# A Cephalometric Study of the Maxillofacial Structure in Obstructive Sleep Apnoea

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# Summary

Sleep is essential for life. We spend somewhere between one quarter and one third of our lives sleeping, depending upon our sleep pattern. Sleep is a normal physiological process, and as with all such processes there are some differences between individuals that are simply variations of normal. Likewise sleep may be associated with variations that are deemed to be outside the boundaries of those considered normal and is therefore considered pathologic. The investigation of a known abnormality of sleep, namely Obstructive Sleep Apnoea (OSA) syndrome, forms the basis of this thesis and an understanding of normal sleep physiology is essential prior to considering the alterations in normal sleep architecture exhibited in this condition. Chapter 1 considers the definition of normal sleep, the diagnosis of pathologic sleep and the characteristic alterations in sleep and wakefulness demonstrated in a person suffering from obstructive sleep apnoea.

Obstructive Sleep Apnoea syndrome is a medical condition that has been intensively studied by investigators in various fields of medicine and dentistry since being characterized by Guilleminault et al (1976). The classical description of patients with this syndrome was made by Burwell et al (1976). These patients were said to be obese, suffering from hypersomnolence, periodic breathing with hypoventilation and cor pulmonale. Pickwickian syndrome was the term used in 1918 by Sir William Osler when describing patients with a similar clinical presentation. The research into obstructive sleep apnoea syndrome has revealed pathophysiologic alterations in a variety of bodily systems in these people when compared to non-apnoeic people. These pathophysiologic findings are discussed in Chapter 2.

Assessment of the upper airway may be performed clinically, endoscopically, radiographically or using other imaging modalities such as magnetic resonance imaging or manometry. The assessment method evaluated in this thesis is lateral cephalometric radiography. In selecting a method of imaging, it is necessary to have a thorough understanding of the anatomy of the region under examination. Chapter 3 of this thesis outlines the pertinent anatomy of the upper airway and an overview of the available methods of imaging is given. Lateral cephalometric assessment of the upper airway is discussed in particular because it was the modality under investigation. Chapter 4 reviews the literature with respect to known errors using this technology. Although only recently used in relation to obstructive sleep apnoea, lateral cephalometric radiographs have long been utilised by orthodontists and oral and maxillofacial surgeons in the diagnosis and treatment planning of people with malocclusion.

Despite a large number of studies reported in the literature regarding lateral cephalometric radiographs and OSA direct comparison between studies is often difficult. Many studies purport to measure the same parameters e.g. pharyngeal airway width but use different

landmarks. Some studies use control subjects who are age and/or weight matched, other studies use controls not matched for these parameters. Yet other studies use no control subjects at all. Chapter 5 and Chapter 6 of this thesis provide a review of the literature where lateral cephalometric radiographs have been used to assess subjects with obstructive sleep apnoea syndrome.

The imaging of the upper airway is obviously no treatment in itself. One of the problems as with many medical conditions, is the cure may not be acceptable to the patients with the condition. In the case of obstructive sleep apnoea syndrome tracheostomy will cure the patient of the upper airway obstruction however at a personal cost not accepted by most patients. The standard treatment for obstructive sleep apnoea syndrome since 1981 has been nasal continuous positive airway pressure ventilation, reported by Sullivan et al (1981). This modality is reported in the literature to be efficacious however compliance remains an issue. Chapter 7 considers the non-surgical treatment modalities that have been and are used in the treatment of obstructive sleep apnoea syndrome. Surgical treatment for obstructive sleep apnoea syndrome, apart from tracheostomy, has been reported in the literature since 1981 when Fujita reported uvulopalatopharyngoplasty as a surgical technique for the treatment of snoring and obstructive sleep apnoea syndrome. Since that time a number of surgical procedures have been reported with varying degrees of success. These surgical modalities reported in the literature are considered in Chapter 8.

On the basis of the literature review, several aims were established using material from the Oral and Maxillofacial Surgery unit, The University of Adelaide and the Thoracic Medicine Unit, The Royal Adelaide Hospital. The objectives of the study were to:

1. Acquire cephalometric data on 100 consecutive patients undergoing a polysomnographic overnight sleep study for investigation of a suspected sleep breathing disorder as assessed by a thoracic medicine physician.
2. Quantify the airway dimensions of this series of patients using measurements previously reported in the literature.
3. Establish whether BMI has any predictive value for OSA.
4. Establish whether neck circumference is related to BMI or the incidence of OSA.
5. Establish whether age or sex has any influence on the incidence of OSA.
6. Compare airway dimensions measured from the lateral cephalometric radiograph and the severity of OSA to determine if any measurement is predictive for the presence of OSA.

7. Compare airway dimensions measured from lateral cephalometric radiograph and quantify any differences between the study population of OSA patients and simple snorers compared with comparable results reported in the literature.
8. Investigate the sources of error in cephalometry and quantify the error associated with the present study.
9. Quantify the error associated with selected cephalometric variables used in this study.
10. Determine if lateral cephalometric radiographs are a useful adjunct to treatment planning for patients with OSA.

Chapter 9 and Chapter 10 report the methodology used in order to achieve the aims of the study.

Chapter 11 reports the results of this study. The linear and angular variables measured from the lateral cephalometric radiographs was initially assessed with respect to body mass index (BMI) and respiratory disturbance index (RDI). Subjects were then divided upon the basis of RDI into groups of "snorers" and "obstructive sleep apnoeics". Further statistical evaluation was then performed on the groups. To allow comparison with other results from the literature, the division by RDI was performed three times, at an RDI 10, 15 and 20 events per hour. These results are discussed and compared with the findings in the literature in Chapter 12.

Chapter 13 summarises the key findings of this thesis and suggests areas of future investigation to further our understanding of the upper airway changes reported in obstructive sleep apnoeic subjects.

A glossary of terms is provided at Chapter 14.

The Appendix is a copy of the consent and patient information given to participants prior to their enrolment in this study.

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# Statement

This thesis is submitted in partial fulfillment of the requirements for the degree of Master of Dental Surgery. I declare that the context of this thesis has not been previously published or written by another person except where due reference is made. The findings are the results of my personal investigations. No part of this work has been previously submitted for a degree in any university.

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# Chapter 1

# Sleep

## 1.1 Introduction

Sleep is a physiological function that occurs for most people on a regular basis, despite our best attempts, at times, to deny its call. Sleep is the antithesis of wakefulness, and for many it is seen as a waste of time, slowing our ability to extract all we can from life. Conversely others in our midst value sleep above all else, or so it may seem. A number of sleep disorders have been identified, not restricted simply to insomnia or hypersomnolence and these will be discussed in Section 1.3.

For most the sleep – wake cycle follows a circadian rhythm, with sleep occurring nocturnally and wakefulness occurring during daylight hours. The average adult sleeps 7 to 8 hours per night although epidemiological studies suggest a range exists from 4 hours to greater than 9 hours. There are variations to sleep time at the extremes of age that are not suggestive of pathological sleep, however there is an increased mortality rate in people who are at the extremes of this distribution (Kryger et al, 1994).

Environmental factors such as occupation, psychological disturbance such as depression, pharmacological use such as caffeine or physical disease such as renal failure may alter this pattern. The production of symptoms that are troubling to a person, or that produce adverse physiologic changes constitutes a sleep disorder.

Sleep disturbance is a considerable problem for a large number of the population. Insomnia has been reported to have a prevalence of 20 – 30% (Soldaton and Lugaresi, 1987) in adults whilst hypersomnolence has been reported to occur in 5% (Soldaton and Lugaresi, 1987) to 10.9% (Johns and Hocking, 1997) of the population. Other sleep disorders such as night terrors, sleepwalking and confusional arousals have been reported in 2 – 4% of the population (Ohayon et al, 1999).

The study of sleep and determination of pathological states associated with disordered sleep are relatively new areas of medical endeavour, becoming prominent over the last twenty five years.

Understanding of normal sleep is essential if clinicians are to recognize disordered sleep. To this end a large amount of time and research effort has been expended resulting in an ability to measure a variety of parameters related to physiologic functioning and thus define sleep "stages". A sleep study, or polysomnogram will be discussed in Section 1.4. There is however a way to go before we truly understand the mechanisms of sleep and its variations which constitute "pathological" or disordered sleep. Obstructive sleep apnoea is but one of a multitude of sleep disorders, its characteristics with respect to alteration of sleep physiology will be discussed in Section 1.5.

## 1.2 Normal Sleep

Sleep onset in a normal person is usually associated with set rituals and occurs at the same time each evening. Sleep occurs under favourable circumstances in about ten minutes. sleep may be inhibited by a number of conditions including pain, anger, stress or any significant disease or discomforting problem such as excessive temperature or unfamiliar surroundings.

Normal sleep is defined in terms of behaviour and electroencephalographic (EEG) patterns that are associated with physiologic processes. A rhythm is established of sleep followed by wakefulness and is heavily influenced by the light – dark cycle. Related to this rhythm is maintenance of the body by way of hormone release governing cell division, growth, immune function, metabolism and body temperature cycle.

Normal sleep is of two general types, referred to as rapid eye movement (REM) and non-rapid eye movement (NREM) sleep. Polysomnography that measures a number of physiologic parameters will be discussed in section 1.4. The pertinent measures that are used to define sleep stage are the EEG and the electro-oculogram (EOG). The understanding of NREM sleep and its patterns is studied in relation to EEG changes that allow division of sleep into four stages. These stages are characterized by an increase in the arousal threshold and a slowing of the EEG measuring cortical activity.

Stage 1 sleep – is a transient phase of sleep usually occurring at sleep onset but may also be entered briefly at the end of body movement or a period of REM sleep.

This stage of sleep is often perceived as pleasant wakefulness, which is an illusion often experienced. At this stage people are unaware they are losing consciousness and believe themselves to still be awake. This may have unfortunate consequences when people are behind the wheel of a car, in a lecture theatre or other inappropriate situation.

Stage 1 sleep is passed through rapidly at the beginning of a sleep period. EEG changes associated with this period of sleep are a change from alpha waves (8 to 12 HZ) with eyes open to beta rhythms (14 and faster Hz) with eyes closed and finally theta waves (3 to 6 Hz) when stage 1 sleep is entered.

Stage 2 sleep is also entered transiently upon falling asleep but is often the predominant phase of NREM sleep experienced towards the end of a normal sleep period. EEG changes in this phase are the appearance of so-called sleep spindles. These are bursts of alpha-like 10 – 14 Hz waves amongst the background theta waves.

Stage 3 sleep usually only seen during the first half of sleep. This is a period of deep sleep with the EEG slowing producing delta waves.

Stage 4 sleep is also only entered during the first half of sleep. This stage sees further slowing of the EEG with the rhythmic slow waves showing synchronization (Pegram and Lucas, 1995).

REM sleep replaces NREM sleep after approximately 90 minutes, which is called the normal REM latency period. REM sleep can be recognized on an EEG as a return to a mixed frequency pattern similar to stage 1 NREM sleep. REM sleep is thought to be the time dreaming occurs, as dream recall is most vivid when people are awakened at this time. Studies have been performed whereby subjects have been selectively deprived of REM sleep and NREM dream recall has been increased. This may be a purely adaptive mechanism whose significance is not known (Cziesler and Richardson, 1998). A pattern of NREM sleep followed by a period of REM sleep is seen throughout the night, usually cycling at intervals of 90 to 110 minutes. Towards morning the periods of REM sleep increase in duration and NREM sleep stages 3 and 4 are not entered, with stage 2 sleep becoming the predominant phase of NREM sleep entered.

Changes in body position occur most frequently in preparation for and following REM sleep. Generally four to five cycles of NREM/REM sleep are experienced during an eight hour period of sleep. Despite the EEG during REM sleep resembling that seen during wakefulness i.e. rapid low voltage waves there is an increased threshold for arousal by sensory stimulation during this period of sleep.

Alterations in many of the bodies functional systems occur during sleep including the cardiovascular, respiratory, neuromuscular and endocrine systems. The significance of these changes in people suffering obstructive sleep apnoea compared with nonapnoeic people are most marked for cardiovascular, respiratory and neuromuscular systems.

Cardiac changes during sleep are principally a slowing of the heart rate relative to awake resting values. This has been postulated to result from decreased sympathetic outflow during NREM sleep (Snyder et al, 1964). These authors did note the heart rate varies with the stage of sleep. They found the mean rate during REM sleep approaches awake resting values in most subjects. Gillis and Flemons (1994) suggested cardiac muscle has increased refractoriness at night, dependant upon circadian rhythms. This results in a slowing of the heart rate, an increase in atrio-ventricular conduction time and prolonged refractory periods. Cardiac dysrhythmias are most likely to occur during REM sleep.

Blood pressure measurements during sleep are found to decrease in both normotensive and hypertensive individuals compared with waking values. Zachariah et al (1988) reported the average mean blood pressure decrease during NREM sleep was to a level 80% of that measured when subjects were awake.

Respiratory changes during normal sleep include altered gas exchange in the pulmonary alveoli resulting in altered blood gas concentrations from those measured during wakefulness (White et al, 1985). They reported an increase $PaCO_2$ of 2 – 8 mm Hg and a decrease in $PaO_2$ of 3 – 10 mm Hg. These authors also noted the metabolic rate decreased 10 – 20% from baseline levels recorded when subjects were awake. Mechanical alterations in respiration, such as increased upper airway resistance (Hudgel et al, 1984) and physiologic alterations such as depressed respiratory drive (White, 1986) have been suggested as possible causes of the respiratory gas changes in non-apnoeic individuals.

Muscle tone has been measured and reported to vary with the sleep stage. During NREM sleep spinal reflexes and skeletal muscle tone are decreased, these are further inhibited during REM sleep (Chandler, 1988). There are periods of sleep, particularly during REM sleep where this inhibition may be temporarily overcome. Brief periods of movement occur and are often associated with autonomic changes, including alteration of respiratory drive. There is also a typical increase in frequency of movement prior to and emerging from REM sleep.

