calculated respiratory variables were the RDI, apnoea duration, and minSaO₂ during apnoeas.

For the current thesis, apnoea was defined as cessation of airflow for at least 10 seconds with oxygen desaturation undefined, or associated with an oxygen desaturation of more than 4%. Hypopnoea was defined as a reduction in
amplitude of airflow or thoraco-abdominal wall movement of greater than 50% of the baseline measurement for more than 10 seconds without oxygen desaturation. Hypopnoea can also be defined with the same reduction in amplitude of airflow with an accompanying oxygen desaturation of at least 4% and associated with arousal. These respiratory events were defined as obstructive if they occurred in association with continued diaphragm EMG activity and thoraco-abdominal wall movement. Central apnoeas were defined as those accompanied by absence of diaphragm EMG activity and thoraco-abdominal wall movement. The definition of OSA for this thesis is a RDI equal to or more than 5.

Figure 2.1  Patient “wired up” for overnight sleep study
Figure 2.2 Close up view of (a) EEG leads, (b) nasal airflow cannula, (c) EOG leads, (d) EMG leads, (e) thoracic band, (f) abdominal band, (g) pulse oximetry and (h) junction box circuit.

Figure 2.3 Sleep Laboratory technician in monitoring room.
2.2.3 Questionnaire for selecting controls

The main aim of the questionnaire was to assess the presence or absence of snoring. If the latter was present, the frequency and intensity of snoring, and daytime sleepiness. The criteria for the selection of normal controls included no history of snoring, or very rare occasions of snoring, with no excessive daytime sleepiness.

The sleep questionnaire was designed to be simple and effective. This was in step with the finding that predictive ability is not significantly improved with multiple questions or a separate roommate questionnaire (Kump et al., 1994). Appendix 3 presents the full questionnaire.

Questions 1, 2 and 3 refer to snoring frequency and intensity which were found to be significantly associated with apnoea (Kump et al., 1994). A 5 point frequency scale was used:

(0) Never.
(1) Rarely, < once/week.
(2) Sometimes, 1-2/week.
(3) Frequently, 3-4 / week.
(4) Almost always, 5-7 /week.
(5) Not sure.

Subjects who answered (0) and (1) for snoring frequency and intensity were accepted as controls.

Question 4 assessed the daytime sleepiness using the Epworth Sleepiness Scale (ESS). The ESS is a simple and reliable method for measuring persistent daytime sleepiness in adults (Johns, 1992). Subjects were asked to rate, on a scale of 0-3, how likely they would be to doze off or fall asleep in each of 8 different situations.
(1) Sitting and reading.
(2) Watching TV.
(3) Sitting, inactive in a public place (e.g. a theatre of meeting).
(4) As a passenger in a car for an hour without a break.
(5) Lying down to rest in the afternoon when circumstances permit.
(6) Sitting and talking to someone.
(7) Sitting quietly after a lunch without alcohol.
(8) In a car, while stopped for a few minutes in the traffic.

A distinction was made between dozing off and simply feeling tired. The numbers selected for the 8 situations in the ESS were added together to give a score for each subject, anywhere between 0 to 24. ESS scores greater than 16 were indicative of a high level of daytime sleepiness encountered in patients with moderate or severe OSA (Johns, 1991). These ESS scores proved capable of distinguishing individuals and diagnostic groups over the whole range of daytime sleepiness. It was also found that this ESS score was correlated with both the RDI and the minSaO₂ recorded during polysomnography in patients with OSA of differing severity (Johns, 1991). Question 5 and 6 assessed the sleep quality of a typical night’s sleep.

2.3 ORTHODONTIC EVALUATIONS

2.3.1 Clinical Assessment

A brief medical and dental history was first taken. The usual route of breathing, nasal patency, history of nasal and airway surgery was noted. A clinical orthodontic examination was then carried out (Appendix 4).

Extra-orally, the soft tissue profile and the vertical proportions were evaluated with the patient in centric occlusion and lips relaxed. An estimate of the mandibular plane angle was then recorded. Next, a functional analysis of the temporomandibular joints was carried out. This included checking the relationship
of the centric occlusion to centric relation, followed by the right, left and protrusive movements of the mandible.

Intra-orally an assessment of the occlusion was carried out. The amount of overjet and overbite, the presence or absence of posterior crossbites were recorded. The shape of the dental arches and depth of the palatal vault were also noted. However, it was difficult to ascertain the size of the tongue and pharyngeal opening. Skeletal maxillary constriction was assessed using the above information. The presence of the following features collectively constituted skeletal maxillary constriction:

a) a narrow and high palatal vault.

b) a corresponding narrow archform.

c) unilateral or bilateral buccal tilting of the maxillary alveolar arches.

d) unilateral or bilateral buccally inclined maxillary posterior teeth in crossbite or edge-to-edge relationship with the lower teeth.

Next, facial and intra-oral photographs were taken. These included an anterior view, a right lateral and a left lateral view of the face. The intra-oral photographs included an anterior view with the teeth in centric occlusion, a right and left buccal view and occlusal views. Upper and lower impressions and a wax squash bite were also taken. No photographs of the controls were taken.

2.3.2 Impressions and Casts

Alginate impression material (Unijel-II, Unitek/3M, Type I fast setting) was mixed according to the manufacturer's instructions. Upper and lower impressions were taken and orthodontic stone poured within half an hour. Orthodontic stone (Whip mix, ADA type III) weighing 900gm was added to 250 ml of water and vacuum mixed for 30 seconds for pouring up a set of upper and lower models with bases. Casts were trimmed with the occlusal plane parallel to true horizontal. All the above procedures were carried out by the author to minimise inter-operator errors.
2.3.3 *Lateral and Postero-anterior cephalograms*

Lateral and postero-anterior cephalograms were taken with the patient in a standing position, using a cephalostat which standardised head position in conjunction with the film holder. Each patient was instructed to close in centric occlusion and to take a breath and slowly exhale. X-rays were exposed during the expiratory phase. The exposure parameters were arranged to maximally observe hard and soft tissue landmarks (Figure 2.4).

Different x-ray machines were used for the sample because patients and controls were recruited from different institutions. For the patients, the distance of the head from the source to the median plane was 135.0 cm, while the distance from the median plane of the head to the film plane was kept at 15.0 cm. For the controls, the distances were 153.0 cm and 15.0 cm, respectively. The x-rays for the patient had a magnification of 11% while the x-rays for the controls had a magnification of 10%.

![Diagram of x-ray setup](image)

*Figure 2.4* Distance between the x-ray source and patient and the film distance from the patient. (Proffit 1993).
2.3.3.1  Tracing

All the radiographs were hand traced by the author on acetate paper over a light-viewing box. Where bilateral landmarks presented two images, the average of the two was drawn in. A month later, 30 randomly chosen lateral and postero-anterior cephalograms were re-traced to check for errors and reproducibility.

The following landmarks and planes were used for the lateral cephalograms

A point  The most posterior point on the curve of the maxilla between the anterior nasal spine and supradentale.
Aa  The most anterior point on the atlas vertebrae.
AH  The most anterior and superior point on the body of the hyoid bone.
Ar (Articulare)  Articulare (posterior), the point of intersection of the inferior cranial base surface and the averaged posterior surfaces of the mandibular condyles.
ANS  Anterior nasal spine, the tip of the median, sharp bony process of the maxilla at the lower margin of the anterior nasal opening.
B point  The point most posterior to a line from Infradentale to Pogonion on the anterior surface of the symphyseal outline of the mandible.
Ba (Basion)  The most inferior, posterior point on the anterior margin of the foramen magnum in the median plane.
Co (Condylion)  The most posterior superior axial point on the curvature of the average of the right and left outline of the condylar head.
Eb  Base of epiglottis.
Gn (Gnathion)  The most anterior-inferior point on the contour of the bony chin symphysis. Determined by bisecting the angle formed by the mandibular plane and a line through Pogonion and Nasion.

Go (Gonion)  The midpoint of the angle of the mandible. Found by bisecting the angle formed by the mandibular plane and a plane through Articulare posterior along the portion of the mandibular ramus inferior to it.

MP  Mandibular plane. A line joining Me to Go intersection.

Me (Menton)  The most inferior point on the symphyseal outline.

Na (Nasion)  The junction of the frontonasal suture at the most posterior point on the curve at the bridge of the nose.

Or (Orbitale)  The lowest point on the average of the right and left borders of the bony orbit.

P  Tip of the soft palate.

PNS  Posterior nasal spine.

Pg (Pogonion)  The most anterior point on the contour of the bony chin. Determined by a tangent through Nasion.

Po (Porion)  The most superior lateral point on the roof of the external auditory meatus.

Pw  Posterior pharyngeal wall. A point directly determined by the extension of the palatal plane to the pharyngeal wall.

S (Sella)  The centre of the pituitary fossa of the sphenoid bone determined by inspection.

S-N  Anterior cranial base.

TT  Tongue tip.

The following landmarks were used for the postero-anterior cephalograms

ZA/AZ  Centre of the root of the zygomatic arch, midpoints, ZA- left, AZ- right.
AG/GA  Points at the lateral inferior margin of the antegonial protruberences, AG -left, GA - right.

JL/JR  Bilateral points on the jugal process at the intersection of the outline of the tuberosity of the maxilla and the zygomatic buttress, J L -left, JR - right.

NL/NR  Maximum left and right curvature of the nasal aperture.

2.3.3.2    Analysis of the lateral cephalograms

Linear measurements, to the nearest millimetre, were made from the tracing. All measurements were adjusted to natural size by taking into account the magnification factor. The following measurements were made (Figure 2.5).