During normal sleep there is a decrease in tonic and phasic activity of the muscles of the upper airway. The result of this decrease in muscle activity at rest (tonic activity) and during respiration (phasic activity) is an increase in airway resistance. The increased resistance is approximately 2 – 3 times greater in NREM sleep than resistance to air movement during wakefulness. There is also increased resistance in the upper airway during inspiration when compared to expiration. During sleep there is a loss of respiratory responses to wakeful stimuli resulting in a decreased sensitivity of the respiratory system, including the musculature to alterations in upper airway resistance. An additional down-regulation of sensitivity of the upper airway is noticeable by the marked attenuation or complete abolition of the cough reflex during sleep (Czeisler and Richardson, 1998).

Endocrine changes during sleep in a normal population have been measured extensively. Most hormones are secreted rhythmically due to interaction between sleep and circadian patterns. Prolactin and growth hormone seem to be dependant upon the sleep – wake cycle with pulses of these hormones released during the deepest (or stage 4 NREM) sleep. This phase of sleep is most regularly entered during the first third of a sleep cycle. TSH (and ACTH), cortisol and melatonin are principally circadian dependant for their rhythmic secretion. Pulses of cortisol and TSH occur during superficial phases of sleep, particularly towards the end of a sleeping period and this may act to prepare the body for physical activity which has been suppressed (Luboshitzky, 2000).

These mechanical and physiologic changes occurring naturally during sleep may be accentuated in patients with OSA and contribute to the disease severity. The understanding

of these basic physiologic responses of the body to sleep is important when considering the effects of sleep disorders upon the body, particularly OSA. The fact that OSA adversely effects sleep quality leads us to suspect long term adverse health effects. This will be further discussed in Chapter 4.

## 1.3 Classification of Sleep Disorders

Sleep disorders have been classified in a number of ways, the following discussion relates to the classification proposed by the American Sleep Disorders Association. This classification proposes four broad categories of sleep disorder, namely dyssomnias (Table 1.3-1), parasomnias (Table 1.3-2), sleep disorders associated with medical/psychiatric disorders (Table 1.3-3) and proposed sleep disorders (Table 1.3-4). Most sleep disorders are characterized by insomnia, hypersomnia, parasomnia or a sleep-wake schedule disturbance.

A dyssomnia is simply a disturbance of sleep that is caused by an intrinsic disorder of sleep, a disturbance in the circadian rhythm or a disturbance of sleep caused by an external factor. Insomnia is difficulty in initiating or maintaining sleep and is a characteristic feature of many dyssomnias. This is reportedly the most frequent cause of a sleep disorder and may be transient or persistent. Insomnia is not generally a complaint of those patients with obstructive sleep apnoea. Most people experience transient insomnia at some time related to pain or anxiety. These may be related to medical conditions or environmental and psychiatric conditions respectively.

Hypersomnia is a dyssomnia that manifests as excessive amounts of sleep or daytime sleepiness (somnolence) or both. Hypersomnia is seen commonly in patients with depression or with excessive use of alcohol or depressant medications. Somnolence is a common complaint of people with obstructive sleep apnoea or narcolepsy. Narcolepsy is an often dangerous condition involving excessive daytime sleepiness and abnormal REM sleep patterns, with REM sleep within 10 minutes of sleep onset. There is associated hypnagogic and hypnopompic hallucinations , cataplexy and sleep paralysis. People are unable to resist the urge to sleep and it can lead to motor vehicle and industrial accidents.

Circadian rhythm sleep disorders involve a misalignment between the desired and actual sleep periods. The cause may be as simple as jet travel, particularly in an east-west direction or shift work, particularly where the roster changes rapidly therefore not permitting time for the body to adapt. Other circadian rhythm sleep disorders are more difficult to define in terms of aetiology and relate to sleep onset too early or too late for the person's lifestyle.

Parasomnias occur suddenly during sleep or at the junction of sleep and waking. They manifest as unusual or undesirable actions, thoughts or misperceptions. Most commonly,

stage 3 or stage 4 NREM sleep is the period of the sleep cycle in which they occur and thus most people have poor recall for the parasomnic event. Arousal disorders usually occur in the first third of sleep in a NREM stage 3 or stage 4 period. Sleep terror disorder is not a nightmare rather the person awakens suddenly, often screaming loudly before either awakening disorientated or falling asleep. These episodes are not always recalled. Sleep walking is similar except the patient leaves the bed and moves around. This condition occurs most commonly in children between the ages of four and eight. Night terrors and sleep walking are thought to be related and occasionally a neurological abnormality, especially in the temporal lobe, is discovered on EEG analysis.

Sleep-wake transition disorders are generally benign conditions. The most common manifestation is sleep talking (somniloquy) which occurs in both children and adults and usually consists of only a few words. These episodes are not usually recalled.

**Table 1.3-1: Dyssomnias**

| Intrinsic Sleep Disorders |
|---|
| Psychophysiologic insomnia |
| sleep state misperception |
| Idiopathic insomnia |
| Narcolepsy |
| Recurrent hypersomnia |
| Idiopathic hypersomnia |
| Posttraumatic hypersomnia |
| Obstructive sleep apnoea syndrome |
| Central sleep apnoea syndrome |
| Central alveolar hypoventilation syndrome |
| Periodic limb movement disorder |
| Restless legs syndrome |
| Intrinsic sleep disorder not otherwise specified |
| **Circadian Rhythm Sleep Disorders** |
| Time-zone change (jet lag) syndrome |
| Shift-work sleep disorder |
| Irregular sleep-wake pattern |
| Delayed sleep phase syndrome |
| Advanced sleep phase syndrome |
| Non-24-hour sleep-wake disorder |
| Circadian rhythm sleep disorder not otherwise specified |
| **Extrinsic Sleep Disorders** |
| Inadequate sleep hygiene |
| Environmental sleep disorder |
| Altitude insomnia |
| Adjustment sleep disorder |
| Insufficient sleep syndrome |
| Limit-setting sleep disorder |
| Sleep-onset association disorder |
| Food allergy insomnia |
| Nocturnal eating (drinking) syndrome |
| Hypnotic-dependant sleep disorder |
| Stimulant-dependant sleep disorder |
| Alcohol dependant sleep disorders |
| Toxin-induced sleep disorder |
| Extrinsic sleep disorder not otherwise specified |

Nightmares are a very common parasomnia and are an example of a parasomnia occurring during REM sleep (Table 1.3-2). This disorder usually occurs towards the end of the sleep cycle. Up to fifty percent of people report suffering occasional nightmares. Bruxism is a common disorder that occurs in 10 to 20 per cent of the population. The patient usually presents complaining of facial muscle aches or headache. On clinical examination there is evidence of tooth wear. The sleeping partners of the patient are often able to confirm the clinical suspicion. The typical age of onset is 17 to 20 years, and spontaneous remission usually occurs by age 40. Sudden infant death syndrome and infant sleep apnoea are also classified as parasomnias occurring during REM sleep.

**Table 1.3-2: Parasomnias**

| **Arousal Disorders** |
|---|
| Confusional arousals |
| Sleep walking |
| Sleep terrors |
| **Sleep-wake Transition Disorders** |
| Rhythmic movement disorder |
| Sleep starts |
| Sleep talking |
| Nocturnal leg cramps |
| **Parasomnias Usually Associated With REM Sleep** |
| Nightmares |
| Sleep paralysis |
| Impaired-sleep-related penile erections |
| Sleep-related penile erections |
| REM sleep related sinus arrest |
| REM sleep behaviour disorder |
| **Other Parasomnias** |
| Sleep bruxism |
| Sleep enuresis |
| Sleep-related abnormal swallowing syndrome |
| Nocturnal paroxysmal dystonia |
| Sudden unexplained nocturnal death syndrome |
| Primary snoring |
| Infant sleep apnoea |
| Congenital central hypoventilation syndrome |
| Sudden infant death syndrome |
| Benign neonatal sleep myoclonus |
| Other parasomnia not otherwise specified |

Psychiatric disorders may present with many different symptoms of disordered sleep including insomnia (e.g. major depressive disorder, generalized anxiety disorder, adjustment disorder with anxiety) and hypersomnolence (e.g. major depressive disorder, dysthymic disorder).

Neurologic disorders, particularly those of a degenerative nature are commonly associated with sleep disorders. Dementia and Parkinsonism are two common medical conditions that may result in altered sleep patterns. Sleep related headaches are of the typical cluster pattern or similar to unilateral headaches. This latter headache is termed chronic paroxysmal hemicrania and is a vascular headache that occurs almost exclusively in association with REM sleep.

Almost any medical condition that causes pain or discomfort may produce concomitant symptoms of insomnia. Treatment of the underlying medical condition will usually alleviate the sleep disturbance.

**Table 1.3-3: Sleep Disorders Associated With Medical/Psychiatric Disorders**

| **Associated With Mental Disorders** |
|---|
| Psychoses |
| Mood disorders |
| Anxiety disorders |
| Panic disorders |
| Alcoholism |
| **Associated With Neurologic Disorders** |
| Cerebral degenerative disorders |
| Dementia |
| Parkinsonism |
| Fatal familial insomnia |
| Sleep-related epilepsy |
| Electrical status epilepticus of sleep |
| Sleep-related headaches |
| **Associated With Other Medical Disorders** |
| Sleeping sickness |
| Nocturnal cardiac ischaemia |
| Chronic obstructive pulmonary disease |
| Sleep-related asthma |
| Sleep related gastro-oesophageal reflux |
| Peptic ulcer disease |
| Fibrositis syndrome |

Table 1.3-4 lists sleep disorders that do not fit any of the above categories and may or may not be true sleep disorders. They principally describe physical symptoms (e.g. fragmentary myoclonus, sleep related laryngospasm) associated with medical conditions or alterations in sleep architecture (e.g. short or long sleepers) that are markedly different from the general population. These proposed sleep disorders are not agreed by all sleep researchers to be identifiable sleep disorders and may indeed not represent sleep disorders at all.

**Table 1.3-4: Proposed Sleep Disorders**

| |
|---|
| Short sleeper |
| Long sleeper |
| Subwakefulness syndrome |
| Fragmentary myoclonus |
| Sleep hyperhidrosis |
| Menstrual-associated sleep disorder |
| Pregnancy-associated sleep disorder |
| Terrifying hypnagogic hallucinations |
| Sleep-related neurogenic tachypnoea |
| Sleep-related laryngospasm |
| Sleep choking syndrome |

## 1.4 Polysomnography

Polysomnography involves the measurement of various physiologic and clinical parameters during sleep to determine variations from normal and hence the presence or absence of sleep disorders. There are a number of sleep disorders, as mentioned in Section 1.3 and qualitative and quantitative measurement of sleep disturbance in these disorders depends upon the findings of a polysomnographic recording.

Polysomnography typically involves monitoring for seven hours of sleep with measurement of the electrophysiologic variables to determine sleep stage. The variables measured are:

1. Electro-encephalogram (EEG); and
2. Electro-oculogram (EOG).

The EEG measures cortical electrical activity following attachment of at least two electrodes to the scalp. The recording of this electrical activity forms the basis of defining the stages of NREM sleep. Eye movement is recorded by an EOG and this in combination with the EEG is used to determine when a patient has entered REM sleep.

Respiratory parameters are also typically measured during a polysomnographic sleep study. The parameters measured by the sleep laboratory at the Royal Adelaide Hospital are:

1. Respiratory inductive plethysmography;
2. Oro-nasal airflow; and
3. Pulse oximetry.

Chest wall and abdominal movement is measured by respiratory inductive plethysmography. The correlation with the respiratory cycle and movement of these muscles may help define the type of breathing disorder. For example if there is chest and abdominal muscle movement during an apnoeic episode then central sleep apnoea is an unlikely cause, because in this condition neural drive to all the respiratory muscles is temporarily abolished. Conversely movement of respiratory muscles during an apnoeic episode is more likely to point to a diagnosis of obstructive sleep apnoea where there is obstruction of the upper airway. Oro-nasal airflow is measured using a thermistor. This device detects a difference in the temperature between inspired and expired air and can help with timing of the respiratory cycle. Pulse oximetry is used to measure blood arterial oxygen concentration in a non-invasive manner. This parameter is used as a measure to detect oxygen desaturation that does occur during apnoeic or hypopnoeic episodes.

Additional physiologic parameters measured during a typical overnight polysomnographic sleep study at the Royal Adelaide Hospital include:

1. Three lead electro-cardiogram (ECG) continuous monitoring;
2. Electro-myogram (EMG);
3. Snoring by means of a small microphone; and
4. Video recording of patients during an apnoeic episode.

Cardiac abnormalities during sleep are a common phenomenon and some authors have suggested an increased incidence of cardiac arrhythmia associated with oxygen desaturation. Oxygen desaturation is invariably present in obstructive episodes and may be used as a criterion for classification of severity of disease and response to treatment (Powell and Riley, 1995). This is reviewed in more detail in Chapter 2. Electromyographic recording of the tibialis anterior muscles which is essential for the diagnosis of a sleep disorder involving periodic limb movement (restless legs syndrome). Figure 1.4-1 shows a subject prepared for overnight monitoring in the Royal Adelaide Hospital sleep laboratory.