(a) ANS to PNS  The length of maxilla.
(b) Aa to PNS  The distance between the anterior point of atlas to posterior nasal spine.
(c) Ba to PNS  The distance between Basion to posterior nasal spine.
(d) PAS  The distance between the posterior pharyngeal wall and the dorsal surface of the base of the tongue. Measured on the line that intersects Go and B point.
(e) MP-H  Distance from the mandibular plane to Ah, the most anterior and superior point on the body of the hyoid bone (H).
(f) Go to Gn  Length of the body of the mandible.
(g) P to PNS  Length of the soft palate.
(h) PW to PNS  Distance from the posterior nasal spine to the posterior pharyngeal wall.
(i) TGLt  Length of the tongue, as measured from TT to Eb.
(j) TGHt  Tongue height. These linear distance along the perpendicular bisector of the Eb-TT line to the tongue dorsum.
Figure 2.5 Lateral cephalogram analysis:

Linear measurements (1) **ANS-PNS**, (2) **Aa-PNS**, (3) **Ba-PNS**, (4) **P-PNS**, (5) **PW-PNS**, (6) **TGHt**, (7) **TGLt**, (8) **PAS**, (9) **Go-Gn**, (10) **MP-H**.
2.3.3.3 Analysis of the postero-anterior cephalograms

The linear measurements, to the nearest millimetre were made from the tracing. Magnification was not accounted for because ratios were used for comparisons (Figure 2.6). The following measurements were made:

- JL-JR Maxillary width.
- AG-GA Mandibular width.
- ZA-AZ Facial width.
- NL-NR Maximum nasal width.

**Figure 2.6** Postero-anterior cephalogram landmarks

- **ZL/ZR** Bilateral points on the medial margin of the zygomatico-frontal suture, at the intersection of the orbits. ZL-left; ZR-right.
- **ZA/AZ** Centre of the root of the zygomatic arch, midpoints. ZA-left; AZ-right.
- **AG/GA** Points at the lateral inferior margin of the antegonial protruberences AG-left; GA-right.
- **JL/JR** Bilateral points on the jugal process at the intersection of the outline of the tuberosity of the maxilla and the zygomatic buttress, JL-left; JR-right.
- **NL/NR** Maximum left and right curvature of nasal aperture.
2.3.4 Assessment of maxillary morphology

2.3.4.1 Assessment of dental and skeletal maxillary constriction

Dental arch constriction is defined by the present author as the presence of two or more posterior teeth in edge to edge cuspal relationship or in frank cross bite either unilaterally or bilaterally.

Skeletal maxillary constriction was distinguished by the following features:

a) a narrow and high palatal vault.
b) a corresponding narrow archform.
c) unilateral or bilateral buccal tilting of the maxillary alveolar arches.
d) unilateral or bilateral buccally inclined maxillary posterior teeth in crossbite or edge to edge relationship with the lower teeth.

2.3.4.2 Arch width measurements

The following linear measurements were recorded from the study models using vernier callipers to the nearest second decimal:

a) Intercuspid distance, the distance between the centroids of the canines. The centroids were identified with a sharp pencil as described by Moyers et al. (1976).
b) Interpremolar distance, the linear distance between the centroids of the first and second premolars.
c) Intermolar distance, the linear measurement between the centroids of the first permanent molars.
Figure 2.7  Measuring Instruments
(a) Profile gauge, (b) Vernier calliper, (c) Bow divider,
(d) Stainless steel ruler.

Figure 2.8  Inter-canine distance measurement.
2.3.4.3 Palatal height measurement

A profile gauge was used to define the cross-sectional shape of the palate at the canine, first and second premolar and first molar regions by lining up the gauge over the centroid of the teeth (Figure 2.9). The palatal heights were then measured off the profile gauge with a steel ruler to the first decimal point (Figure 2.10). Pencil tracings of the cross-sectional shape of the palate at the different regions were transferred to graph paper.

Figure 2.9 The profile gauge used to define the shape of the palate.

Figure 2.10 Palatal height measurement with a ruler.
2.3.4.4 Maxillary depth measurement

The depth of the maxilla was measured with a pair of Bow dividers (Figure 2.11), between the midpoint of the most labial point of the central incisors to the maxillary first molars at the distal midpoints.

Figure 2.11 Maxillary depth measurement.
2.3.5 Description of archform with polynomial equation

The maxillary casts were photocopied at a 1:1 magnification. The mid-points of the incisal edges and the buccal cusp tips of the maxillary teeth were marked and traced on graph paper. A line drawn through the distal contact points of the first molars was orientated parallel to the x-axis, with the co-ordinates (0,0) coinciding with the contact point of the central incisors. Corresponding coordinates were then determined from the marked incisal edges and buccal cusp tips of the cast. The coordinates were fed into a computer to generate a 4th order polynomial curve and equation. (Microsoft Excel software).

![Graph paper overlay on top of photocopied image of cast locating archform co-ordinates.](image)

**Figure 2.12** A graph paper overlay on top of photocopied image of cast locating archform co-ordinates.

\[ y = 3E-05x^4 + 0.0005x^3 + 0.0328x^2 - 0.0692x - 1.2527 \]

![Computer generated best fit 4th order polynomial equation.](image)

**Figure 2.13** A computer generated best fit 4th order polynomial equation.
2.4 MEASUREMENT ERRORS

All arch dimensions of patients were re-measured a month later. The mean differences between the two measurements were recorded and the standard deviation of the difference calculated. The measurement error was derived using the following formula according to Houston (1983).

\[ S_e = \sqrt{\frac{S_d^2}{2}} \]

\( S_e = \) Error of a single series of measurement  
\( S_d = \) Standard deviation of the difference between the replicate (Houston, 1983).

The index of reliability was the correlation between the repeat measurements which evaluated the contribution of random errors. In the absence of random errors, the index of reliability was equal to 1 (Houston, 1983).

2.5 STATISTICAL ANALYSES

2.5.1 Data storage and statistical software

Data were stored and analysed on a computerised spreadsheet software package (Microsoft Excel, Microsoft Corporation) designed for personal computers.

2.5.2 Statistics

A univariate analysis of categorical data was conducted using a Chi\(^2\) test without Yates correction. Parametric significance testing was carried out by unpaired t-tests for maxillary arch measurements, cephalometric variables, the
polynomial coefficients and anthropometric variables. Paired t-tests were used for
the analyses of errors of measurements of the arch dimensions, polynomial
coefficients, and the cephalometric variables. Non-parametric tests such as the
Wilcoxon Rank Sum test (Mann-Whitney U test) were used when the data were not
normally distributed. Linear regression was used to evaluate the relationship
between two numerical variables. Results of the linear regression were expressed
in terms of the correlation coefficient ($r$) and the ($p$) value. The Spearman rank
correlation ($r_s$) was used if the distribution of numerical variables was skewed, if
any outlying observations existed, or to describe the relationship between two
ordinal (or one ordinal and one numerical) characteristics.

All values in this thesis are presented as mean ± standard error of the mean,
unless otherwise stated. A ($p$) value of less than 0.05 was considered significant.
3 RESULTS

3.1 CHARACTERISTICS OF PATIENTS AND CONTROLS

3.1.1 Anthropometric data for patient and controls

The physical characteristics of the patients and controls are summarised in Table 3.1. There were 4 females in the patient group and 3 females in the control group. All patients and controls were Caucasians adults. Patients were significantly older (48 ± 2 vs 40 ± 2 years) and heavier (100 ± 3 vs 81 ± 3 kg) than control subjects. There was also a trend towards shorter stature in the patients, but the height difference did not reach statistical significance (173 ± 2 vs 176 ± 2 cm, p= 0.06). Figure 3.1 depicts the age distribution.

Table 3.1 Anthropometric data for patients and controls.

<table>
<thead>
<tr>
<th>Subjects</th>
<th>Patients</th>
<th>Controls</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>40</td>
<td>21</td>
<td></td>
</tr>
<tr>
<td>Age (Years)</td>
<td>49.0 ± 2.0</td>
<td>40 ± 2</td>
<td>&lt;0.005</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>173.0 ± 2.0</td>
<td>176 ± 2</td>
<td>0.060(NS)</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>99.0 ± 3.0</td>
<td>81 ± 3</td>
<td>&lt;0.005</td>
</tr>
<tr>
<td>BMI</td>
<td>33.3 ± 0.8</td>
<td>26 ± 1</td>
<td>&lt;0.005</td>
</tr>
</tbody>
</table>

Figure 3.1 Age distribution and mean age of patients and controls
3.1.2 Sleep data of patients

All patients had varying degree of OSA with the symptoms of snoring and excessive daytime sleepiness. Apnoea severity was defined by the RDI and min SaO₂. The RDI ranged from 5 to 101 with a mean of 32 ± 4 and the minSaO₂ ranged from 53% to 93% with a mean of 81% ± 2.

3.1.3 Questionnaire data of controls

A total of 35 subjects completed the questionnaire. Only 12 subjects who answered they “never snore”, and 9 subjects who answered that they “rarely snore”, i.e. less than once a week, were accepted as controls. Their ESS ranged from 0 to 10 with a mean of 4.7 ± 0.7. This value was within the normal range of 2-10. Two subjects who returned an ESS score of 10 rated their sleep quality as short, interrupted and slightly restless.

The remaining 14 subjects, who answered that they snore 1-2 times a week or more, were not suitable as controls even though their ESS score was within the normal range.

3.2 MAXILLARY MORPHOLOGY

3.2.1 Prevalence of crossbites and maxillary constriction

Twenty patients and one control had transverse discrepancies. Eight patients had bilateral crossbites, five had bilateral edge to edge cuspal relationship and the remaining seven had unilateral crossbite. The proportion of patients having crossbite was highly significant (p<0.001). Statistics on the prevalence of crossbite are shown in Table 3.2.
Table 3.2  Chi-squared ($\chi^2$) contingency table for posterior crossbites at 1° of freedom.

<table>
<thead>
<tr>
<th></th>
<th>Patients</th>
<th>Controls</th>
<th>Total</th>
<th>$\chi^2$</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>With crossbite</td>
<td>20</td>
<td>1</td>
<td>21</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Without crossbite</td>
<td>20</td>
<td>20</td>
<td>40</td>
<td>12.48</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Total</td>
<td>40</td>
<td>21</td>
<td>61</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

3.2.2 Palate cross-sectional tracings

The coronal shape of the palate at the 1st molar, 1st and 2nd premolar and canine region were traced from the profile gauge on to graph paper to provide a graphical illustration and comparison of the shape of the palate in patients and controls.

Figure 3.2  Coronal palate shape of casts of three random patients at the 1st molar, 1st and 2nd premolar, and canine region.
Figure 3.3  Coronal palate shape of casts of three random control subjects at the 1st molar, 1st and 2nd premolar, and canine region.

3.2.3 Linear measurements

There were significant differences in the inter-canine distance (P=0.005), the first inter-premolar distance (P=0.0046), the second inter-premolar distance (p=0.004), the inter-molar distance (P=0.025) and the maxillary depth (P=0.04) between the two groups. Patients had narrower inter-tooth distances and shorter maxillary depths. There were no significant difference in the palate heights. The ratio of the palatal height to the lateral dimensions, that is, the palatal index was only significantly different at the second inter-premolar (P=0.033) and the first inter-molar (P=0.03) regions, but not at the inter-canine (P=0.265) and the first inter-premolar (P=0.11) regions. The data are summarised in Table 3.3.
### Table 3.3  Maxillary arch measurements in patients and controls

<table>
<thead>
<tr>
<th>Maxillary arch Measurements</th>
<th>Patients N=40</th>
<th>Controls N=21</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inter-canine distance (mm)</td>
<td>32.00 ± 0.40</td>
<td>34.40 ± 0.40</td>
<td>&lt;0.005</td>
</tr>
<tr>
<td>1st Inter-premolar distance (mm)</td>
<td>36.00 ± 0.50</td>
<td>38.50 ± 0.40</td>
<td>&lt;0.005</td>
</tr>
<tr>
<td>2nd Inter-premolar distance (mm)</td>
<td>40.60 ± 0.50</td>
<td>43.10 ± 0.60</td>
<td>&lt;0.005</td>
</tr>
<tr>
<td>1st Inter-molar distance (mm)</td>
<td>45.90 ± 0.60</td>
<td>48.50 ± 1.00</td>
<td>&lt;0.050</td>
</tr>
<tr>
<td>Palatal height at canine (mm)</td>
<td>8.20 ± 0.40</td>
<td>8.05 ± 0.50</td>
<td>(NS)</td>
</tr>
<tr>
<td>Palatal height at 1st premolar (mm)</td>
<td>15.50 ± 0.50</td>
<td>15.00 ± 0.50</td>
<td>(NS)</td>
</tr>
<tr>
<td>Palatal height at 2nd premolar (mm)</td>
<td>20.00 ± 0.40</td>
<td>19.00 ± 0.50</td>
<td>(NS)</td>
</tr>
<tr>
<td>Palatal height at 1st molar (mm)</td>
<td>22.00 ± 0.40</td>
<td>21.20 ± 0.50</td>
<td>(NS)</td>
</tr>
<tr>
<td>Maxillary depth (mm)</td>
<td>33.60 ± 0.50</td>
<td>35.40 ± 0.60</td>
<td>&lt;0.050</td>
</tr>
<tr>
<td>Palatal index at canine</td>
<td>0.26 ± 0.01</td>
<td>0.23 ± 0.02</td>
<td>(NS)</td>
</tr>
<tr>
<td>Palatal index at 1st premolar</td>
<td>0.43 ± 0.02</td>
<td>0.39 ± 0.02</td>
<td>(NS)</td>
</tr>
<tr>
<td>Palatal index at 2nd premolar</td>
<td>0.49 ± 0.01</td>
<td>0.45 ± 0.02</td>
<td>&lt;0.050</td>
</tr>
<tr>
<td>Palatal index at 1st molar</td>
<td>0.47 ± 0.01</td>
<td>0.44 ± 0.02</td>
<td>&lt;0.050</td>
</tr>
</tbody>
</table>

The significant data are represented in Figure 3.4.
Figure 3.4  Mean ICD, 1st IPD, 2nd IPD and IMD of the patients and controls.

ICD = Inter-canine distance measured between centroids of maxillary canines.
IPD = Inter-premolar distance measured between centroids of first premolars.
IMD = Inter-molar distance between centroids of first maxillary molar.
3.2.4 Maxillary arch polynomial coefficients

Table 3.4 shows the comparison of the coefficients between the patients and the controls. The quadratic ($x^2$) term depicting the degree of tapering of the arch, was significantly different ($P=0.024$) between the two groups. This result and the previous results on inter tooth distances, when combined together, suggest that the arch form is more tapered in the patient group when compared to the controls.

Table 3.4 Mean maxillary archform polynomial coefficients

<table>
<thead>
<tr>
<th>Coefficient</th>
<th>Patients (N=40)</th>
<th>Controls (N=21)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>$X$</td>
<td>0.0100 ± 0.0190</td>
<td>-0.0400 ± 0.0400</td>
<td>0.49</td>
</tr>
<tr>
<td>$X^2$</td>
<td>0.0270 ± 0.0023</td>
<td>0.0200 ± 0.0020</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>$X^3$</td>
<td>9.1E-05 ± 5E-05</td>
<td>1.5E-05 ± 7E-05</td>
<td>0.40</td>
</tr>
<tr>
<td>$X^4$</td>
<td>2.9E-05 ± 3E-06</td>
<td>3.3E-05 ± 4E-06</td>
<td>0.54</td>
</tr>
<tr>
<td>Constant</td>
<td>0.4400 ± 0.0950</td>
<td>-0.5100 ± 0.1100</td>
<td>0.62</td>
</tr>
</tbody>
</table>
3.3 CEPHALOMETRIC VARIABLES

Table 3.5 summarises both the lateral cephalometric and postero-anterior cephalometric analyses for the patients and controls. Not all patients had x-rays, because the facilities were under renovation and, on recall, some patients did not arrive for the x-rays. Therefore, only 29 postero-anterior cephalograms and 30 lateral cephalograms were included in the analysis of patient data. All 21 controls had both lateral and postero-anterior cephalograms. The linear measurements were adjusted according to the magnification factor.

3.3.1 Lateral cephalometric variables

There were significant differences between patients and controls in the length of the maxilla (p=0.002) measured from ANS to PNS, and in the length of the mandible (p=0.001) measured from Go to Gn. In the patient group, both the maxilla and mandible were shorter in absolute terms when compared to the controls.

Patients had a greater mandibular plane to hyoid bone distance (p=0.012) and longer soft palates (p=0.002) than controls. Patients also had longer tongues (p=0.002) than controls. However, there was no significant difference in tongue height (p=0.38).

There was no significant difference between the two groups in the posterior airway space and the bony nasopharynx parameters as measured by the Aa-Pns and Ba-Pns distance.
### Table 3.5  Lateral cephalometric data of patients and controls

<table>
<thead>
<tr>
<th>Cephalometric Variables</th>
<th>Patients (N=30)</th>
<th>Controls (N=21)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>PAS</td>
<td>10.17 ± 0.46</td>
<td>10.45 ± 0.76</td>
<td>0.740</td>
</tr>
<tr>
<td>ANS-PNS</td>
<td>51.11 ± 0.59</td>
<td>54.85 ± 0.99</td>
<td>0.002</td>
</tr>
<tr>
<td>Aa-PNS</td>
<td>32.84 ± 0.60</td>
<td>32.90 ± 0.74</td>
<td>0.970</td>
</tr>
<tr>
<td>Ba-PNS</td>
<td>41.83 ± 0.56</td>
<td>43.05 ± 0.75</td>
<td>0.280</td>
</tr>
<tr>
<td>Go-Gn</td>
<td>72.36 ± 0.78</td>
<td>77.18 ± 1.15</td>
<td>0.001</td>
</tr>
<tr>
<td>MP-H</td>
<td>18.45 ± 1.20</td>
<td>14.44 ± 1.23</td>
<td>0.012</td>
</tr>
<tr>
<td>PNS-P</td>
<td>40.64 ± 0.90</td>
<td>36.90 ± 0.81</td>
<td>0.002</td>
</tr>
<tr>
<td>TG Ht.</td>
<td>37.20 ± 0.60</td>
<td>37.97 ± 0.62</td>
<td>0.380</td>
</tr>
<tr>
<td>TG Lt.</td>
<td>81.35 ± 1.10</td>
<td>75.21 ± 1.60</td>
<td>0.002</td>
</tr>
<tr>
<td>PNS-Pw</td>
<td>23.44 ± 0.80</td>
<td>24.85 ± 0.59</td>
<td>0.090</td>
</tr>
</tbody>
</table>

### 3.3.2 Postero-anterior cephalometric variables

In the transverse dimension, there was a significant difference between the two groups in the ratio of the maxillary width/ mandibular width (P=0.001) and the ratio of the maxillary width/ facial width (P=0.001). However, there was no significant difference in the ratio between the nasal width to the maxillary width between the two groups. These data are summarised in Table 3.6

### Table 3.6  Linear ratios derived from postero-anterior cephalograms analysis

<table>
<thead>
<tr>
<th>Linear measurements ratio</th>
<th>Patients (N=29)</th>
<th>Controls (N=21)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maxillary width / Mandibular width</td>
<td>0.68 ± 0.007</td>
<td>0.73 ± 0.012</td>
<td>0.001</td>
</tr>
<tr>
<td>Maxillary width / Facial width</td>
<td>0.44 ± 0.005</td>
<td>0.47 ± 0.005</td>
<td>0.001</td>
</tr>
<tr>
<td>Nasal width / Maxillary width</td>
<td>0.51 ± 0.009</td>
<td>0.48 ± 0.010</td>
<td>0.100</td>
</tr>
</tbody>
</table>
3.4 ANALYSES OF MEASUREMENT ERRORS

3.4.1 Analysis of errors of arch dimension measurements

Table 3.7 summarised the mean difference between the repeated measurements of 40 random maxillary casts, approximately a month apart.