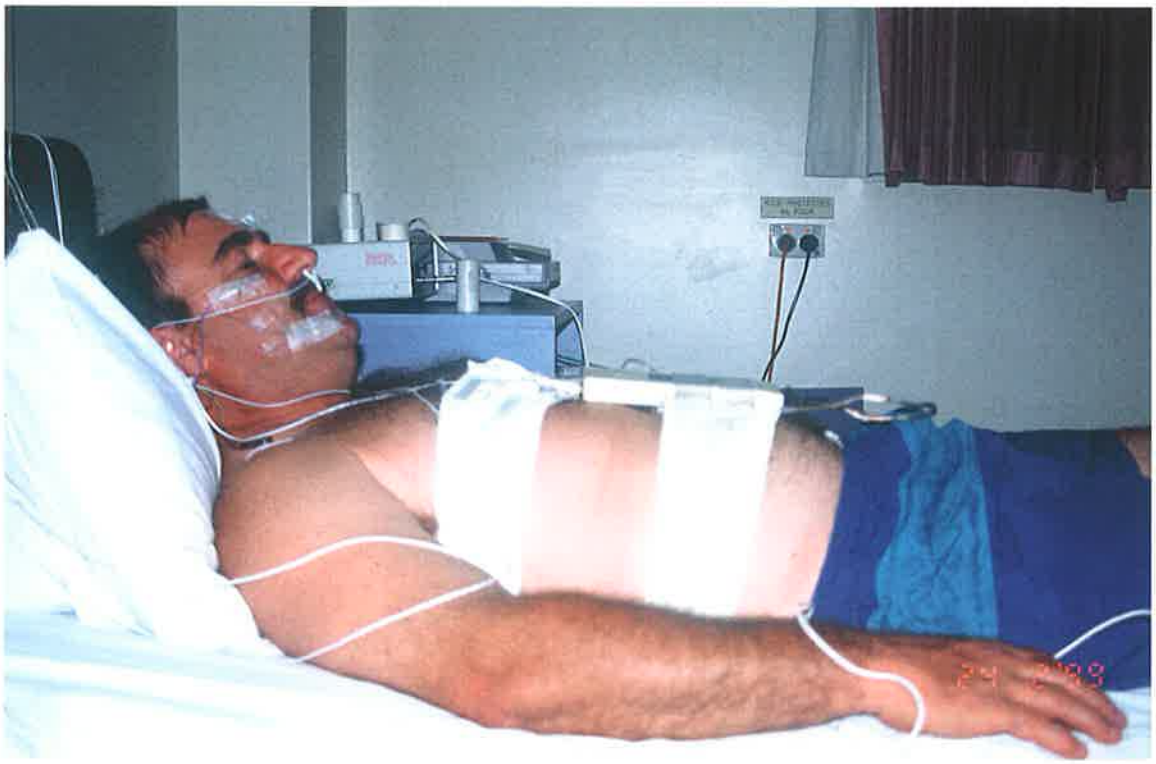

**Figure 1.4-1 Patient undergoing polysomnography**

The raw physiologic data obtained from a sleep study is usually presented both graphically and numerically in table format. Figure 1.4-2 shows a sample sleep study. This data is then analysed by a respiratory physician who notes such information as:

1. Sleep latency;
2. Early morning awakening;
3. Sleep efficiency;
4. Apnoea index;
5. Hypopnoea index;
6. Nocturnal myoclonus index;
7. REM latency; and
8. Sleep-onset REM period.

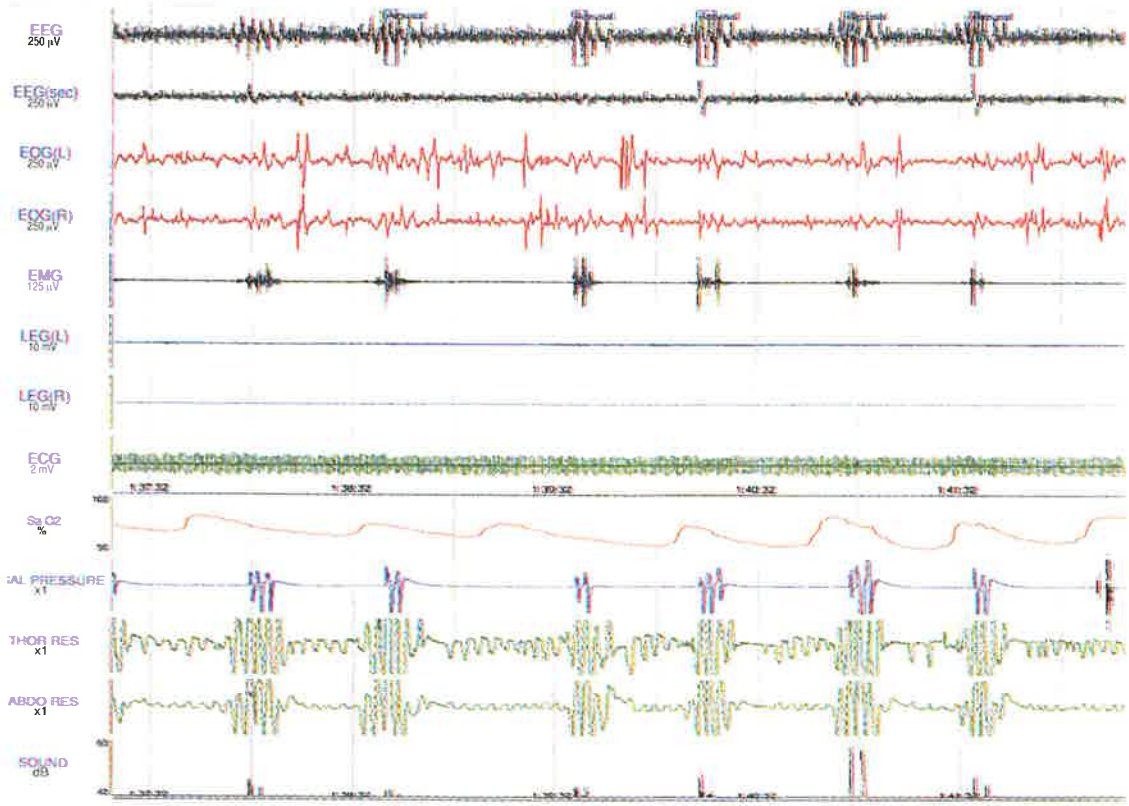


**Figure 1.4-2 Sample sleep study data**

The definition of sleep latency is not clear, with some investigators measuring the time from lights out until the first EEG sign of sleep, whilst others measure from the time of turning off the lights until the subject has entered stage 2 sleep (Carskadon et al, 1986; Thorpy, 1992). The accepted standard for normal sleep latency is greater than ten minutes. For sleep studies at the Royal Adelaide Hospital lights are turned off between 2230 and 2300. Early morning awakening is determined by recording the period of time that elapses from the final arousal from sleep and the termination of the sleep study. Most sleep studies conclude at 0700. Sleep efficiency is determined by the following formula expressed as a percentage: Total sleep time/ total time of the sleep record x 100. Apnoeic events are recorded as the cessation of airflow for greater than 10 seconds. This is expressed as an Apnoea Index, which is the number of apnoeic events per hour of sleep. Hypopnoeic events are recorded when oxygen saturation falls below 95% in a subject who normally saturates at 97% on room air. This parameter is often combined with the apnoeic events to form the Apnoea/Hypopnoea Index (AHI) which is simply the total number of apnoeic or hypopnoeic events per hour of sleep. The nocturnal myoclonus index records the number of periodic leg movements per hour. REM latency is the elapsed time from entering the first stage 1 sleep period until the first REM period of the night. A sleep-onset REM period is not normally entered during the first 10 minutes of sleep.

Polysomnography is the gold standard by which sleep disorders are diagnosed. History, either from the subject themselves, or from a person who observes the subject sleeping, may allow a diagnosis of a sleeping disorder to be made without resort to a time intensive overnight sleep study. In some cases, such as nightmare or sleep enuresis history is sufficient for diagnosis. Other conditions, such as obstructive sleep apnoea require correlation of history with objective findings of physiologic parameters during sleep. There is agreement that this syndrome is diagnosed upon measurement of various physiologic parameters as the patient sleeps in addition to findings during clinical examination. There is disagreement in the literature over which parameters must be measured for a "sleep study" to be valid (Pack, 1993). This author contends that the inclusion of all measuring parameters for a standard overnight polysomnographic sleep study had not been well substantiated in the literature. A meta analysis of the literature regarding diagnosis of obstructive sleep apnoea (Ross et al, 2000) reported the gold standard for diagnosis of obstructive sleep apnoea as reported in the literature was overnight polysomnography. They suggested of the alternative diagnostic procedures reported in the literature, only partial channel or partial time polysomnography looked promising for the replacement of full overnight polysomnography. Partial channel polysomnography records blood oxygen saturation (oximetry), airflow and thoraco-abdominal movement. Partial time polysomnography records sleep for only a small portion of the sleep

cycle, as opposed to standard overnight polysomnography which records all sleep in an eight hour period.

## 1.5 Obstructive Sleep Apnoea Syndrome

Patients suffering from a sleep apnoea syndrome have a dyssomnia classified as an intrinsic sleep disorder (see Table 1.3-1). Gastaut et al (1969) further classified sleep apnoea syndromes into:

1. Central apnoea;
2. Upper airway (obstructive) apnoea; and
3. Mixed sleep apnoea.

In central sleep apnoea the neural drive to respiratory muscles is transiently abolished. This form of sleep apnoea is uncommon and not as easily treated as other forms of sleep apnoea (White, 1994). Upper airway (obstructive) apnoea involves mechanical obstruction of the upper airway during sleep Mixed sleep apnoeic subjects have elements of central and upper airway (obstructive) apnoea.

Obstructive sleep apnoea has been defined at the 1990 meeting of the American Sleep Disorders Association as being " . . . characterized by repetitive episodes of upper airway obstruction that occur during sleep, usually associated with a reduction in blood oxygen saturation..." with associated features of daytime sleepiness and snoring (Thorpy, 1990). This definition highlights the importance not only of obstruction but also the often associated reduction in blood oxygen saturation. This decrease in blood oxygen saturation may occur when the upper airway is totally obstructed, during and apnoeic episode, or during partial upper airway obstruction. Loud, persistent snoring with episodes of choking or cessation of breathing are commonly reported by people sleeping in close proximity to a person with obstructive sleep apnoea. Daytime sleepiness is a very common complaint of people with obstructive sleep apnoea and its likely aetiology will be discussed in further detail in Chapter 3. One of the problems with this definition is it fails to define the number of obstructive episodes that must occur before being considered pathologic. Another shortcoming is the failure to define whether oxygen desaturation alone, in the absence of total upper airway obstruction is pathologic.

Guilleminault et al (1975) defined apnoea as the "cessation of airflow at the nose and mouth lasting at least 10 seconds". During an obstructive episode respiratory effort continues in contrast to central apnoea where respiratory effort ceases.

Guilleminault (1978) and Block et al (1979) were two of the early pioneers in quantification of OSA. They found apnoeic events occurring with greater frequency than five per hour were unusual in the normal population. Thus an AI > 5 was proposed as one of the diagnostic criteria which when present on a sleep study indicated the patient was suffering OSA. Gould et al (1988) performed polysomnographic recording of patients who exhibited the clinical signs of OSA such as excessive daytime sleepiness and hypertension. They found a group of patients who did not obstruct their upper airway but who had a significant decrease in airflow and an associated arterial oxygen desaturation. This reduction in airflow with associated arterial oxygen saturation may be defined as hypopnoea. This work helps justify the inclusion of hypopnoea in the index of measurements when diagnosing OSA from a sleep study.

Further problems arise when the additional criterion of hypopnoea is added. At what point do we consider an hypopnoeic episode significant? Do we count only those hypopnoeas that cause a greater than 50% reduction in airflow, or do we consider any decrease in airflow significant? Do we only count those hypopnoeic episodes associated with arterial oxygen desaturation or arousal? Another factor complicating the use of hypopnoea in the assessment of OSA is the inter-laboratory variability in the detection of hypopnoeic episodes.

Notwithstanding the above problems with detecting and quantifying a hypopnoea most investigators utilize the respiratory disturbance index (RDI) or apnoea hypopnoea index (AHI) to quantify the degree of sleep disordered breathing. These indices give a measure of combined apnoeic and hypopnoeic events per hour of sleep in the criteria for diagnosis of a patient with OSA and allow more objective comparison between populations. There is no consensus as to what number of respiratory events must occur per hour to be pathologic. Several investigators have documented an increase in morbidity and mortality associated with an abnormal RDI on polysomnographic testing, this is discussed in Chapter 2.

The severity of OSA is determined by the number of apnoeas per hour of sleep, the Apnoea Index (AI) or by the combined total of apnoeas and hypopnoeas per hour of sleep, the Apnoea/Hypopnoea Index (AHI). Episodes of apnoea generally last from 20 – 40 seconds during NREM sleep, but may last up to 100 seconds during REM sleep. These obstructive episodes may occur from 200 – 600 times per night (Thawley, 1985).

Most authors consider an AI > 5 or an AHI > 10 per hour of sleep to be pathologic. Several studies indicate that mortality increases in-patients who remain untreated for their OSA if the AHI > 20 (for detail see Chapter 2). This increase in mortality is associated with an increased incidence of vascular disease or of accidents, occurring at work or in a motor vehicle that the affected person is driving.

Guilleminault et al (1976) stated the diagnosis of OSA is confirmed if the polysomnograph shows at least 30 apnoeic episodes in REM and NREM sleep over a seven-hour period of nocturnal sleep. Some of these episodes must appear in a repetitive sequence in NREM sleep.

The incidence of obstructive sleep apnoea in the population is not clearly available in the literature. The principle reason is the failure of clinicians to agree on a set of criteria that allows diagnosis of obstructive sleep apnoea. This makes comparison between studies difficult. Young et al (1993) reported on a random sample of 602 people who underwent an overnight polysomnographic sleep study. They concluded that 2% of women and 4% of men met their criteria for diagnosis of obstructive sleep apnoea. These subjects had an AHI > 5 and reported daytime somnolence. If AHI > 10 alone were the criteria then 10.5% of men and 3.6% of women would be diagnosed with obstructive sleep apnoea. Ferini-Strambi et al (1999) reported an incidence of obstructive sleep apnoea amongst women to be 7.7% (RDI > 10). The incidence in other studies has been reported as low as 1% (Lavie, 1983) in an Israeli working population to 42% (Ancoli-Israel, 1987) who studied an elderly population. A recent report by Tsai et al (1999) scored ninety-four randomly selected overnight polysomnographic sleep studies to determine what effect various definitions for the apnoea-hypopnoea index has on the prevalence of obstructive sleep apnoea. They considered the data in three groups: the first (Type A) had hypopnoea 10 seconds and a > 4% decrease in oxygen saturation ($SaO_2$); the second (Type B) had hypopnoea 10 seconds and a > 4% decrease in oxygen saturation ($SaO_2$) or an arousal; whilst the third group (Type C) had hypopnoea 10 seconds and an electroencephalographic arousal only. Comparing the three groups they found that an extra case of obstructive sleep apnoea would be diagnosed every 14 to 31 sleep studies if the Type B definition was used instead of Type A.