Table 3.7 Mean differences between measurements, standard deviation of the difference, error and reliability index.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean diff.</th>
<th>Std.Dev.of diff.</th>
<th>Error</th>
<th>Reliability Index</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inter-canine width</td>
<td>0.05</td>
<td>0.110</td>
<td>0.077</td>
<td>0.998</td>
</tr>
<tr>
<td>1st Inter-premolar width</td>
<td>0.06</td>
<td>0.112</td>
<td>0.079</td>
<td>0.998</td>
</tr>
<tr>
<td>2nd Inter-premolar width</td>
<td>0.05</td>
<td>0.120</td>
<td>0.080</td>
<td>0.998</td>
</tr>
<tr>
<td>Inter-molar width</td>
<td>0.03</td>
<td>0.116</td>
<td>0.080</td>
<td>0.998</td>
</tr>
<tr>
<td>Maximum palatal Ht</td>
<td>0.03</td>
<td>0.187</td>
<td>0.130</td>
<td>0.996</td>
</tr>
<tr>
<td>Maxillary depth</td>
<td>0.09</td>
<td>0.200</td>
<td>0.140</td>
<td>0.998</td>
</tr>
</tbody>
</table>
The measurement errors were low and the index of reliability was high. The latter indicate that the random error was minor.

3.4.2 Analysis of errors of polynomial coefficients

To assess the errors involved in obtaining the polynomial coefficients, 30 randomly chosen casts from the patient group were photocopied again at 1:1 magnification and their co-ordinates re-determined. Differences between the polynomial coefficients derived from the first and second sets of data are summarised in Table 3.8. Errors were small, contributing less than 10% for all coefficients.

Table 3.8 Mean differences between repeated determination of polynomial coefficients, standard deviation of the differences, errors and reliability index.

<table>
<thead>
<tr>
<th>Coefficients</th>
<th>Mean difference</th>
<th>Std.Dev.of diff.</th>
<th>Error</th>
<th>Reliability Index</th>
</tr>
</thead>
<tbody>
<tr>
<td>x</td>
<td>0.0059</td>
<td>0.022</td>
<td>0.015</td>
<td>0.97</td>
</tr>
<tr>
<td>x² (x 10)</td>
<td>0.2100</td>
<td>0.240</td>
<td>0.160</td>
<td>0.98</td>
</tr>
<tr>
<td>x³ (x 10⁴)</td>
<td>0.0080</td>
<td>0.680</td>
<td>0.480</td>
<td>0.98</td>
</tr>
<tr>
<td>x⁴ (x 10⁵)</td>
<td>0.0200</td>
<td>0.050</td>
<td>0.030</td>
<td>0.91</td>
</tr>
</tbody>
</table>

3.4.3 Analysis of errors of cephalometric measurements

Fifteen lateral cephalograms and 15 postero-anterior cephalograms were randomly chosen from both groups. That is a total of 30 lateral cephalograms and 30 postero-anterior cephalograms were retraced and the measurements repeated one month apart. Table 3.9 summarises the repeated measurements, the calculated errors and the reliability index.
Table 3.9  Mean difference of repeated cephalometric measurements, standard deviation of the difference, error and the reliability index

<table>
<thead>
<tr>
<th>Variables</th>
<th>Mean Difference</th>
<th>Std. Dev. of diff.</th>
<th>Error</th>
<th>Reliability Index</th>
</tr>
</thead>
<tbody>
<tr>
<td>PAS</td>
<td>0.10</td>
<td>0.24</td>
<td>0.17</td>
<td>0.997</td>
</tr>
<tr>
<td>ANS-PNS</td>
<td>0.41</td>
<td>0.50</td>
<td>0.35</td>
<td>0.985</td>
</tr>
<tr>
<td>Aa-Pns</td>
<td>0.18</td>
<td>0.35</td>
<td>0.24</td>
<td>0.994</td>
</tr>
<tr>
<td>Ba-Pns</td>
<td>0.53</td>
<td>0.47</td>
<td>0.33</td>
<td>0.983</td>
</tr>
<tr>
<td>Go-Gn</td>
<td>0.25</td>
<td>0.41</td>
<td>0.28</td>
<td>0.997</td>
</tr>
<tr>
<td>MP-AH</td>
<td>0.20</td>
<td>0.38</td>
<td>0.26</td>
<td>0.996</td>
</tr>
<tr>
<td>PNS-P</td>
<td>0.42</td>
<td>0.53</td>
<td>0.37</td>
<td>0.990</td>
</tr>
<tr>
<td>TG Ht</td>
<td>0.22</td>
<td>0.36</td>
<td>0.25</td>
<td>0.990</td>
</tr>
<tr>
<td>TG Lt</td>
<td>0.47</td>
<td>0.56</td>
<td>0.40</td>
<td>0.990</td>
</tr>
<tr>
<td>PNS-Pw</td>
<td>0.30</td>
<td>0.45</td>
<td>0.32</td>
<td>0.990</td>
</tr>
<tr>
<td>Max. width</td>
<td>0.10</td>
<td>0.27</td>
<td>0.19</td>
<td>0.997</td>
</tr>
<tr>
<td>Mand. width</td>
<td>0.29</td>
<td>0.44</td>
<td>0.31</td>
<td>0.996</td>
</tr>
<tr>
<td>Facial width</td>
<td>0.32</td>
<td>0.47</td>
<td>0.33</td>
<td>0.996</td>
</tr>
<tr>
<td>Nasal width</td>
<td>0.50</td>
<td>0.50</td>
<td>0.36</td>
<td>0.980</td>
</tr>
</tbody>
</table>

The cephalometric variables that had the most variation were Ba-Pns and Ans-Pns. This outcome was due to the difficulty of consistently identifying points Ans, Pns and Ba. Nevertheless, errors were minor in both the lateral and postero-anterior cephalometric analyses and the high correlation between the two sets of readings indicated a high estimate of reproducibility.
3.5 RELATIONSHIP BETWEEN MAXILLARY ARCH DIMENSIONS AND POSTERIOR AIRWAY SPACE

Univariate regression analysis showed a significant correlation between the lateral dimensions of the maxilla and the posterior airway space (PAS) in the patient group. The correlation coefficient was significant at the canine (r=0.37, p<0.05), 1st premolar (r=0.41, p<0.05) and molar (r=0.37, p<0.05) region. The association of the palatal indices to PAS is not significant.

Table 3.10 summarises the correlation data and level of significance. Figures 3.7 to 3.9 show the scattergram plots of the significant correlations.

Table 3.10 Univariate correlations of maxillary arch dimensions with posterior airway space (PAS).

<table>
<thead>
<tr>
<th>Maxillary arch dimensions</th>
<th>Posterior airway space</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>N= 30</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Inter-canine width</td>
<td>r = 0.37</td>
<td>p &lt;0.05</td>
</tr>
<tr>
<td>1st Inter-premolar width</td>
<td>r = 0.41</td>
<td>p &lt;0.05</td>
</tr>
<tr>
<td>2nd Inter-premolar width</td>
<td>r = 0.23</td>
<td>p &lt;0.10</td>
</tr>
<tr>
<td>Inter-molar width</td>
<td>r = 0.37</td>
<td>p &lt;0.05</td>
</tr>
<tr>
<td>Canine palatal index</td>
<td>r = -0.21</td>
<td>ns</td>
</tr>
<tr>
<td>1st premolar palatal index</td>
<td>r = -0.16</td>
<td>ns</td>
</tr>
<tr>
<td>2nd premolar palatal index</td>
<td>r = -0.14</td>
<td>ns</td>
</tr>
<tr>
<td>Molar palatal index</td>
<td>r = -0.18</td>
<td>ns</td>
</tr>
<tr>
<td>Maxillary depth</td>
<td>r = 0.21</td>
<td>ns</td>
</tr>
</tbody>
</table>
Results

Figure 3.7 Scattergram plot of inter-canine distance and posterior airway space (PAS)

Figure 3.8 Scattergram plot of 1st inter-premolar distance and posterior airway space (PAS)

Figure 3.9 Scattergram plot of 1st inter-molar distance and posterior airway space (PAS)
3.6 RELATIONSHIP BETWEEN MAXILLARY ARCH DIMENSIONS AND APNOEA SEVERITY

No significant correlations were found between maxillary arch dimensions and apnoea severity, the latter, defined by the RDI and the minSaO₂. Table 3.11 summarises the correlation coefficients \( (r) \) for the relationship between the different maxillary arch dimensions, RDI and minSaO₂.

<table>
<thead>
<tr>
<th>Variable</th>
<th>RDI</th>
<th>minSaO₂</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>ICD</td>
<td>( r = 0.150 )</td>
<td>( r = 0.005 )</td>
<td>ns</td>
</tr>
<tr>
<td>1st IPD</td>
<td>( r = -0.050 )</td>
<td>( r = 0.050 )</td>
<td>ns</td>
</tr>
<tr>
<td>2nd IPD</td>
<td>( r = 0.060 )</td>
<td>( r = 0.005 )</td>
<td>ns</td>
</tr>
<tr>
<td>IMD</td>
<td>( r = -0.050 )</td>
<td>( r = 0.200 )</td>
<td>ns</td>
</tr>
<tr>
<td>ICD palatal index</td>
<td>( r = 0.010 )</td>
<td>( r = -0.070 )</td>
<td>ns</td>
</tr>
<tr>
<td>1st IPD palatal index</td>
<td>( r = 0.090 )</td>
<td>( r = -0.030 )</td>
<td>ns</td>
</tr>
<tr>
<td>2nd IPD palatal index</td>
<td>( r = 0.148 )</td>
<td>( r = 0.063 )</td>
<td>ns</td>
</tr>
<tr>
<td>IMD palatal index</td>
<td>( r = 0.040 )</td>
<td>( r = -0.070 )</td>
<td>ns</td>
</tr>
</tbody>
</table>
3.7 RELATIONSHIP OF CEPHALOMETRIC MEASUREMENTS AND APNOEA SEVERITY

Univariate regression analysis showed no significant association between maxillary length (ANS-PNS), soft palate length (PNS-P), tongue height (TG Ht), tongue length (TG Lt), retro-palatal space (PNS -PW) and posterior airway space (PAS) and the severity of apnoea. The latter was defined by RDI and minSaO₂.