Young et al (1997) reported on 4925 employed adults who had been screened by questionnaire for sleep disordered breathing (including obstructive sleep apnoea). A subset of 1090 underwent overnight polysomnography to estimate the screen detected obstructive sleep apnoea. They estimate from their results that the prevalence of undiagnosed moderate or severe obstructive sleep apnoea was 93% of women and 82% of men with obstructive sleep apnoea.

There is a male predisposition to the development of obstructive sleep apnoea with two studies reporting approximately 85% of subjects diagnosed as being male (Guilleminault and Dement, 1978; and Kales et al, 1985). This bias has been reported to continue into old age with Ancoli-Israel (1987) reporting 19% of women and 31% of men studied to have obstructive sleep apnoea. The average age of participants in this study was 72.4 years.

The incidence of obstructive sleep apnoea increases with age. Ancoli-Israel (1989) reported increasing incidence with age. Jennum and Wildschiodtz (1987) reported an increase incidence in the Danish population they studied from the fourth to the seventh decade. They reported 1.5% of subjects to have an AHI > 30 in the fourth decade rising to 12% in the seventh. Interestingly Bixler et al (1998) reported increasing prevalence of obstructive sleep apnoea with age but decreasing severity.

Many hypotheses have been forwarded in an attempt to explain the pathologic mechanism of upper airway obstruction and OSA. Remmers et al (1978) suggested obstruction may result when there is an imbalance between the activity of the upper airway muscles and thoracic muscles. The thoracic muscles produce a subatmospheric pressure that allows inflation of the lungs but also places a closing pressure on the upper airway.

Howard (1971) thought the upper airway could be likened to a Starling resistor. He determined that the upper airway behaved like a collapsible not a rigid tube. During normal sleep resistance to closure (or obstruction) of the upper airway is balanced principally by pharyngeal muscle tone. During sleep pharyngeal muscle tone decreases and there is an increased resistance to airflow two to three times that encountered whilst awake (Hudgel and Hendricks C, 1988). If sufficient proximal resistance to airflow exists e.g. nasal obstruction then the critical closing pressure in the distal pharynx can be exceeded and the airway will collapse. Patency of the upper airway depends upon:

1. Upstream resistance; and
2. Transmural pressure with-in the collapsible segment.

When the upstream resistance to airflow is high there is a tendency for collapse because the pressure within the more distal pharynx will be more subatmospheric than if the resistance were low. The tone in these muscles decreases during sleep, especially stage 4 and REM, predisposing to collapse. Similarly if there is a high tissue pressure, such as that produced by adipose tissue or oedema in the pharyngeal wall, there may be tendency for collapse. Sub-atmospheric pressure is the collapsing force, usually opposed by upper airway musculature contracting in synchrony with the respiratory muscles. This hypothesis is consistent with the findings of many studies that collapse seems to occur at a localized site in subjects with OSA and narrowing occurs at a localized site in normal subjects. This will be reviewed more extensively in Chapter 7.

Eisele et al (1997) electrically stimulated the hypoglossal nerve, causing contraction of genioglossus and protrusion of the tongue resulting in increased airflow in the upper airway of patients suffering OSA. The effect of this observation is interesting from two perspectives. Firstly it is suggestive that muscle tone of the upper airway musculature, including the tongue

has an influence on the volume of air passing into the upper airway. Secondly it suggests that protrusion of the tongue by surgical or non-surgical means may be beneficial for OSA patients in that the volume of air entering the upper airway is increased. Non-surgical management of OSA is discussed in Chapter 7. Unfortunately the investigators did not measure the effect CN VIII stimulation had on the AHI index, and therefore it is not possible to directly conclude there is any therapeutic benefit for OSA patients in altering the tone of genioglossus muscle.

A familial relationship for the development of OSA has been sought by a number of authors. Guilleminault et al (1995) suggested craniofacial familial features can be a strong indicator of risk for the development of OSA, however this group had problems gaining large numbers of relatives and age-matched controls to complete the study. They also had problems with data collection, particularly the lateral cephalometric radiograph. Redline et al (1995) also reported a significantly greater incidence of sleep disordered breathing in the first degree relatives of patients with OSA than among control patients. Again this study did not use polysomnography to confirm OSA among family or control subjects. Mathur and Douglas (1995) studied first degree relatives of people diagnosed with obstructive sleep apnoea and age, sex, height and weight matched controls. They excluded people with a BMI > 30 $kg/m^2$ or those with gross retrognathia, hypothyroidism, acromegaly or neuromuscular disorders. A significant difference was found between the controls and the first degree relatives when the parameters of snoring, daytime sleepiness and AHI were compared. Lateral cephalometric radiographs were also taken of all subjects in this study and the findings suggest the first degree relatives had a narrower upper airway, a retrognathic maxilla and mandible and a longer, thicker soft palate. They concluded there is a strong familial component in the incidence of obstructive sleep apnoea that may be caused by differences in facial structure.

Obstructive sleep apnoea has only been well characterized over the past 25 years and much investigative work remains before we understand this condition. The diagnosis of obstructive sleep apnoea is controversial with respect to which physiologic parameters are the most accurate predictors of severity. Polysomnography is an important diagnostics tool however clinical history also plays a role in diagnosis. The mechanics of upper airway collapse are not fully understood and the significance of airway narrowing in predisposing to obstructive sleep apnoea is yet to be determined.

# Chapter 2

# Complications of Obstructive Sleep Apnoea

## 2.1 Introduction

Obstructive sleep apnoea syndrome is a result of intermittent narrowing and collapse of the upper airway. The immediate effect of this is to decrease airflow (hypopnoea) or cease airflow completely (apnoea). Oxygen saturation may fall if the hypopnoea or apnoea are prolonged and/or occur frequently. Narrowing of the upper airway and the resulting increased resistance to airflow that occurs in obstructive sleep apnoea causes partial collapse of a segment in the upper airway. Fluttering of the pharyngeal walls or the soft palate causes snoring. Obstructive sleep apnoea is characterized by snoring and there is a spectrum from occasional snorers through heavy, persistent snorers to the varying degrees of obstructive sleep apnoea. These are the obvious and common characteristics of subjects with obstructive sleep apnoea. There is however, a number of other changes seen in the patient with obstructive sleep apnoea that may have far reaching consequences. Our understanding of obstructive sleep apnoea, and the apparent increased morbidity and mortality associated with this condition, has led to a large body of research being undertaken to document the clinical and physiological changes occurring that contribute to this increased morbidity and mortality.

Alterations in the cardiovascular and respiratory systems are most easily seen to contribute to increased morbidity and mortality. Endocrine and neurologic effects have also been linked to obstructive sleep apnoea. Hypersomnolence is the most common complication of obstructive sleep apnoea and this symptom has been linked to decreased cognitive function in this population. Benaim et al (1992) reported an increase in mortality being associated with age, those patients aged less than fifty years old were at greater risk of dying than patients with the same severity of OSA aged greater than fifty years old. This finding has driven many clinicians to target younger subjects with obstructive sleep apnoea for treatment.

The cardiovascular and respiratory changes that occur in subjects with obstructive sleep apnoea will be considered first before a review of the reported complications associated with these systems. These two systems are probably the most commonly affected by obstructive sleep apnoea. There is also a high incidence of cardiovascular and respiratory disease within the general community and the associated morbidity with chronic disease of either of these systems may be increased by the development of obstructive sleep apnoea.

## 2.2 Cardiovascular Changes in Obstructive Sleep Apnoea

In non-apnoeic subjects a number of changes occur in the cardiovascular system during sleep. These include alterations in the balance of the autonomic nervous system with decreased heart rate due to increased parasympathetic and decreased sympathetic nervous system activity

(Baust and Bohnert, 1969). There is also a decrease in systemic blood pressure which peaks in NREM sleep at 80% of the awake pressure (Coccagna et al, 1971). In addition to these changes there is increased myocardial tissue refractoriness which is circadian dependant. This further contributes to the slowing in heart rate, delays atrioventricular conduction and prolongs the refractory period of cardiac muscle (Gillis and Flemons, 1994). The most common arrhythmia seen in non-apnoeic subjects are sinus bradycardia and sinus arrhythmia.

A number of changes in the cardiovascular system have been noted in subjects diagnosed with obstructive sleep apnoea syndrome. In common with all changes to this system some are related principally to the heart whereas others have their pathogenesis in the vascular system. The haemodynamic changes occur in response to a combination of frustrated inspiratory effort, hypercapnia, hypoxia and arousal at the cessation of an apnoeic event.

A number of changes have been noted to occur only during apnoeic episodes. Obstruction causes increased effort to inspire air against the obstruction and is associated with a decrease in arterial blood pressure (Lea et al, 1990). Pleural pressure (the pressure in the apace between the lungs and the chest wall) is reduced by the inspiratory effort. This increase in negative intrathoracic pressure causes an increase in left ventricular afterload (Karam et al, 1984) and reduced left ventricular emptying. Clinically this is analogous to left-sided cardiac failure. There is decreased left ventricular stroke volume and compromised cardiac output (Tolle et al, 1983) during apnoea. Associated with these changes is bradycardia caused by increased vagal activity (Tilkian et al, 1978). Hypoxia has also been shown to contribute to bradycardia (Zwillich et al, 1982). The bradycardia is related to the duration of the apnoea. Gainer (1987) postulated that these hypoxic episodes may also contribute to the deposition of atheromatous plaques in the walls of the large blood vessels.

In addition to compromised left ventricular function, right ventricular workload has also been noted to increase during apnoea. The venous return increases during an apnoeic event because of the decrease in pleural pressure (Guyton et al, 1957), although this is probably limited by collapse of the great veins (Natori et al, 1979). Cardiac output is adversely affected because of decreased left ventricular compliance due to distension of the right ventricle by the increased venous return (Tolle et al, 1983).

A documented association with obstructive sleep apnoea is a decrease in arterial oxygen saturation during an apnoeic or hypopnoeic episode (Davies et al, 1993). This group of investigators noted that the population of obstructive sleep apnoeic patients they were studying was generally obese. They speculated underlying cardiovascular disease may be associated with the obesity and this may simply be exacerbated by the obstructive sleep apnoea not caused by it. Bradley (1992) reported pulmonary hypertension in association

with OSA probably results from a combination of OSA, obesity and diffuse obstructive airways disease, a so-called overlap syndrome. Chaudhary et al (1984) also reported the presence of pulmonary oedema, secondary to cardiac failure, in patients with obstructive sleep apnoea.

During apnoeic periods blood pressure has been noted to increase. Shephard (1985) reported an increase during apnoea of both the diastolic and systolic blood pressure of approximately 25%, peaking at the cessation of the apnoea. The aetiology of this intermittent hypertensive episode is likely to be sympathetically mediated and related both to increased heart rate and increased peripheral vascular resistance. There has been reported a sympathetically mediated tachycardia during arousal (Davies et al, 1993). Multiple studies (Briskin et al, 1978 and Hedner et al, 1988) have shown that activation of the sympathetic nervous system plays an important role in the systemic hypertensive response during apnoeic episodes of OSA patients. Interestingly, recent studies have not implicated OSA in the development of daytime hypertension (Hedner et al, 1990; Hoffstein and Mateika, 1994; and Stradling and Crosby, 1991). Left ventricular hypertrophy, often found in patients with longstanding hypertension and indicating cardiac compensation, has been demonstrated in patients with obstructive sleep apnoea and no daytime hypertension (Hedner et al, 1990). Age and obesity appear to be the factors correlated most closely with hypertension in this group of patients.

A large number of sleep apnoeic subjects have been noted to have cardiac arrhythmia however there is disagreement in the literature as to whether there is a significantly increased incidence in obstructive sleep apnoeic subjects. Greater than 75% of obstructive sleep apnoeic subjects have been reported to demonstrate a sinus bradytachyarrhythmia (Guilleminault et al, 1983). The pattern of these changes was as noted as an initial bradycardia changing to tachycardia at the cessation of an apnoeic event.

The development of cardiac arrhythmia is reportedly greater in patients with OSA than in simple snorers. A large polysomnographic study of 458 consecutive patients found 125/214 (58%) of patients diagnosed with OSA also showed an arrhythmia compared with 103/214 (42%) of simple snorers ($p<0.001$) (Hedner et al, 1988). Hung et al (1990) reported an association between obstructive sleep apnoea and myocardial infarction in men. A possible cause of these myocardial infarctions is the arrhythmia so commonly reported in this group of patients.

In contrast to these studies Flemons et al (1993) compared non-apnoeic controls with obstructive sleep apnoeic subjects using Holter monitors and found no significant difference in the prevalence of cardiac arrhythmias.

## 2.3 Respiratory Changes in Obstructive Sleep Apnoea

The respiratory parameters of arterial carbon dioxide and oxygen partial pressure and oxyhaemoglobin saturation have been reported to alter in "normal" individuals during sleep White et al (1985) reported a fall in the arterial partial pressure of oxygen of 3 – 10 mm Hg and a concomitant fall in arterial oxygen saturation of 2%. They also reported an increase in the arterial partial pressure of carbon dioxide of 2 – 8 mm Hg. The mechanism of these changes has not been confirmed although they generally reflect a decrease in gaseous exchange.