Similarly, there was no significant association between mandibular plane to hyoid bone distance (MP-H), ratio of maxilla to mandibular width (Max/Mand), ratio of nasal width to maxillary width (Nasal/Max), and mandibular body length (Go-Gn), and the severity of apnoea. Table 3.12 summarises the data.

Table 3.12 Correlation of cephalometric measurements and apnoea severity as defined by RDI and minSaO₂.

<table>
<thead>
<tr>
<th>CEPH. Variables</th>
<th>RDI</th>
<th>p Value</th>
<th>MIN.SAO₂</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Max/ Mand</td>
<td>r = -0.300</td>
<td>p =0.1</td>
<td>r = 0.01</td>
<td>ns</td>
</tr>
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<td>ns</td>
<td>r = 0.33</td>
<td>p =0.1</td>
</tr>
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<td>p =0.1</td>
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</tr>
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<td>r = 0.04</td>
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</tr>
<tr>
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</tr>
<tr>
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<tr>
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<td>ns</td>
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<td>ns</td>
</tr>
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4 DISCUSSION

Although anatomical and neuromuscular abnormalities have been implicated in the pathogenesis of OSA, the relative contribution of these abnormalities and their interactions requires further definition. Numerous studies with various imaging techniques, especially cephalometry, have revealed that craniofacial abnormalities occur commonly in patients with OSA (Riley et al., 1983; Partinen et al., 1988; Pepin et al., 1992). It is thought that these abnormalities result in upper airway narrowing thereby predisposing to airway closure during sleep. A recent study has indicated that the shape and dimensions of the maxilla are important determinants of nasal airway resistance and OSA severity in patients with Marfan's syndrome (Cistulli, 1994). In addition, an uncontrolled study has suggested that there is a high prevalence of maxillary constriction in the general OSA population (Palmisano et al., 1994). The findings of these last two studies have led to the hypothesis that patients with OSA have different maxillary morphology when compared to non-snoring and non-apnoeic controls and that the shape and size of the maxilla has a role to play in the development of OSA. Therefore, the aim of this investigation was to test this hypothesis.

4.1 DEFINITION OF MAXILLARY CONSTRICION

Maxillary constriction is not well defined in the orthodontic literature. Furthermore, the terminology is used interchangeably to describe either dental arch constriction or skeletal base constriction, or both. In the diagnosis of lateral dimensional discrepancies there is a tendency to focus only on the intercuspation of teeth, and not enough attention is paid to the morphology of the palatal vault and the inclination of the dental alveolar processes and teeth.
The critical diagnostic feature of skeletal constriction is the width of the palatal vault and its relationship to the corresponding width of the maxillary inter-tooth distance. Proffit (1993) states that skeletal maxillary constriction is distinguished by:

1. a narrow palatal vault, and
2. a big discrepancy between the width of the palatal vault and its corresponding inter-tooth distance.

In addition to these criteria, the absence of cross-bites does not necessarily mean that the maxilla is not constricted. Situations can arise where both upper and lower alveolar arches could be constricted simultaneously or dento-alveolar compensation is successful in masking the lateral discrepancy.

4.2 MAXILLARY MORPHOLOGY IN OSA

4.2.1 Prevalence of crossbites and skeletal maxillary constriction in OSA patients

In their assessment of orthodontic abnormalities in OSA patients, Palmisano et al. (1994) found a high prevalence (62%) of maxillary constriction in a sample of 35 patients. Constriction was diagnosed by the presence of posterior crossbites. No distinction was made between dental or skeletal constriction and no postero-anterior cephalograms were used to assess the skeletal width of the maxilla. Furthermore, their patient sample also had a high prevalence of Class III malocclusion and lacked a control group.

The prevalence of posterior crossbites in the current study was significantly higher in patients (50%) than in the controls (5%). Since it is possible to have skeletal maxillary constriction and simultaneous maxillary and mandibular arch constrictions without the presence of crossbites, the above result is considered to be conservative. In addition, the mean ratio of the maxillary width to mandibular
width, derived from the postero-anterior cephalometric analysis, was significantly smaller in the patients when compared to that of the controls. Since the widths of the mandibles for all patients and controls were within the normal range, when assessed against the standard inter-tooth distances (Moyers et al., 1976), this finding clearly demonstrates that the maxillae of these OSA patients were narrower than those of the controls. The present findings support the hypothesis that skeletal maxillary constriction is a common feature in patients with OSA.

4.2.2 Maxillary archform in OSA patients

The results clearly showed that, as a group, the patients in this study have significantly narrower archforms when compared with normal controls. This result was true for all the four measures of arch width; namely, the inter-canine width, the first inter-premolar width the second inter-premolar width, and the first inter-molar width. Differences between patients and controls varied from 2.4 to 2.5 mm in all the above measurements. In addition, the patients also had a significantly shorter maxillary arch length indicating that, the maxilla was not only constricted, but was also shorter in length. These result were similar to the findings of Cistulli (1994) in OSA patients with Marfan's syndrome.

Studies by Sinclair and Little (1983, 1985) showed that mandibular arch width and arch length decrease with time and can be quite significant from 20 to 30 years of age. Thereafter, the constrictive trend is much less. Since it is assumed that alignment of the lower arch serves as a template around which the upper arch develops, it is also assumed that the upper arch width and length would also decrease with age. However, although the patients and controls in the present study were not exactly matched for age, the age difference was not significant with only 3 controls under the age of 30. Hence, the constriction of the arch width and decrease in arch length observed in the patients could not be due to ageing alone. It is more likely that the constriction of the dental arch is a reflection of the skeletal constriction, as shown by the difference in the ratio of the maxillary and mandibular widths from the postero-anterior cephalogram analysis.
The shape of the dental arches has been described qualitatively by many terms (Biggerstaff, 1972). However, in order to describe the archform in more detail, fourth order least squares polynomial regression equations were used because they give the most accurate and reproducible likeness of archform and arch length (Sanin et al., 1970). Furthermore, the lateral dimensions of the maxilla were measured at specific points, whereas an equation described a continuous curve, providing a more precise outline of the lateral dimension of the whole dental arch. This is the first study to use fourth order polynomial equations to describe maxillary archform in OSA patients.

The results showed a significant difference in the mean quadratic ($x^2$) coefficients between the two groups. The patients had a larger mean ($x^2$) coefficient and thus more tapered archforms. This finding further supports the earlier results that the maxillae of OSA patients are narrower and constricted.

4.2.3 Maxillary palate shape in OSA patients

In his study of patients with Marfan’s syndrome with OSA, Cistulli (1994) used the maximum palatal height as the only determinant of palate height. Therefore, the palatal indices he obtained for the canine and first premolar regions may not be accurately descriptive of the shape, or "degree of constriction", at these regions. The maximum palatal height of the hard palate was defined by Cistulli (1994) as the maximum perpendicular distance from the occlusal plane of the maxillary arch to the hard palate. However, there was no description of the method by which the measurement was determined. Moreover, in his study there were significant differences in all the palatal indices between the patients and controls. His result was to be expected since there was already a significant difference in the inter-tooth distances, but the maximum palatal heights were of the same value.
In the present study there were no differences between the palatal heights at the canine, 1st and 2nd premolar and 1st molar regions between the patients and controls. However, the palatal indices were significantly different at the 2nd inter-premolar and 1st molar regions. These data suggest that the height of the palate, by itself, is not a reliable indicator of maxillary constriction and the width must also be taken into account when assessing constriction. From the findings of this study, it is concluded that the palatal index is more descriptive of the constriction towards the posterior portion of the palate. Therefore, the results suggest that the shapes of the palate at the second premolar and first molar region were quite different between the two groups.

Recently, Guilleminault et al. (1995) reported a high odds-ratio (95%) for the presence of a high narrow hard palate in the immediate family relatives of index cases with OSA. They classified the hard palate into different classes of (1) high arch and narrow, (2) mid-placed; or (3) low placed using the dorsum of the tongue as a reference plane and at a mouth opening of 20-degree angle. This method is subjective and difficult to standardise. Nevertheless, their study further reinforces the likely importance of maxillary constriction in OSA patients.

In addition, an abstract from the Stanford Sleep Disorders Clinic and Research Center (Kushida et al., 1996) reported the development of a clinical morphometric model for the prediction of OSA:

$$P + \left( Mx - Mn \right) + 3 \times OJ + 3 \times (BMI - 25) \times (NC - BMI)$$

where $P =$ palatal height, distance from the dorsum of the tongue at the median lingula sulcus to the highest point of the palate measured with the tongue in a relaxed position and the maxillary incisor tips subtending an angle of 20 degrees from the mandibular condyle, $NC =$ neck circumference (cm), $Mx =$ Distance between the mesial surfaces of the crowns of the maxillary second molars, $Mn =$ Distance between the mesial surfaces of the crowns of the mandibular second molars, $OJ =$ overjet, horizontal overlap of the crowns of the maxillary and mandibular central incisors, $BMI =$ Body Mass Index.
Kushida *et al.* have prospectively studied a total of 300 patients to test the clinical utility of the model in predicting OSA and found that patients with values equal to or more than 70 typically had OSA. They claimed that the sensitivity of the model was 98%, the specificity was 100%, and the predictive value of a positive test was 100% at the level of $p<0.0001$. This significance demonstrates that the lateral dimensions of the maxilla and the mandible, and the shape of the maxilla, are important parameters to consider and may have contributing roles to play in the development of OSA.

### 4.3 RELATIONSHIP BETWEEN MAXILLARY MORPHOLOGY AND APNOEA SEVERITY

OSA has a multifactorial aetiology with numerous anatomical and neuromuscular elements interacting with each other. Obesity occurs in a majority of patients with OSA and is considered to be a major risk factor for its development. Patients with OSA have been shown to have big necks when compared with both non-apnoeic snorers and weight matched controls (Katz *et al.*, 1990; Hoffstein and Mateska, 1992). Neck circumference also correlates better than BMI with apnoea severity (Davies *et al.*, 1992). However, not all patients with OSA are obese and some of these non-obese patients have obvious abnormal craniofacial structures (Ran *et al.*, 1990; Davies and Iber, 1983; Coccagna *et al.*, 1976; Conway *et al.*, 1977). Lateral cephalometric abnormalities such as posterior air space (Partinen *et al.*, 1988), hyoid position (Bacon *et al.*, 1988), mandible position (Rivlin *et al.*, 1984) and soft palate and tongue size (Strelzow *et al.*, 1988) have all been shown to be significant determinants of apnoea severity.

Cistulli’s, 1994 study of OSA patients with Marfan’s syndrome showed that maxillary constriction correlates with nasal resistance (after decongestion) and apnoea severity. It was thought that the high arched palate might be associated with a relatively narrow nasal passage resulting in a higher nasal resistance.
However, the relationship between nasal resistance and OSA is complex and not well understood. Numerous studies have shown that acute increases in nasal resistance can induce, or exacerbate, sleep-disordered breathing (Olsen et al., 1981; Zwillich et al., 1981; Lavie et al., 1983; Suratt et al., 1986). Nevertheless, the relevance of chronically increased nasal resistance in the pathogenesis of OSA is unclear. It was also hypothesized that a constricted maxilla would also cause a low tongue posture, resulting in retroglossal narrowing, and further contributing to a narrow airway and a predisposition to collapse of the airway.

In the present study, no significant correlations were found between all the maxillary arch measurements and the apnoea severity as defined by the RDI and minSaO₂. This result does not concur with the result of the study of patients with Marfan’s syndrome (Cistulli, 1994) where it was shown that there was a significant correlation (r=0.63, p<0.05) between the palatal index and the apnoea severity. One explanation for the discrepancy between the two results could be that a basic difference exists in the nature and location of the maxillary constriction in patients with Marfan’s syndrome compared to those of the OSA population studied herein. The results in the present study showed that the degree of severity of constriction of the maxilla is located more towards the posterior region. In patients with Marfan’s syndrome the constriction appears to be more pronounced and tends to extend up to the inter-canine region, although this feature has not been specifically studied. Hence, it is logical to infer that the dimension of the nasal valve is affected by this constriction in the canine region in patients with Marfan’s syndrome, which in turn, may well cause the increase in nasal resistance as evident in the study by Cistulli (1994).

Another reason for the lack of correlation between the maxillary dimensions and apnoea severity of the current study is that there is a considerable night-to-night variation of the RDI and minSaO₂, especially in the mild range of apnoea severity (Stradling and Mitchell, 1989). This night-to-night variability may influence the results of the correlation. In addition, it is likely that the sample size is too small to show the correlation between maxillary measurements and apnoea severity. The result of this study suggests that the lateral dimensions of the maxilla
on its own are not sensitive or predictive enough to have an effect on apnoea severity. However, it does not preclude a role in a morphometric model with other variables such as overjet, neck circumference and BMI in the prediction of OSA (Kushida et al., 1996). Further investigations are required to determine the mechanism by which maxillary constriction contributes to the development of OSA.

4.4 CRANIOFACIAL ABNORMALITIES AND OSA

4.4.1 Cephalometry in Sleep Apnoea research

Cephalometry has been used extensively in the fields of orthodontics and anthropology to record craniofacial form. Recently, it has been suggested that lateral cephalometry could be an adjunctive procedure for assessing craniofacial patterns and soft tissue airway structures associated with OSA (deBerry-Borrowiecik et al., 1988; Strelzow et al., 1988; Lyberg et al., 1989; Tangugsorn et al., 1995). Different head orientation techniques such as natural head position, or the more conventional horizontal Frankfort plane orientation, were used in different studies. Some studies (Yildirim et al., 1991; Pracharktam et al., 1994; Pae et al., 1994) compared the cephalometric variables in the erect and supine position. Interestingly, the results of these studies were controversial although a supine cephalogram may provide more physiologic data since it is obtained in the usual sleeping position. However, in all studies the radiographs were taken in the awake state and, according to Hudgel et al. (1989), the measurements of the upper airway calibre in the awake state do not predict upper airway resistance during sleep. Despite all these drawbacks and the obvious limitations of a two dimensional method, the upright cephalogram remains the most popular clinical tool to assess upper airway size for preliminary diagnostic purposes.
4.4.2 Lateral cephalometric analyses in OSA patients

Table 4.1 summarises the comparison of some of the cephalometric variables from the current investigation with past studies. The landmarks used were identical.

4.4.2.1 Maxillary length

Tangugsorn et al. (1995), Strelzow et al. (1988), Andersson and Brattstrom (1991), and Bacon et al. (1990) found that the length of the maxilla was significantly shorter in their patients than in the control subjects. The maxillary length of patients in this study was also significantly shorter compared to the controls and hence in agreement with the above studies. Furthermore, the maxillary depth measurements from the dental casts in the present study were also shorter in the patients than the controls and, therefore, in accordance with the cephalometric analysis.

4.4.2.2 Mandibular body length, mandibular plane to hyoid bone, soft palate length.

Andersson and Brattstrom (1991) and Strelzow et al. (1988) found a significantly shorter mandibular body length, defined by the distance between Go and Gn, in their patient sample. The present study also found a significantly shorter mandibular body in the patients, as defined by the same landmarks. Studies by Riley et al. (1983); Jamieson et al. (1986); Lowe et al. (1986) and Lyberg et al. (1989), all reported a significant increase in the distance of the hyoid bone to the mandibular plane. The length of the soft palate was also significantly longer in the patient sample in studies by Tangugsorn et al. (1995) and Lyberg et al. (1989). The patients in the present study also had a longer mandibular plane to hyoid distance as well as longer soft palates.
Table 4.1  Comparison of cephalometric variables with previous studies, differences are significant p<0.05

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<tr>
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<td>shorter in pat</td>
<td>shorter in pat</td>
<td>shorter in pat</td>
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<td>NA</td>
<td>shorter in pat</td>
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</tr>
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<td>Aa-PNS</td>
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</tr>
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<td>NA</td>
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<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>PAS</td>
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<td>shorter in pat</td>
<td>NA</td>
<td>shorter in pat</td>
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<td>No diff</td>
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</tr>
<tr>
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<tr>
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</table>

NA = not applicable  
Diff. = significant difference  
Pat. = patients
4.4.2.3 Bony and soft tissue pharynx dimension

Tangugsorn et al. (1995) and Bacon et al. (1990) showed a decrease in the horizontal dimension of the bony pharynx in their patients defined by the measurements from PNS to Ba and PNS to Aa. Andersson and Brattstrom (1991) also found a decrease in the linear measurement between PNS to Pw in their patient sample. The present study does not show significant differences in these measurements between the patients and controls. A possible explanation is that, the sample size was too small in comparison with the other studies. This conjecture is further supported by the fact that a study with a small sample size of 30 patients and 12 controls by deBerry-Borowiecki et al. (1988) also reported no significant difference in the PNS to Aa measurements.

The significance of a shorter maxilla in the antero-posterior dimension in patients with OSA is not clear. Bacon et al. (1990) explained that, together with the reduced skeletal dimensions of the pharynx, it may contribute to posterior facial compression and hence cause airway narrowing and a predisposition for airway collapse. The situation can be worsened if the soft palate is elongated as well. A shorter maxilla also means a shorter nasal cavity, but how this short maxilla relates to OSA is not known. Further research is needed to determine the relevance of these findings.

4.4.2.4 Posterior Airway Space

The PAS was first described by Riley et al. (1983). It is defined as the distance between the posterior pharyngeal wall and the dorsal surface of the base of the tongue, measured on the line that intersects point Go and B point. A narrow PAS is reported to be associated with OSA by numerous authors (Jamieson et al., 1986; Andersson and Brattstrom, 1991; Lyberg et al., 1989; Tangugsorn et al., 1995). The present study, however, found no significant difference in the PAS between the patients and controls. This finding is in agreement with studies by Yildirim et al. (1991); and Maltais et al. (1991) which also showed no significant
difference in the PAS between the patients and controls. The most likely explanation for the lack of a significant difference of PAS between patient and control is that a Type II error had occurred. The current study, and those investigators who found no difference in the PAS measurement, possibly had too small a sample size to detect a true difference.

The validity, reliability and reproducibility of the variable, PAS has been questioned (Miles et al., 1996). In the first instance, it is a two dimensional estimate of a three-dimensional structure. Secondly, it depends on the position of the tongue, a dynamic structure at the time of the exposure to the x-ray, which is likely to be affected by even subtle changes in head posture (Yildirim et al., 1991). On the other hand, Pracharktam et al. (1994), concluded from their study that upright cephalometric evaluation of the morphology of the upper airway was found to provide the same discriminant information as supine cephalometric evaluation.

4.4.2.5 Tongue length and tongue height

Lowe et al. (1986), Strelzow et al. (1988), and deBerry-Borowiecki et al. (1988), reported longer tongues in their patients. However, the study by Lowe et al. 1986 defined the tongue length differently from the other two studies. This present study, using the same landmarks as defined by Lowe et al. (1986), also found that the patients had significantly longer tongues. In the present study, there is no difference in the tongue heights between the patients and controls.