The most obvious alteration affecting the respiratory system during sleep in an obstructive sleep apnoeic patient is repetitive distinct episodes of decreased airflow (hypopnoea) or cessation of airflow (apnoea) of at least ten seconds duration. These events are often associated with a greater than 2% decrease in arterial oxygen saturation. Lugaresi et al (1994) measured intrathoracic pressures on obstructive sleep apnoeic subjects and found these pressures are up to five times lower than that needed for quiet awake tidal breathing. This causes high flow rates in the upper airway and as discussed in Chapter 1 increased flow rates, particularly across narrow areas of the airway may contribute to further narrowing or collapse.

Obesity, which is commonly associated with obstructive sleep apnoea, has been noted to result in ventilation/perfusion mismatching (Tucker and Sieker, 1960). A mismatch of this sort results when there is an area of lung that is perfused with blood but is not filled with air during inspiration. Conversely a mismatch may occur, such as in pulmonary embolism, when an area of lung is no longer perfused by blood but air is still causing the alveoli to expand during inspiration. Obesity may contribute to the former ventilation/perfusion mismatch scenario causing hypoxia. There may also be a decrease in lung oxygen stores and therefore further impairment of ventilation, such as another apnoeic or hypopnoeic event may cause more rapid oxygen desaturation.

This can be seen in any patient with pulmonary disease. Davila (1995) reported patients with pulmonary disease desaturate faster during apnoeic events because of their position on the oxyhaemoglobin dissociation curve (Figure 2.3-1). Further obstruction, either total or partial would potentiate this desaturation. They would gradually move farther from the plateau region of the curve with repeated obstructions, resulting in faster desaturation, thus complicating their problems. There has also been reported a rightward shift of the oxyhaemoglobin dissociation curve due to increased levels of 2,3-diphosphoglycerate (Maillard et al, 1991). This substance is an intermediate in red blood cell synthesis and binds preferentially to deoxyhaemoglobin. The levels of this intermediary are increased during

acclimatization to altitude until erythropoietin stimulates increased production of erythrocytes. The net effect is to make oxygen more readily available to the tissues. This has been proffered as a possible mechanism however given patients with obstructive sleep apnoea have a chronic condition it might be expected relative polycythaemia would be the compensatory mechanism seen, although to my knowledge this has not been reported in the literature.

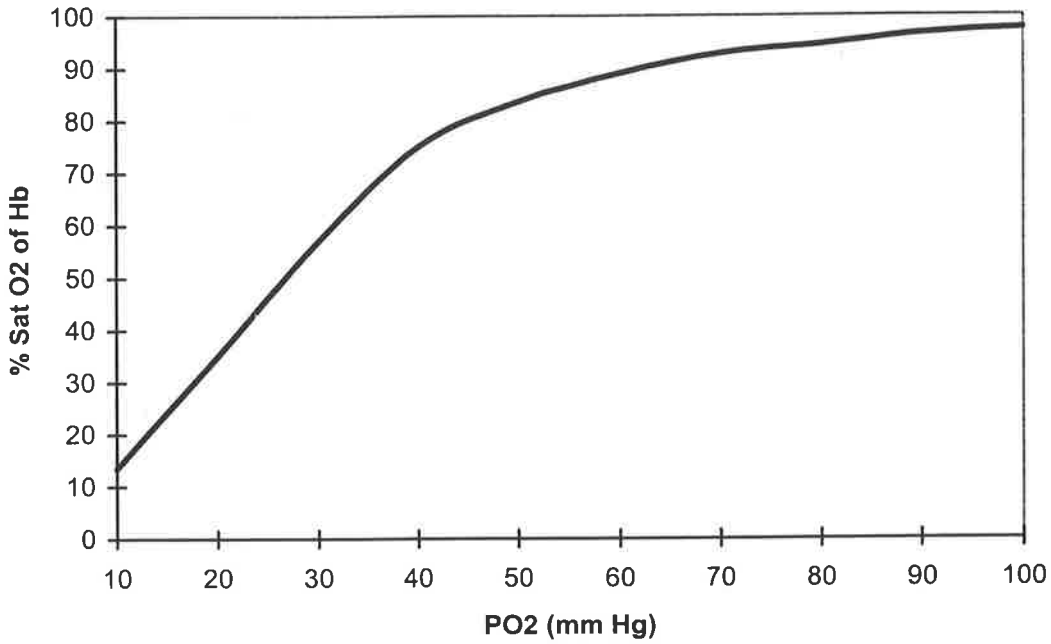


**Figure 2.3-1 Oxyhaemoglobin dissociation curve**

## 2.4 Hypersomnolence

Daytime sleepiness (hypersomnolence) is a common complaint of patients with OSA. The incidence of reported hypersomnolence in a sample of the population varies from five to twelve per cent (Bixler et al, 1979; Klink and Quan, 1987; and Lavie, 1983). Excessive sleepiness is not restricted to people with obstructive sleep apnoea and simple sleep deficiency will cause some degree of hypersomnolence in most people, varying with the amount of sleep lost. The mechanism for excessive sleepiness in people with obstructive sleep apnoea is reportedly microarousal at the conclusion of an apnoeic event. Guilleminault et al (1995) reported normal subjects had a range of arousal during sleep of five to fifteen per hour. They also induced EEG arousal using auditory stimuli in a group of normal subjects. These arousals lasted 11 seconds. They reported decreased sleep latency in this group

(measured by multiple sleep latency test scoring) after one night of sleep fragmentation without sleep deprivation.

Moldofsky (1992) suggested dividing sleepiness into three broad categories:

1. Mild sleepiness

   No involountary sleep, frequent yawning, impaired concentration with momentary inattention, lapses in performing a vigilancè test or irritability.

2. Moderate sleepiness

   Falling asleep against persons wishes whilst passively engaged in a sedentary activity.

3. Severe sleepiness

   Involountary or unwanted sleep attacks when the person is engaged in some physical activity.

These grades of sleepiness are one subjective system for classifying the degree of sleepiness a patient is experiencing based on history alone. Other clinical rating scales for sleepiness have been developed, both self rating techniques and performance tasks. These tests may be valuable in the assessment of patients when considering the initial diagnosis. They are widely used by Sleep Physicians when determining the necessity for a polysomnographic sleep study. An example of such a questionnaire is shown in Figure 2.4-1, the Epworth Sleep Questionnaire.

More subjective testing of patients somnolence can be determined by the multiple sleep latency test or the maintenance of wakefulness test which are both polysomnographic procedures. The basis of the multiple sleep latency test is that the degree of sleepiness can be measured by how quickly a subject will fall asleep if given the opportunity to do so. Typically a subject will be located in a quiet room and seated in a comfortable chair at various times throughout the day. The patient is monitored and sleep latency is measured by the time it takes for the subject to fall asleep This test is considered by many authors to be the gold-standard for assessment of somnolence (Carskadon et al, 1986; Thorpy, 1992). The accepted normal sleep latency is a time greater than ten minutes for sleep onset, moderate sleep latency if sleep occurs between five and ten minutes of the test commencing and severe sleep latency if sleep occurs within five minutes.

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

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

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

0 = Would never doze

1 = Slight chance of dozing

2 = Moderate chance of dozing

3 = High chance of dozing

| **Situation** | **Chance of dozing** |
|---|---|
| Sitting and reading Watching TV | |
| Sitting, inactive in a public place (e.g. a theatre or a meeting) | |
| As a passenger in a car for an hour without a break | |
| Lying down to rest in the afternoon when circumstances permit | |
| Sitting and talking to someone | |
| Sitting quietly after a lunch without alcohol | |
| In a car, while stopped for a few minutes in the traffic | |

A score above seven indicates increased somnolence and further sleep history and appropriate investigation is indicated.

**Figure 2.4-1Epworth Sleepiness Questionnaire (Adapted from Johns, 1991)**

The maintenance of wakefulness test, as the name suggests, is a test of a subject's ability to remain awake. Subjects are monitored and are seated in a comfortable chair in a dimly lit room. The sleepiness of a subject is measured by how rapidly they fall asleep during the day in surroundings conducive to sleep. Variations in sleep latency for normal subjects will vary depending upon the definitions used to determine when the subject is sleeping. There is no accepted standard across the literature. Doghramji et al (1997) considered this problem and performed maintenance of wakefulness testing on 64 healthy subjects. They measured sleep latency to onset of brief sleep and sleep latency to onset of sustained sleep. Brief sleep was defined as a microsleep or onset of any stage of sleep. For their subjects they determined a mean sleep latency of 18.1 +/- 3.6 minutes and a lower normal limit (two standard deviations

below the mean) of 10.9 minutes for the onset of brief sleep. Using the criteria of sleep latency to onset of sustained sleep they measured a mean sleep latency of 35.2 +/- 7.9 minutes and a lower normal of 19.4 minutes. A drawback of these tests is that they are time consuming and therefore costly to perform.

There is not a simple relationship between respiratory disturbance during sleep, as measured by counting the number of apnoeic and hypopnoeic events per hour. A study by Young et al (1993) reported nine per cent of women and 24% of men aged 30 to 60 years had an RDI > 5, although only 2% of women and 4% of men had both a RDI > 5 plus self-reported excessive sleepiness. They defined excessive sleepiness based upon patient history of excessive sleepiness during the day on at least two days per week, waking up unrefreshed irrespective of the time spent sleeping or the experience of uncontrollable daytime sleepiness that interfered with daily living. This study suggests either an RDI of five is too low to be useful in identifying subjects with excessive daytime sleepiness or there are additional mechanisms actively contributing to hypersomnolence in a subgroup of the population with RDI > 5.

The clinical significance of hypersomnolence and the impact on survival of people suffering obstructive sleep apnoea syndrome has been investigated with respect to motor vehicle and industrial accidents. Gonzalez-Rothi et al (1988) compared the incidence of motor vehicle accidents or near misses between a group of 78 subjects diagnosed with obstructive sleep apnoea and 28 control subjects. They found 32% of the apnoeic subjects had experienced this situation compared with 7% of the control group. Jennum et al (1993) reported more striking results with 50 subjects admitting they fell asleep whilst driving and 54% had subsequently had an accident because of falling asleep.

Findley et al (1986) reported an association between driving performance and OSA, with patients who were hypoxic (SaO2 < 90% asleep and SaO2 < 75 mmHg awake) documented with markedly delayed reaction times and difficulty maintaining concentration. 8/9 patients in this study were classified as cognitively impaired and the degree of hypoxaemia was significantly correlated with the degree of cognitive impairment in these patients.

Findley et al (1989) tested subjects on driving simulators and found untreated OSA patients do significantly worse in both city and country driving. Treated these same patients show no significant difference in driving ability from control subjects.

## 2.5 Endocrine Disorders

Atrial natriuretic peptide release is increased during apnoeic events, probably due to an increase in atrial transmural pressure caused by decreased pleural pressure (Guyton et al,

1957). The release of this peptide probably causes the sleeping natriuresis seen in untreated obstructive sleep apnoeic patients (Krieger et al, 1991).

Another finding in many obese OSA patients is an increased fasting insulin level. Strohl (1996) investigated the relationship between obesity, insulin level and OSA. Weight matched obese control subjects (AHI < 5) have an insulin level 50% of that found in patients with an AHI > 20. This finding is consistent with the elevated catecholamine levels found during the day and increased nocturnal serum cortisol levels of OSA patients.

## 2.6 Neurologic Complications

Neurological disease is a contributing factor in a small number of cases of obstructive sleep apnoea. A diverse range of neurologic diseases have been associated with obstructive sleep apnoea and the majority are postulated to interfere with the neural control of the muscles of the upper airway, thus increasing the likelihood of partial or complete obstruction. Direct spinal cord injury resulting in signs of obstructive sleep apnoea in four patients was reported by Bonekat et al (1990).

Obstructive sleep apnoea has been suggested as an underlying cause of pulmonary hypertension and failure to thrive in severely intellectually impaired children. Seid et al (1990) reported on ten such children who were diagnosed with obstructive sleep apnoea but did not have adenotonsillar hypertrophy, the most common cause of upper airway obstruction in children. They performed surgery on the soft palate (uvulopalatopharyngoplasty) and also the adenotonsillar lymphoid tissue and successfully treated the upper airway obstruction in ten of the children. The cause of obstructive sleep apnoea in this group of children was thought to be palatal hypotonicity and redundancy of the soft palate.

Arnold-Chiari malformation is a condition where the cerebellar tonsils and associated meninges (meningocele) or meninges and cord (meningomyelocele) herniate through the foramen magnum and incompletely closed cervical spinal canal. This malformation may result in pressure being placed on the lower cranial nerves and associated vocal cord obstruction. This mechanism has been reported as a cause of airway obstruction during sleep (Holinger and Holinger, 1976).

Shy-Drager syndrome usually presents with signs of autonomic dysfunction initially, before progressing to develop other neurologic dysfunction within five years. This is syndrome is an example of multiple system atrophy accompanied by autonomic failure. This syndrome is associated with laryngeal dysfunction and it has been postulated may be a cause of obstructive sleep apnoea in this group of patients (Kavey et al, 1989).

Amyotrophic lateral sclerosis is the most common progressive motor neuron disease that initially effects upper or lower motor neurons, but in the late stages of the disease most patients exhibit signs of involvement of both systems. Degenerative lesions in the brainstem and cerebellum may cause initial problems with chewing, swallowing, and movements of the face and tongue. Other problems encountered may include autonomic deficits. Five of ten subjects exhibiting these signs were found to have obstructive sleep apnoea (Chokroverty et al, 1984).

Duchenne muscular dystrophy is an X-linked recessive disorder that has been associated with sleep disordered breathing and obstructive events (Hill et al, 1992). This disease is slowly progressive and nocturnal disordered breathing associated with arterial oxygen desaturation are late-stage events. Peripheral muscle weakness occurs first with progressive impairment of respiratory muscles such that a common cause of death in the second decade is pulmonary infection.

In the developed world polio is much less prevalent than in the past, however post poliomyelitis syndrome has been implicated as a cause of obstructive sleep apnoea (Steljes et al, 1990). There is impairment of respiratory muscle function in many of these patients. Vocal cord weakness or hypotonicity of the muscles of the upper airway during sleep have been implicated as the cause of obstruction (Bye et al, 1990).

Obstructive sleep apnoea syndrome may occur because of other pathologic processes or in association with them. Not all of these diseases are life threatening, however there is a clear association between obstructive sleep apnoea in people less than 50 years of age and premature death, principally from cardiovascular causes. The cost to the person and community of an probable increased demand on health services due to comorbidities associated with obstructive sleep apnoea also needs to be considered when assessing the need to manage a person with this condition. Most people suffering from obstructive sleep apnoea are unaware of the potential seriousness of the condition and the possibility of decreased life expectancy if it remains untreated.

Many patients with obstructive sleep apnoea are overweight or obese. Most studies reporting a link between cardiovascular complications and obstructive sleep apnoea do not mention whether the obstructive sleep apnoea is causing the cardiovascular morbidity or whether obesity is the main culprit. It is likely that the obstructive sleep apnoeic patient who is hypersomnolent and lacks energy will tend to lead a sedentary life, thus increasing the likelihood of increasing weight. The increase in weight may lead to increased fat deposition in the pharyngeal walls, thus exacerbating the airway narrowing.

The hypersomnolent obstructive sleep apnoeic patient is also more likely to be involved in motor vehicle accidents. This poses a threat not just to the patient but also those in the community around them. There are no reports on the incidence of industrial accidents associated with obstructive sleep apnoea however it is easily hypothesized this would be the case based upon reports of increased motor vehicle accidents. The social and financial costs of such accidents are not small and provide further evidence for the continued investigation and management of obstructive sleep apnoea syndrome.

# Chapter 3

# Imaging of the Upper Airway

## 3.1 Anatomy of the Upper Airway

The definition of obstructive sleep apnoea syndrome is obstruction of the upper airway and the presence of hypersomnolence and snoring (Thorpy, 1990). Research into the pathogenesis of obstructive sleep apnoea syndrome has not surprisingly been most concerned with upper airway anatomy in such patients. Comparison with non-apnoeic control subjects who may or may not snore is the most commonly used control.

The upper airway may be divided into four anatomic areas for convenience:

1. the nose;
2. the nasopharynx;
3. the oropharynx; and
4. the hypopharynx.

Patency of these areas may be compromised by a number of anatomical and iatrogenic alterations that may be of importance in patients with obstructive sleep apnoea. Knowledge of the structure and function of the various components of the upper airway is important for clinicians that consult patients with suspected or confirmed obstructive sleep apnoea syndrome. The clinician must understand variations from normal and whether differences noted may constitute a pathologic change, thus contributing to the incidence or severity of obstructive sleep apnoea syndrome. All examinations of patients suspected of suffering obstructive sleep apnoea syndrome begin, as is usual, with a detailed history and clinical examination with particular emphasis on the upper airway.

### 3.1.1 Nose

The nasal cavity extends from the nostrils anteriorly, communicating with the external environment to the posterior nasal choane posteriorly, communicating with the nasopharynx. The nasal cavity is divided along its entire length by a septum, consisting of cartilage anteriorly and bone posteriorly.

The nasal cavity is extensive in anteroposterior and vertical dimensions, but narrowed in its lateral dimensions, especially superiorly where it lies between the orbits. The lateral nasal wall has three projections into the nasal cavity, the superior, middle and inferior nasal conchae. The area beneath these projections is called a meatus. The paranasal sinuses (sphenoid, ethmoid, frontal and maxillary sinuses) all communicate directly with the nasal cavity.

The nasal airway serves not only as a valve for gaseous exchange but also modifies the properties of inhaled air. The internal surface area of the nose is large, covered superiorly by olfactory mucous membrane and inferiorly by respiratory mucous membrane. The line of demarcation is the superior nasal conchae.

The purpose of the ciliated columnar or cuboidal respiratory epithelium is to warm, moisten and clean inspired air. There is a rich plexus of veins in the submucosal tissue that accomplishes the warming process. These areas are best developed over the nasal conchae. The respiratory epithelium also contains mucous secreting goblet cells and mucous glands. The secretions moisten inspired air and also cause the surface of the nasal respiratory epithelium to be sticky. This traps inspired particles. Additionally hair is present which acts to filter larger particles from inhaled air.

Hudgel (1992) proposed that three anatomic abnormalities of the nose might contribute to upper airway obstruction. Firstly, nasal obstruction (with mouth closed) leading to an obstructed airway. Secondly, nasal congestion and mouth breathing, with subsequent posterior displacement of the mandible and attached soft tissues might narrow the hypopharyngeal airway. Thirdly, nasal congestion producing turbulent airflow causes a large inspiratory pressure drop across the nose resulting in potential collapse of the pharyngeal airway due to negative pressures developed.

### 3.1.2 Nasopharynx

The nasopharynx extends from the posterior border of the nasal turbinates to a horizontal tangent on the upper border of the soft palate. During breathing the position of the soft palate is maintained by tonic activity of tensor veli palatini muscles and levator veli palatini (Tangel et al, 1991). Tensor veli palatini arises from the upper end of the posterior border of the medial pterygoid plate (scaphoid fossa) and the lateral side of the cartilagenous auditory tube. The muscle fibres converge and form a tendon that passes through buccinator muscle and loops medially around the pterygoid hamulus. The tendons from the right and left sides join to form the palatine aponeurosis. The motor supply is derived from the mandibular division of the trigeminal nerve. Levator veli palatini arises anterior and medial to the carotid canal, located on the petrous temporal bone and the adjacent cartilagenous portion of the auditory canal. The muscle passes superiorly to the superior constrictor and inserts into the posterior border of the palatine aponeurosis between the two heads of palatopharyngeus. The motor supply arises from the pharyngeal plexus derived from the glossopharyngeal and vagus nerves.

The posterior border of the nasopharynx comprises the superior constrictor. This muscle is a thin sheet arising from the lower two thirds of the posterior border of the medial pterygoid and from the posterior end of the mylohyoid line on the lingual side of the mandible. Between the upper and lower origins fibres arise from the pterygomandibular raphe where they meet fibres of buccinator muscle. The superior constrictor muscles are paired and meet in the posterior pharyngeal wall at the pharyngeal ligament and raphe. Superiorly this pharyngeal ligament inserts into the pharyngeal tubercle on the basilar part of the occipital bone. The motor nerve supply to the superior constrictor arises from the pharyngeal plexus of nerves.

Hudgel (1992) suggested the patency of this section of the upper airway could be compromised by local mass lesions, such as lymphatic tissue, scarring secondary to surgery, underdevelopment of the bony skeleton or enlargement of the soft palate musculature. The enlargement of the soft tissue may result from oedema or hypertrophy. Lymphatic tissue most commonly hypertrophied in the nasopharynx is the pharyngeal tonsil, or adenoid, which is located beneath the mucous membrane lining the upper part of the posterior wall. Skeletal malposition might presumably arise from underdevelopment or by retroposition of a normal maxilla. This would effectively place the soft palate attached to the posterior maxilla closer to the posterior pharyngeal wall.

### 3.1.3 Oropharynx

The oropharynx extends from the tangent on the upper border of the soft palate to the tip of the epiglottis. The oral cavity opens into the oropharynx anteriorly through the oropharyngeal isthmus, bounded on either side by the palatoglossal arch, formed by the palatoglossus muscle. The palatoglossus muscle arises from the palatine aponeuorosis laterally and is inserted into the side of the tongue. The left and right muscles act to elevate the posterior tongue and narrow the oropharyngeal isthmus. This occurs normally during swallowing. The motor supply to this muscle arises from the pharyngeal plexus. A second ridge of soft tissue lies posterior to the palatoglossal arch, the palatopharyngeal arch, formed by the palatopharyngeus muscle. The palatopharyngeus muscle arises by two heads, one attached to the posterior border of the hard palate and one from the posterior surface of the palatine aponeurosis. They are separated by the insertion of levator veli palatini. The muscle inserts into the posterior border of the thyroid cartilage and the inferior constrictor muscle. The motor supply to this muscle arises from the pharyngeal plexus. This muscle acts to elevate the larynx and pharynx or to depress the soft palate. The palatine tonsil is located between these two soft tissue elevations. During inspiration these muscle contract and dilate the oropharyngeal airway (Hudgel, 1992).

The middle constrictor arises anteriorly from the stylohyoid ligament, the lesser cornu and the upper border of the greater cornu of the hyoid bone. Similar to the superior constrictor the fibres from each side converge in the midline posteriorly and form the pharyngeal raphe. The upper fibres pass superficial to the lower fibres of the superior constrictor, whilst the lower fibres pass behind the inferior constrictor. As for the superior constrictor, the motor supply arises from the pharyngeal plexus.

The tongue is a large muscle that lies in the oral cavity and the oropharynx. The size, tone and position of the tongue influence the patency of the oropharyngeal airway. In addition Hudgel (1992) has identified enlargement of the palatine tonsils or soft palate, by oedema or hypertrophy, to be possible sources of anatomic narrowing of the oropharynx.

### 3.1.4 Hypopharynx

The hypopharynx extends from the tip of the epiglottis to the inferior border of the cricoid cartilage. The tongue makes up the anterior wall of the hypopharynx. The muscles of the tongue are divided into intrinsic and extrinsic groups. Intrinsic muscles determine the shape of the tongue whilst the extrinsic muscle determine the position of the tongue. The genioglossus is a midline muscle, arising from the hyoid and extending into the body of the tongue. The remaining extrinsic muscles (hypoglossus, styloglossus, chondroglossus and palatoglossus) arise laterally and control tongue position. The hypoglossal nerve provides motor supply to these muscles, with the exception of palatoglossus, which is supplied by the pharyngeal plexus.

The position of the hyoid may alter the dimensions of the hypopharynx. When the muscles attached to the hyoid bone contract to stabilize its position the muscles of the tongue and pharynx can exert their effects. The muscles attached to the hyoid bone can be grouped as suprahyoid and infrahyoid muscles. In addition to opposing one another and stabilizing hyoid bone position the suprahyoid group elevate the hyoid bone whilst the infrahyoid group depress the hyoid bone.

Hudgel (1992) identified macroglossia, mandibular retrognathia or posterior or superior displacement of the hyoid as possible anatomic variations contributing to narrowing of the hypopharyngeal airway.

In addition to the above factors obstructive sleep apnoea has complicated specific anatomic abnormalities. These factors are listed in Table 3.1-1. This table lists anatomic abnormalities that may affect the soft tissues such as an ectopic thyroid or lymphoma, the hard tissues such as micrognathia or rheumatoid arthritis affecting the temporomandibular joint or conditions that may affect both hard and soft tissue such as acromegaly.

**Table 3.1-1: Anatomic Abnormalities Complicated by Obstructive Sleep Apnoea (from Hudgel, 1992)**

| Anatomic Abnormalities Complicated by Obstructive Sleep Apnoea |
|---|
| Adenoid and tonsillar hypertrophy in children and adults |
| Glottic web |
| Vocal cord paralysis |
| Acromegaly |
| Lymphoma or Hodgkin's disease within the pharyngeal lymphoid tissue |
| Micrognathia of various causes |
| Ectopic thyroid |
| Upper airway radiation oedema or fibrosis |
| Retrognathia (congenital or secondary to trauma), inadequate repair of fractures |
| Systemic diseases involving the mandible e.g. RA |
| Correction of velopharyngeal incompetence in infants |
| Severe kyphoscoliosis |
| Cushings disease or syndrome |

Hudgel et al (1988) also listed physiologic abnormalities reported in the literature that may predispose to obstructive sleep apnoea (Table 3.1-2). These physiologic abnormalities may arise due to impaired functioning of the nervous system such as epilepsy, hormonal changes such as hypothyroidism or drug effects such as excessive use of sedatives.

**Table 3.1-2: Physiologic Abnormalities Predisposing to Obstructive Sleep Apnoea (from Hudgel, 1988)**

| Physiologic Abnormalities Predisposing to Obstructive Sleep Apnoea |
|---|
| Poliomyelitis, muscular dystrophy, amyotrophic lateral sclerosis and other diseases with bulbar incoordination secondary to brain stem abnormalities |
| Acquired dysautonomia |
| Diaphragm pacing for primary alveolar hypoventilation |
| Hypothyroidism |
| Flurazepam and other sedative-hypnotic agents induced |
| Testosterone administration |
| Epilepsy |
| Encephalitis |

Fujita (1987) has proposed a classification system for the upper airway to allow easy reference to the site of obstruction. He classified the level of upper airway obstruction into four categories based upon visual inspection. Patients with Type I obstruction had an oropharyngeal narrowing with a normal palatal arch. Type IIa obstruction consisted of a narrowing of the oropharynx coupled with a low palatal arch and relative macroglossia. The hypopharyngeal airway was normal. Type IIb obstruction occurred where both the oropharynx and hypopharynx were narrowed. Type III obstruction involved narrowing of the hypopharynx with a normal oropharynx.

Assessment of the upper airway to determine the site of obstruction has involved many modalities. The inability to visualize the upper airway in three dimensions during an apnoeic episode is the basic problem faced by all that investigate the patient suspected of obstructive sleep apnoea. Computer tomography scans and magnetic resonance imaging are obviously capable of producing three-dimensional images, however the problem lies in obtaining this image during an obstructive episode.

Imaging of a patient during an obstructive episode is not easily obtained, particularly CT or MRI imaging for a multitude of reasons. These include the requirement, particularly with MRI for the patient to remain stationary during the imaging process for an extended period of time (five minutes or more). Secondly many patients would find it difficult to sleep within a machine. The patient would be unlikely to fall asleep in a position suitable for imaging, and if they did would the position be similar to that usually assumed during sleep in their own bed?