Miles et al. (1996) performed a qualitative and meta-analysis of the OSA and orthodontic literature to examine the foundation for any relationship between craniofacial structure and OSA. The analyses revealed 32 review articles, 16 case reports and 95 sample studies. He found that only one of these studies satisfied all of the qualitative criteria for a well designed study. He also found that the most consistent, strong effect sizes with the highest potential diagnostic accuracies were for the variables, mandibular plane to hyoid, mandibular plane angle, and mandibular body length. Only mandibular body length demonstrated a clinically significant diagnostic accuracy for OSA. However, he cautioned that association
does not necessarily imply causality and the reason for this positive association is most likely due to differing magnification factors and other methodologic differences among studies. The present study, after having taken magnification factors into consideration, showed that the mandibular length, defined by the landmark Go to Gn, was also significantly shorter in patients than the controls.

4.4.3 Postero-anterior cephalometric analysis of OSA patients

The present study is the first to use postero-anterior cephalometry in OSA research. It was found that the maxillary/mandibular width ratios in the patients were significantly smaller than in the controls. This finding means that the constriction is truly skeletal and not dental, at least at the inter-molar region, which is the widest part of the maxilla where the measurements were recorded.

In the practice of orthodontics, the primary aim for obtaining a postero-anterior cephalogram is to evaluate the severity and degree of asymmetry, or transverse deficiency. The head is fixed by a cephalostat so that the central x-ray beam penetrates the skull of the patient in a postero-anterior direction and perpendicularly bisects the transmeatal axis. The Frankfort horizontal relationship of the head with the floor must be maintained during this procedure because any tilting of the head will affect the vertical dimensions. In the current study, whether the head was tilted or not was not crucial because only width measurements were recorded. According to a study by Ishiguro et al. (1976) a change of ± 10 degrees of up-down movement, or right-left rotation, is less than the method error and is, therefore, a negligible factor in width measurements. Because of the individual variation in facial depth the image magnification of each film will be different. Furthermore, there will be an overlap of all the anatomical structures making it quite impossible to define a particular cross sectional region. In order to circumvent these problems it was decided to use ratios of width measurements for comparisons between the patients and controls. This also meant that the breadth measurement would have to be at the widest point of the structure to be measured.
Anatomically, the floor of the nasal cavity is also the roof of the mouth. Therefore, the ratio of nasal width to maxillary width should not be significantly different between the patients and controls. The results of the present study support this inference.

4.5 RELATIONSHIP OF MAXILLARY ARCH DIMENSIONS AND POSTERIOR AIRWAY SPACE

The current study showed that there is a correlation between the lateral dimension of the dental arch and posterior airway space in the patient group. The hypothesis is that a constricted upper arch leads to a constricted oral space which may cause a low tongue posture. The low tongue posture in turn, results in retroglossal narrowing. Previous studies have reported decreased PAS in OSA patients (Riley et al., 1983; Jamieson et al., 1986; Tangugsorn et al., 1995; Andersson and Brattstrom, 1991). However, the present study did not detect a decrease in the PAS in the patients compared to the controls. The most likely explanation is that the sample size was not large enough to detect the difference. Nevertheless, a low tongue posture has been associated with a constricted maxilla and posterior cross bites in individuals who suffered from chronic upper airway obstruction (Subtelney, 1954; Rickëtts, 1958). In addition, a low position of the hyoid bone also reflects a low tongue position (Solow et al., 1993).

4.6 RELATIONSHIP OF CEPHALOMETRIC VARIABLES AND APNOEA SEVERITY.

Numerous authors have reported that various cephalometric measurements correlate with the degree of OSA. Specifically, Davies and Stradling (1990) reported significant univariate correlations between oxygen "saturation dip rate" and hyoid position. However, the results of the present study did not identify a significant correlation between the cephalometric variables and apnoea severity.
One reason could be the small sample size of patients or that the “noise” of the scoring of apnoea severity could have affected the result.

Similarly, Partinen et al. (1988) found that PAS was a significant predictor of apnoea, while Cistulli (1994) in his study of OSA patients with Marfan's syndrome, also reported a significant correlation between PAS and minSaO₂. The current study, however, did not show any correlation between PAS and apnoea severity defined by RDI and minSaO₂. This outcome is hard to explain other than to conclude that sample size was not large enough to show any correlation.

4.7 RELATIONSHIP OF FACIAL DEVELOPMENT AND OSA

The orthodontic literature highlights the occurrence of similar craniofacial abnormalities in young patients who suffered from chronic upper airway obstruction (Andersson and Brattstrom, 1991; Jamieson et al., 1986). The reported abnormalities are similar to those present in relation to OSA patients. Commonly reported abnormalities are increased craniocephalic angulation, decreased mandibular size, retrognathia and a steep mandibular plane. Oral breathing, consequent to nasal obstruction, has been implicated to cause a modification of head posture, which may influence facial development (Solow and Kreiborg, 1977) and dentofacial growth (McNamara, 1973). Other authors have postulated that abnormal oral pressure during respiration hinders palatal descent resulting in adverse maxillary development (Linder-Aronson and Bachstrom, 1960).

The effect of altered respiration on craniofacial growth has also been studied in animal models. Harvold et al. (1973) occluded the nasal passages of nine growing rhesus monkeys, forcing them to breathe orally, and compared their growth radiographically to that of a group of nine control monkeys. The occluded group showed significantly increased facial height, decreased maxillary dental arch length, decreased maxillary and mandibular inter-canine distance, and alteration of tongue and mandibular morphology. Miller et al. (1978, 1980, 1982) conducted
electromyographic studies on the neuromuscular changes associated with the morphologic changes in different monkeys who were forced to breathe orally. Miller et al. found new neuromuscular patterns with rhythmic recruitment of specific craniofacial muscles. Removal of the obstructions often reversed morphologic changes that had occurred during the oral respiration period, but the response was quite variable. Some of the monkeys did not change their newly acquired neuromuscular patterns at all (Miller et al., 1984). These results are of considerable interest because they provide a plausible explanation for the evolution of OSA in humans. The results of the animal experiments lead to an interesting hypothesis that factors present early in life might influence facial development in a way that predisposes to OSA in later life, particularly if there is additional obesity. This concept is supported by Guilleminault et al. (1989) who showed that cephalometric abnormalities accounted for persisting snoring and upper airway obstruction in adolescents who had undergone tonsillectomy and adenoidectomy before puberty for upper airway obstruction. Furthermore, there is some evidence that the facial shape may to some extent be inherited and could account for some familial aggregation of sleep apnoea and snoring, particularly in the non-obese (Redline et al., 1992; Douglas et al., 1993).

The significance of the above observations lies not only in the potential to treat the underlying craniofacial abnormalities but, more importantly, there is a possibility that the early detection and treatment of children at “high risk” of developing OSA may prevent the disorder. This concept needs to be explored further with well designed studies in children with OSA.

4.8 TREATMENT IMPLICATIONS

The current treatment of choice for OSA is nasal CPAP (Sullivan et al., 1981). It is a very effective treatment with few side effects, but it does not cure the underlying condition and is also associated with significant rates of non-compliance. With the recent identification of a potential role of maxillary constriction in the development of OSA it has been proposed that maxillary expansion may be a beneficial treatment for OSA. Rapid maxillary expansion has the potential to improve or normalise upper airway dimensions and has been
suggested as a novel treatment modality for mild to moderate OSA (Cistulli et al., 1994).

Preliminary data indicate that rapid maxillary expansion can result in complete resolution of symptoms and return the RDI back into the normal range (Cistulli et al., 1996; Palmisano et al., 1996). These data strongly suggest a causative association between maxillary constriction and OSA, adding validity to the results of the current study.

4.9 STUDY LIMITATIONS

1) Attempts at recruitment and matching of controls for age, weight, height and sex met with only limited success. However, this difficulty does not reduce the validity and importance of the results reported in this study.

2) Ideally, the controls should have undergone overnight polysomnography to verify the absence of snoring and sleep apnoea. This diagnostic test was not feasible for all controls because of the expense of the procedure. Although screening by questionnaire cannot be completely accurate, the method used in this study has been validated and proven to be reliable in other studies.

3) The radiographs were taken with two separate machines and by different personnel. Nevertheless, the magnification factors were taken into consideration and even if there were minor differences these would not have significantly influenced the results.

4) As in other studies using cephalometry, this study has the limitation of a two-dimensional technique assessing three-dimensional structures. In addition, these radiographs were taken in the awake and erect state. Therefore, the results must be interpreted with these limitations in mind.

5) Assessment of dynamic structures of the airway, such as the tongue and soft palate, presents many difficulties.
5 SUMMARY AND CONCLUSIONS

A number of observations suggest a link between maxillary morphology and obstructive sleep apnoea (OSA). Firstly, maxillary constriction is associated with high nasal resistance and mouth breathing, which are features typically seen in OSA patients. Secondly, patients with Marfan’s syndrome characteristically have maxillary constriction and have been shown to have a high prevalence of OSA (Cistulli, 1994). Recent findings suggest that the shape and dimensions of the maxilla are important determinants of nasal airway resistance and OSA severity in patients with Marfan’s syndrome (Cistulli, 1994). Thirdly, an uncontrolled study (Palminsano et al., 1994) has suggested that there is a high prevalence of maxillary constriction in typical OSA patients. These findings have led to the hypothesis that patients with OSA have different maxillary morphology compared to non-snoring, non-apnoeic subjects and that the size and shape of the maxilla play an important role in the development of OSA. The following aims were used to test this hypothesis.

1) To determine the prevalence of maxillary dental and skeletal constriction in a random sample of OSA patients using information from dental casts and postero-anterior cephalograms.

2) To compare the dental archform between OSA patients and normal controls using polynomial equations and inter-tooth measurements.