Imaging of the upper airway has been performed using direct visualisation, sleep nasendoscopy, plain radiography, CT and MRI. No one imaging modality has been shown conclusively to predict or demonstrate the site of obstruction.

## 3.2 Direct Visualisation

Direct visualization of the upper airway has been attempted by a number of authors, however distortion of the anatomy is a potential problem. The simplest method of direct visualization is inspection of the oral cavity, including the soft tissues of the palate and tonsils. Direct visualization is also used to assess the nasopharynx. The nose and nasopharynx are assessed for septal deviation, turbinate hypertrophy and the presence of nasal polyps or masses.

Obviously this method of visual assessment has significant limitations, not least of which is the limited segment of the upper airway seen. Inspiration also causes enlargement of the

airway, therefore the clinician must be aware of the phase of respiration the patient is in when assessing the patency of the upper airway.

Notwithstanding the above reservations regarding the efficacy of direct visualization of the tissues of the oral cavity and soft palate this is an important examination when investigating any patient with suspected upper airway obstruction. Gross anatomical abnormalities may still be detected with this method of examination.

The nasal airway is little mentioned in the literature concerning obstructive sleep apnoea. The nose may contribute significantly to upper airway resistance, reportedly up to one third of upper airway resistance occurs in the nasal airway.

Deviation of the nasal septum (usually cartilaginous), hypertrophic nasal conchae, nasal polyps from obstructed glands or other masses may obstruct nasal airflow. Patients presenting for investigation of suspected obstructive sleep apnoea need these problems rectified for two reasons. In some patients this may be therapeutic, in others it may improve compliance with therapy such as nCPAP by decreasing the ventilatory pressures required. Series et al (1993) reported relief of airway obstruction may be accomplished by decreasing the resistance in the nasal airway, especially in mild obstructive sleep apnoea patients or snoring patients. The treatment may involve the use of nasal decongestants, steroids or dilators or surgical correction of underlying anatomic anomalies.

Patients diagnosed with obstructive sleep apnoea and who are to be treated therapeutically with nCPAP may also benefit from thorough examination and correction of underlying nasal pathology. Lower pressures may be needed when titrating nCPAP thus potentially improving patient compliance through greater comfort.

Examination of the oral cavity should be next on the clinical examination. Particular notice should be taken of the soft palate, the width of the hard palate, the tonsils and the tongue. Abnormal size of these structures has been variously reported as contributing to obstructive sleep apnoea. Additionally, when examining the oral cavity and oropharynx, the patient will open their mouth to maximum interincisal distance, a position of their jaws almost certainly not achieved and maintained during sleep The effect of mouth opening utilizes muscles of the tongue and upper airway, thus moving them from their position of rest.

Mallampati et al (1985) proposed a rating system based on the degree of visualization of the tongue, tonsillar pillars, soft palate and uvula. This clinical rating divides patients into three grades with an increased probability of snoring and obstructive sleep apnoea with increasing grade. The assessment is made with the patient having a wide open mouth and extended tongue (see Figure 3.2-1).

1. Grade 1 allows visualization of the tonsillar pillars, soft palate and uvula with at least 5mm between the uvula and the tongue;
2. Grade 2 allows visualization of the tonsillar pillars and soft palate, but the tongue base obscures the tip of the uvula;
3. Grade 3 allows visualization of the soft palate only.

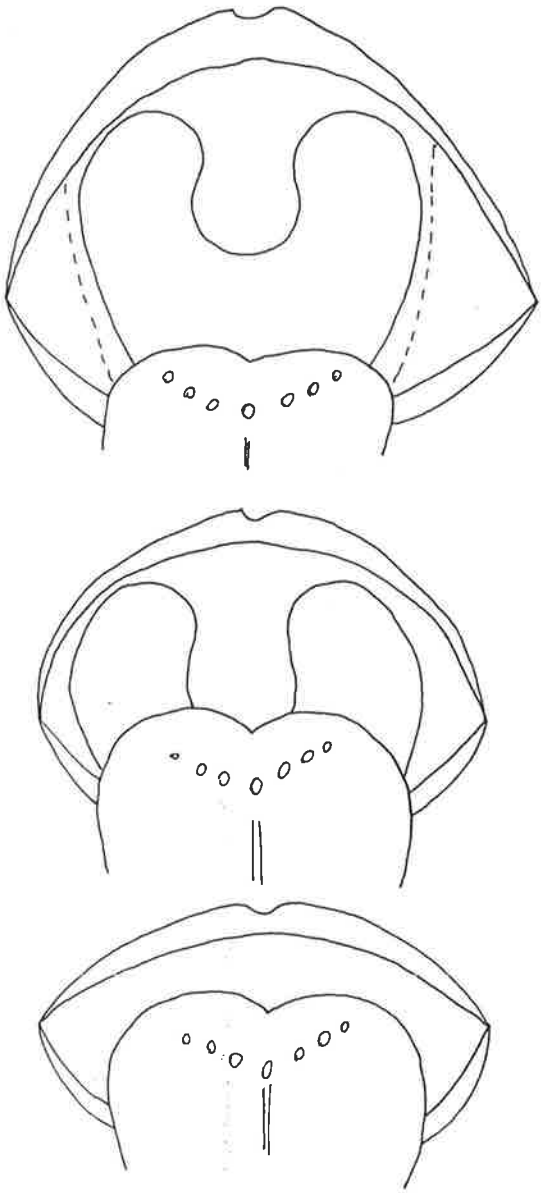

**Figure 3.2-1 Grading of the oropharynx (after Mallampati)**

Differentiation must be made between an enlarged tongue or soft palate causing the obstructed view of the uvula. Macroglossia may be evidenced by abnormal tongue thrusting on swallowing, elevation of the tongue above the occlusal plane of the mandibular teeth or obvious indentations on the lateral border of the tongue in dentate patients. Relative macroglossia may occur in patients with a deficient maxilla, such as a person with trisomy 21 or with a deficient mandible.

Soft palate enlargement may only involve the uvula, alternatively there may be generalized hypertrophy extending to include the posterior pillar of the fauces. Anatomic variation in uvula length is marked, and a better estimation of the tendency for an enlarged uvula may not be length but volume. A long, thin uvula may well have its tip below the level of the tongue and not visible on inspection of a patient with their mouth open. This pattern however is unlikely to cause obstruction of the upper airway, however a shorter uvula with increased lateral or AP dimensions is more likely to effectively obstruct the upper airway.

Identification of a problem with the soft tissue size of the tongue or soft palate by clinical examination should be attempted and further investigated by diagnostic imaging where appropriate. All patients with enlarged soft tissues are not obstructive sleep apnoeics, but where a high index of suspicion is held it obligates the clinician to fully investigate the patient.

Adult patients with obstructive sleep apnoea may have enlarged tonsils, however more commonly the relative prominence of the tonsils is due to displacement of the tonsillar fossa by lateral pharyngeal wall enlargement. This is most likely to be due to fat depositions in the submucosal tissue. The presence or absence of tonsils in the obstructive sleep apnoea patient treated by UPPP has not been reported of significance in the success or otherwise of this treatment. It seems likely that the presence or absence of tonsillar tissue may not play a large role in the development of upper airway obstruction, except in the case of obvious tonsillar hypertrophy. The rationale for removal of tonsils that are prominent due to underlying submucosal adipose tissue deposits lies in the difficulty in safely and effectively removing this adipose tissue.

Clinical examination of the soft tissues of the posterior oral cavity obviously presents a problem in patients who have undergone previous palatal ablation surgery for "simple snoring" and are later diagnosed as obstructive sleep apnoeic patients.

Although many investigators have sought an anatomical basis for obstruction it is probable that a functional element also exists. Hudgel and Hendricks (1988) suggested an additional factor must be responsible when noting that at the conclusion of an apnoeic event there is sudden opening of the upper airway. The airway resistance for the next few breaths is low,

suggesting there is not a fixed anatomic narrowing. He also noted that increasing the drive to breath during sleep, such as increasing $PaCO_2$, causes a reduction or complete elimination of the ventilatory oscillations and apnoeas.

## 3.3 Nasendoscopy

Fibre optic nasendoscopy has been widely used in assessment of the upper airway (Crumley et al, 1987; Skatvedt, 1993; and Woodson and Wooten, 1994). This modality has been used in an attempt to visualize the upper airway of patients awake or asleep, upright or supine.

An early study using endoscopy by Crumley et al (1987) assessed patients post UPPP. Some of these patients had failed to respond to surgery. The authors found the upper airway at the soft palate was narrower antero-posteriorly than laterally. They also found the antero-posterior dimension at the tongue base decreased in size when patients moved from an upright to supine position.

The authors compared these findings with those using cine-CT in awake and asleep patients and found greater accuracy using this imaging modality. The reasons for this are principally because the cine-CT scan could be done with the patients asleep, whereas the nasendoscopy was performed with patients awake.

They concluded that endoscopic visualization of the upper airway was incapable of predicting failures from UPPP. The site of obstruction did differ in awake and asleep patients, with asleep patients often obstructing at more than one site. This indicates that studies on awake patients to identify sites of airway narrowing may not correlate with sites of narrowing and obstruction when asleep. There is also the added problem that more than one site of the upper airway on some patients contributes to the obstructive events during sleep, complicating the diagnosis of the site of obstruction and potentially its management.

Some investigators have utilised the Mueller manouvre in an attempt to precipitate upper airway obstruction in an awake patient. This manouvre has been postulated to reflect upper airway compliance during wakefulness. These studies hypothesize the site of obstruction produced correlates to that which occurs during sleep.

Blocking the external nares and asking the subject to inspire against this obstruction, keeping the mouth closed throughout performs Mueller's manouvre. There is no movement of air between the atmosphere and the upper airway but a closing pressure is produced within the upper airway. The patient is usually in a seated position during performance of this manouvre. Imaging, either direct or indirect, may then be used to assess the degree and pattern of collapse.

Skatvedt (1993) used fibreoptic nasopharyngoscopy and pressure measurements of patients performing the Mueller manouvre and attempted to correlate these findings with manometric measurements of obstruction during sleep. He found no correlation between the level of obstruction during sleep as measured by manometry and the findings of measurement of the waking Mueller's manouvre.

Skatvedt (1993) postulated Mueller's manouvre is not a satisfactory method of determining the site of upper airway obstruction during sleep. Instead it can only identify the degree and orientation of upper airway collapse whilst awake. There is no certainty that the same pattern of obstruction occurs during sleep based upon these findings.

## 3.4 Manometry

Nasendoscopy has been combined with pressure measurements using a manometer. The manometer measures pressure in the upper airway during sleep, and the authors used this information to compare site of obstruction during sleep and site visualized by nasendoscopy.

Skatvedt (1993) reported that only 5/20 patients had the same site of obstruction, or absence of obstruction demonstrated with both methods. Significantly 12/20 patients failed to show obstruction whilst awake with nasendoscopy, but did demonstrate obstruction whilst asleep with manometry. These results reinforce those found by other authors that direct measurement of the airway in awake patients may fail to detect obstruction that is present in the sleeping subject.

Woodson & Wooten (1994) examined awake obstructive sleep apnoea patients upper airway by clinical evaluation and nasendoscopy. An attempt was made to correlate findings at this examination with findings of upper airway obstruction in the same group of patients with manometry and videoendoscopy during sleep. When using manometry complete obstruction must occur for registration, however the use of videoendoscopy allowed measurement of severely narrowed upper airway that was not necessarily occluded.

The group consisted of 22 patients, on clinical examination 19 were classified as Fujita type IIb (tongue base obstruction only). The other 3 patients were classified as Fujita type IIa with obstruction limited to the palate. Manometry found almost opposite results, with only 5 patients found to have initial obstruction at the tongue base. The remaining 17 patients had no evidence of tongue base obstruction with either manometry or endoscopy, obstruction being confined to the palate.

This study is interesting for the different measures of obstructive episodes found at different times during the respiratory cycle. The following are the incidence of sleeping tongue base obstruction objectively measured by this study:

1. Manometric early inspiration 5/21 (24%);
2. Manometric late inspiration 11/21 (52%);
3. Endoscopic late inspiration 14/19 (74%); and
4. Endoscopic expiration 8/19 (42%).

These results indicate that for both methods of objective measurement variations are found depending upon the phase of the respiratory cycle the measurement is taken. No attempt was made by the authors of this study to determine which measurements are physiologically or clinically significant.

The authors found the only characteristic found on physical examination that correlated with tongue base obstruction during sleep was near total collapse of the tongue base on supine endoscopy. Importantly they found no correlation between Muellers manouvre and tongue base collapsibility during sleep.

Shepard and Thawley (1990) utilized manometry to determine the effect of body position, sleep state and UPPP on the regions over which the upper airway collapses during sleep. They studied 18 obese (BMI 37+/-2 $kg/m^2$ range 25 – 48 $kg/m^2$) patients with an AHI 62+/-8/h.

They found the region of collapse remained constant throughout the night for a given sleep state and body position. During NREM sleep 10/18 (56%) had collapse of the nasopharyngeal segment only, whilst in 6/18 (33%) collapse extended into the oropharynx. In only 2/18 (11%) was collapse initiated in the oropharynx.

The area of collapse was found to alter according to the sleep stage. 7/9 (78%) had collapse of their upper airway extend caudally during REM sleep.

Only 2/10 patients had alteration of site of obstruction following change of position from supine to the lateral cubitus position.

This study reinforces the fact that obstructive sleep apnoea may be due to collapse of multiple segments of the upper airway. It also shows that nasopharyngeal collapse of the upper airway cannot always be satisfactorily treated with UPPP.

## 3.5 Somnofluoroscopy

Other imaging modalities have been used in an attempt to identify patients who may respond to UPPP for treatment of obstructive sleep apnoea. Katsantonis and Walsh (1986) utilized somnofluoroscopy to record the airway whilst patients were undergoing polysomnography. This study demonstrated five different patterns of airway collapse:

1. oropharyngeal initiation propagating into the hypopharynx;
2. hypopharyngeal initiation propagating into the oropharynx;
3. nasopharynx only;
4. oropharynx only; and
5. hypopharynx only.