3) To compare the shape of the palate between the two groups using palatal indices.

4) To examine the relationships between maxillary skeletal and dental arch morphology and apnoea severity. This was conducted by comparing the relationship between maxillary arch dimensions and cephalometric variables with the Respiration Disturbance Index (RDI) and the minimum saturation of oxygen \((\text{minSaO}_2)\) using univariate regression analysis.

The patient sample consisted of 40 randomly selected patients. There were 36 males and 4 females with a mean age of 49 ± 2 years and a mean Body Mass Index (BMI) of 33.3 ± 0.8. All patients had overnight polysomnography and were diagnosed with OSA. The definition of OSA is an RDI ≥ 5. The control group
consisted of 21 subjects comprising 18 males and 3 females with a mean age of 40 ± 2 and a mean BMI of 26 ± 1. The controls were selected with a validated questionnaire to exclude snoring and excessive daytime sleepiness, the major symptoms of OSA. Dental casts were obtained for all patients and controls. A total of 51 lateral cephalograms (30 patients, 21 controls) and 50 postero-anterior cephalograms (29 patients, 21 controls) were hand traced and analysed. Double determinations were used to check measurement errors and the index of reliability was more than 98% for all measurements.

The results indicate that the prevalence of posterior crossbites was significantly higher in OSA patients (50%) than in the controls (5%). In addition, the postero-anterior cephalogram analysis showed that patients have narrower maxillae than controls.

Patients were also found to have narrower arch forms than controls. There were significant differences between the inter-canine width (p<0.005), first inter-premolar width (p<0.005), second inter-premolar width (p<0.004) and first inter-molar width (p<0.05). Interestingly, there were no significant differences between the palatal heights at the canine, first and second premolars and first molar regions. However, the palatal indices were significantly different at the second premolar (p<0.05) and first molar (p<0.05) regions. This finding suggests that the difference in shape of the palate was more towards the posterior region of the maxilla. In addition, not only were the dental archforms narrower in patients, they were also shorter (p<0.05).

This was the first study to use fourth order polynomial equations to describe maxillary archform in OSA patients. Archform coordinates were plotted on to overlay graph paper from the dental casts and these data were used by a computer to generate the equations. The results showed that patients had more tapered arch forms than control subjects.

In this study univariate regression analysis revealed no significant correlation between the maxillary arch dimensions and apnoea severity. Similarly, there was no significant correlation between the cephalometric variables and apnoea severity. Apnoea severity was defined by RDI and minSaO₂. There were,
however, significant correlations between the lateral dimensions of the maxilla at the canine \((r=0.37, p=0.04)\) premolar \((r=0.41, p=0.02)\), and molar \((r=0.37, p=0.04)\) regions and the posterior airway space (PAS) in the OSA patients. This investigation suggests that maxillary constriction could influence tongue posture which may, in turn, affect PAS.

The results of this study clearly demonstrate that maxillary constriction is a common feature in OSA patients. Whilst a direct relationship between maxillary measurements and sleep apnoea severity was not determined in this study, it does not negate a potentially important role for maxillary constriction. Recent preliminary data (Palmisano et al., 1996; Cistulli et al., 1996) suggest that rapid maxillary expansion results in a significant improvement of breathing during sleep, suggesting a causal association between maxillary constriction and OSA. However, further work is required to establish the mechanism involved.

In conclusion, the new findings of this thesis are,

1) Maxillary skeletal constriction is a common feature in patients with OSA.
2) Patients with OSA also have narrower, shorter and more tapered archforms compared to normal controls.
3) The shape of the palate is different in OSA patients when compared to normal controls.
4) A significant correlation exists between the lateral dimensions of the maxilla and posterior airway space at the canine \((r=0.37, p=0.04)\), premolar \((r=0.41, p=0.02)\) and molar \((r=0.35, p=0.04)\) regions.
References


References


References


References


Linder-Aronson S., Backstrom A. (1960) A comparison between mouth and nose breathers with respect to occlusion and facial dimensions. Odont Revy. 11,343-346


References


References


References


### Estimate of prevalence of sleep apnoea

<table>
<thead>
<tr>
<th>Reference</th>
<th>Country</th>
<th>Population</th>
<th>Age Range</th>
<th>Method</th>
<th>Criteria</th>
<th>Estimated whole Popn. prevalence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Franceschi et al. (1982)</td>
<td>Italy</td>
<td>2518 consecutive hospital admission males and females</td>
<td>6–92</td>
<td>(1) Questionnaire</td>
<td>AI &gt; 10/h</td>
<td>1%</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(2) Polysomnography on subgroup of 87 (no oximetry)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lavie, (1983)</td>
<td>Israel</td>
<td>1,262 working men</td>
<td>18–67</td>
<td>(1) Questionnaire</td>
<td>AI &gt; 10/h</td>
<td>2.7%</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(2) Polysomnography on 78 (no oximetry)</td>
<td>AI &gt; 20/h</td>
<td>0.7%</td>
</tr>
<tr>
<td>Telakivi et al. (1987)</td>
<td>Finland</td>
<td>Approximately 278 males</td>
<td>41–50</td>
<td>(1) Part of a twin study</td>
<td>AHI &gt; 20/h</td>
<td>0.4%</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(2) 25 snorers and 27 non-snorers had polysomnography</td>
<td></td>
<td>1.4%</td>
</tr>
<tr>
<td>Gislason et al. (1988)</td>
<td>Sweden</td>
<td>3100 males</td>
<td>30–69</td>
<td>(1) Questionnaire</td>
<td>AHI &gt; 10/h</td>
<td>0.9%</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(2) 61 sleepy snorers had polysomnography</td>
<td>AHI &gt; 5/h</td>
<td>1.4%</td>
</tr>
<tr>
<td>Cirignotta et al. (1989)</td>
<td>Italy</td>
<td>355 males</td>
<td>30–39</td>
<td>(1) Questionnaire</td>
<td>AI &gt; 10/h</td>
<td>0.2–1.0%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>738 males</td>
<td>40–59</td>
<td></td>
<td>AI &gt; 10/h</td>
<td>3.4–5.0%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>417 males</td>
<td>60–69</td>
<td>(2) Polysomnography</td>
<td>AI &gt; 10/h</td>
<td>0.5–1.1%</td>
</tr>
<tr>
<td>Stradling and Crosby (1991)</td>
<td>UK</td>
<td>1000 males</td>
<td>35–65</td>
<td>Oximetry on 893</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gleadhill et al. (1991)</td>
<td>Northern Ireland</td>
<td>920 males</td>
<td>40–64</td>
<td>(1) Questionnaire</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(2) Oximetry</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bearpark et al. (1991)</td>
<td>Australia</td>
<td>309 males</td>
<td>40–65</td>
<td>(1) Questionnaire</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(2) Home monitoring</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Young et al. (1993)</td>
<td>USA</td>
<td>1843 females</td>
<td>30–60</td>
<td>(1) Questionnaire</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>1670 males</td>
<td>30–60</td>
<td>(2) Polysomnography</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Approximate Average</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>&gt; 20 events/h</td>
<td>0.3–0.7%</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>&gt; 10 events/h</td>
<td>0.8–3%</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>&gt; 5 events/h</td>
<td>1.5–5%</td>
</tr>
</tbody>
</table>
SLEEP QUESTIONNAIRE FOR CONTROL SUBJECT

Name: 
Age: 
Today's date: 
Sex: 

1) Do you snore? 
Yes/No

2) Snoring frequency: 
On average, how many days/nights during the last month have you snored or been told that you snore? 
(0) Never 
(1) Rarely; < once/week 
(2) Sometimes; 1-2/week 
(3) Frequently; 3-4/week 
(4) Almost always; 5-7/week 
(5) Not sure

3) Snoring intensity: 
During the past month, has your snoring been: 
(0) I have never snored 
(1) Only slightly louder than heavy breathing 
(2) About as loud as mumbling or talking 
(3) Louder than talking 
(4) Extremely loud, can be heard through a closed door 
(5) Do not know

4) 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 
Use the following scale to choose the most appropriate number for each situation:

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

<table>
<thead>
<tr>
<th>Situations</th>
<th>Chance of dozing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sitting and reading</td>
<td>( )</td>
</tr>
<tr>
<td>Sitting, inactive in a public place (e.g. a theatre or 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 a 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>
5) Please rate the quality of your typical night's sleep

(a) very light
    light
    average
    deep
    very deep

(b) very short
    short
    average
    long
    very long

(c) very interrupted
    interrupted
    no interruption

(d) very restless
    slightly restless
    not restless

6) What is your normal number of hours of sleep per night? .............Hours.

THANK YOU FOR YOUR COOPERATION
Orthodontic Examination of OSA / Control patients

Date: ___________________________  Reason for referral: ___________________________
Date of birth: ___________________________  Weight: ___________________________
Height: ___________________________  BMI: ___________________________

Medical History

Asthma: ___________________________  Tonsils: ___________________________
Allergies: ___________________________  Adenoids: ___________________________
Medication: ___________________________  Nasal patency: ___________________________
Snoring: ___________________________  Mode of breathing: ___________________________

Dental History

History of Orthodontics: ___________________________
Jaw surgery: ___________________________
Bruxism: ___________________________

Clinical Evaluation

Profile
vertical thirds: ___________________________
  nose: ___________________________
  chin: ___________________________
  mand. Pl. Angle: ___________________________
  lips: ___________________________

Frontal
vertical thirds: ___________________________
  nose: ___________________________
  symmetry: ___________________________
  shape: ___________________________
  lips: ___________________________

Functional analysis

TMJs: ___________________________
CR/CO: ___________________________

Palate: ___________________________

Tongue: ___________________________

Pharynx: ___________________________

Molars: RHS LHS  Canines: RHS LHS
Overbite: ___________________________
  Overjet: ___________________________
Crossbites: ___________________________