These results obviously differ from the previous study of Shepard and Thawley (1990) who used manometry to assess the location of upper airway collapse. Katsantonis and Walsh (1986) also report that 20/26 (77%) of had upper airway obstruction corresponded with the narrowest site of the upper airway. They also report an obstructive event generally commences at the end of the expiratory phase of respiration, this is in contrast to Woodson & Wooten (1994) who found with endoscopy only 42% of patients obstructed at this phase of the respiratory cycle.

## 3.6 CT and MRI

CT studies in awake apnoeic and non-apnoeic patients have been performed to assess the changes in upper airway shape that occur during normal respiration (Schwab, Gefter, Hoffman et al, 1993; Schwab, Gefter, Pack et al, 1993). Four distinct changes in airway dimensions have been described during the respiratory cycle.

There is an increase in upper airway volume at the beginning of inspiration, presumably due to the action of dilatory muscles. During the remainder of inspiration the volume of the upper airway remains relatively constant, postulated to represent equilibrium between the upper airway dilator muscles and negative intraluminal airway pressure tending to cause collapse of the airway. Airway volume increases again at the beginning of expiration to its maximum size. During this time there is a change from negative intraluminal pressure to positive intraluminal pressure. The end of expiration sees the airway collapse to varying degrees, no longer being maintained open by intraluminal pressure or the action of dilatory muscles.

The conclusion intuitively reached from these studies is that the upper airway is most vulnerable to collapse at the end of expiration.

Kuna et al (1988) utilised cine-CT and Mueller's manouvre in their study of upper airway collapse on awake patients. They found obstructive sleep apnoea patients upper airway's collapsed in a lateral to medial direction with this manouvre, whilst control patients upper airway's collapsed in an antero-posterior direction.

A study using MRI by Schwab et al (1995) compared 21 subjects with obstructive sleep apnoea (AHI>15 events/h), 21 subjects who snored and/or had mild obstructive sleep apnoea (AHI<15 events/h) and 21 normal subjects (AHI<3 events/h). These subjects were not age or weight matched with significant differences ($p<0.0001$) between all groups for both BMI and age. Patients with obstructive sleep apnoea were older and significantly heavier than either other group.

This study found the airway was narrower in the apnoeic patient, and that this narrowing is predominately in the lateral direction. There were no significant differences in A-P upper airway dimension between the apnoeic and normal patients. In apnoeic patients the spatial orientation of fat in the subcutaneous tissues of the pharynx differs from control subjects, as does the volume of fat in the lateral and posterior pharyngeal walls. Interestingly there was not an increased amount of fat at the site of airway narrowing, indicating that this fat is probably not causing compression of the upper airway alone. The authors also measured the distance between the mandibular rami of apnoeic and control subjects and found no significant difference between the two groups.

CT has also been utilized to allow three-dimensional viewing of the upper airway. Studies using this modality necessitate the patient is awake, and again the effect of sleep on the upper airway cannot be directly assessed using this method. Shephard et al (1990) compared 17 obstructive sleep apnoea patients with 13 controls (not age or weight matched) by CT imaging and assessment of airway collapsibility by way of continuous negative airway pressure (CNAP). They found no evidence of excessive fat around the airway of either group, although the obstructive sleep apnoea patients had BMI of 36+/-2 $kg/m^2$. They found the minimum airway cross sectional area was located in the velopharyngeal segment in and 16/17 (94%) of obstructive sleep apnoea patients 12/13 (92%) of controls. The authors found no difference in the collapsibility of the airway between the two groups. CPAP at 10cm of $H_2O$ increased the minimum airway cross sectional area in the obstructive sleep apnoea and control groups 59% and 62% respectively. They measured this area at 0, +10 and –5cm of $H_2O$, with the minimum upper airway cross sectional area being 36 – 41% smaller at all pressures in obstructive sleep apnoea patients compared with the controls.

Lowe et al (1995) used CT to evaluate the airway of 80 patients with obstructive sleep apnoea and compared them with 25 controls not matched for age or weight. The measure of upper airway volume found significantly larger tongue volume ($p<0.000$), soft palate volume ($p<0.000$) and upper airway soft tissue volume ($p<0.001$) in the apnoeic population. These authors concluded increased BMI probably accounted for the difference in these soft tissue measurements between the two groups.

## 3.7 Lateral Cephalometry

Cephalometry is the most widely reported method of imaging the upper airway of patients with obstructive sleep apnoea. A lateral cephalometric radiograph is widely used by oral and maxillofacial surgeons in treatment planning for orthognathic surgery and by orthodontists prior to treatment. Compared with a CT of the upper airway a lateral cephalometric radiograph is low cost and exposes the patient to a small radiation dose.

The advantages of using a lateral cephalometric radiograph include:

1. Longitudinal comparison is possible, thus allowing time based analysis of natural or therapeutically induced changes in craniofacial anatomy;
2. Comparison of the size and shape of craniofacial structure among different individuals is possible in a reproducible and quantifiable manner.
3. Comparisons of relative size, shape and position of anatomic structures within the same individual as in the counterpart analysis.

The major disadvantage of a lateral cephalometric radiograph is its two-dimensional representation of a three-dimensional structure (Hans and Goldberg, 1993). Many studies have been performed using these radiographs to determine if meaningful conclusions may be drawn from them about the three-dimensional structure of the UAW from this two dimensional image. Figure 3.7-1 shows an example of a lateral cephalometric radiograph.

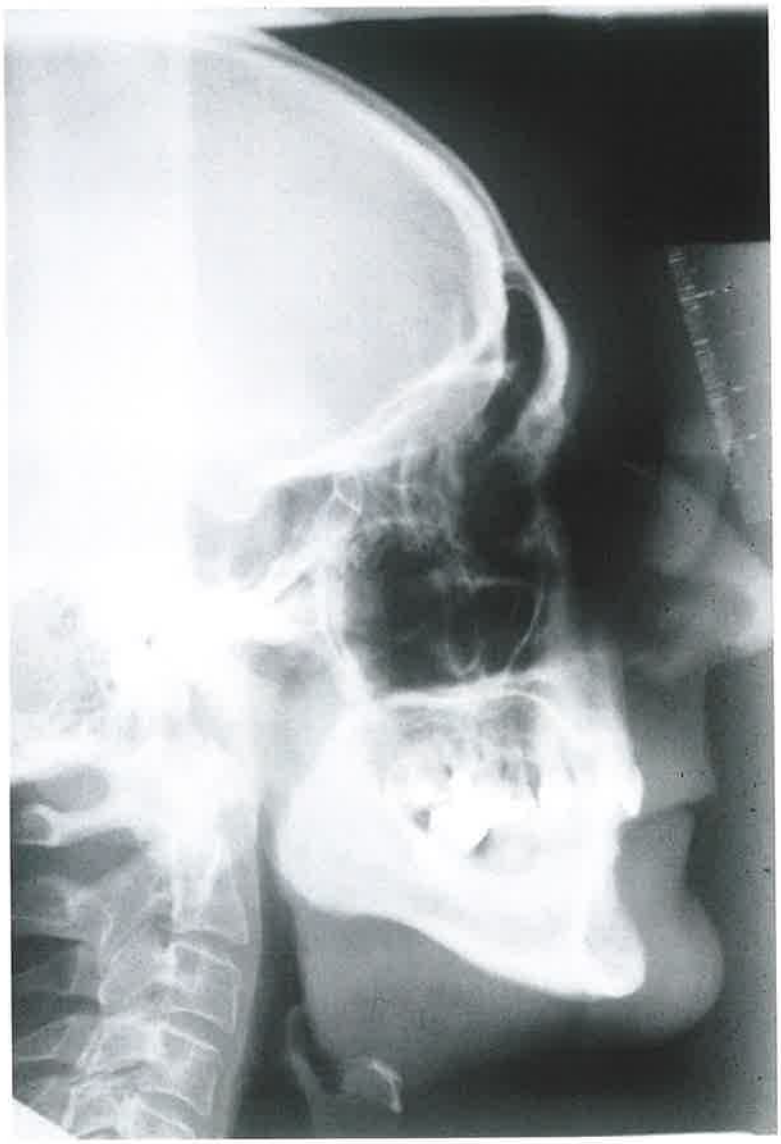

**Figure 3.7-1 Lateral cephalometric radiograph**

Gender and racial variation in the incidence of OSA and the cephalometric measures of the upper airway and facial skeleton have been reported (Lee et al, 1997; Lowe et al, 1996; Redline et al, 1997). African-American, Hispanic and Caucasian subjects, both male and female, had cephalometric variables compared by Lee et al (1997). Racial differences were found with the cephalometric measures of maxillary and mandibular anteroposterior position in male subjects. African-American subjects had a more prognathic maxilla than Hispanic or Caucasian men, whilst African-American men had a more prognathic mandible than Caucasian counterparts. There were no racial differences in cephalometric measures between women. Gender differences were reported between Caucasian men and women. The women were found to have a significant difference in the length of the soft palate, the distance from the hyoid bone to the mandibular plane and the width of the posterior nasal space at the level of the tongue. The study did not specify whether these distances were increased or decreased in Caucasian women compared to Caucasian men. Lowe et al (1996) separated subjects on the basis of skeletal pattern (ClassI, II or III) and gender. They found female subjects with obstructive sleep apnoea had increase soft palate and tongue dimensions compared to an homologous control group, but no difference in skeletal measurement on lateral cephalometric radiographs. Redline et al (1997) compared African-Americans with a Caucasian population. They found the African-American population with an increased RDI were younger and concluded that young African-Americans may be at increased risk for obstructive sleep apnoea.

Other studies have been published in the literature reporting differences in some craniofacial measures from lateral cephalometric radiographs in other racial groups when compared to a Caucasian population. Differences have been found in Japanese (Alcade et al, 1998; and Miyajima et al, 1996), Chinese (Cooke and Wei, 1988; Shen et al, 1994) Korean (Park et al, 1989) and African-American (Lee et al, 1997; and Redline et al, 1997).

Despite a large number of studies reported in the literature regarding lateral cephalometric radiographs and obstructive sleep apnoea, direct comparison between studies is often difficult. Many studies purport to measure the same parameter e.g. pharyngeal airway width but use different landmarks. Some studies use control subjects who are age and/or weight matched, other studies use controls not matched for these parameters. Yet other studies use no control subjects at all.

The more severe the obstructive sleep apnoea the greater the number and severity of abnormalities measured (deBerri-Borwiecki et al, 1988). Another finding from this study is the tendency for obstructive sleep apnoeic patients to have a retrognathic, dolichofacial (elongated) appearance with a narrowed posterior airway space.

Pracharktam et al (1994) suggested there is a mismatch between the head form and the facial form of obstructive sleep apnoea patients. They found most obstructive sleep apnoea patients in a small sample had brachycephalic (round-short) head, leptoproscopic (long-narrow) form whilst controls were randomly distributed.

A large study by Lowe et al (1996) of 347 obstructive sleep apnoea patients and 101 controls divided the subjects into groups according to the Angle Classification. They found some parameters to be significantly different for all obstructive sleep apnoea patients irrespective of the occlusal relationship of the teeth. The significant measurements are generally for soft tissue parameters such as soft palate length and thickness and tongue length. Interestingly they found the obstructive sleep apnoea patients with an Angle Class I relationship had a significantly smaller maxilla and a significantly smaller and more posteriorly positioned mandible than control subjects with the same occlusal relationship. Other authors have found similar results (Battagel and L'Estrange, 1996).

These findings of bimaxillary retrusion are compatible with an Angle Class I relationship if the anterior cranial base length is also reduced. The patients with an Angle Class II relationship had a significantly smaller maxilla than control subjects with the same occlusal relationship. This latter group also had a significantly narrowed velopharyngeal and oropharyngeal upper airway compared with control subjects. These findings are consistent with the evidence that when considering abnormalities of cephalometric measurements on obstructive sleep apnoea patients the skeletal proportions must also be examined.

Tsuchiya et al (1992) proposed, on the basis of measurements made from lateral cephalometric radiographs for 84 obstructive sleep apnoea patients and 18 control subjects, that two distinct groups of obstructive sleep apnoea patients exist. The first, more numerous group of patients are characterized by soft tissue abnormalities. The second group is characterized by skeletal abnormalities. These authors further analysed the two groups of patients and found those with predominately soft tissue abnormalities had a high BMI and low apnoea index. Conversely, a low (or normal) BMI and a high apnoea index characterized those patients with skeletal abnormalities. Partinen et al (1988) also supported this hypothesis in their published results.

Hoffstein et al (1991) and Pracharktam et al (1994) have considered the upper airway anatomy on cephalometric radiographs in the supine and upright position. They did not find any significant differences in the cephalometric analysis of upright and supine patients.

In contrast Ono et al (1996) reported neck extension and anterosuperior movement of the hyoid bone in obstructive sleep apnoeic subjects with a Class I malocclusion. Yildirim et al (1991) measured the dimensions of the upper airway on subjects with obstructive sleep

apnoea and a control group without evidence of sleep disordered breathing. They found in all subjects the retropalatal airway became narrower on changing position from upright to supine, whilst the retroglosssal (oropharyngeal airway) space increased anteroposteriorly. A follow-up study conducted by the same group (Douglas et al, 1993) measured the effects of breathing and posture on the activity of the genioglossus muscle. They found the genioglossus had increased EMG activity supine compared with sitting in subjects with obstructive sleep apnoea and control subjects without sleep disordered breathing. They did not conclude that the increased genioglossus activity results in increased retroglossal space because other muscles attached to the hyoid may also be involved. The possibility is raised, however, that the genioglossus may act alone or in concert with other regional muscles to position the tongue and hyoid anterosuperiorly, accounting for the increase in retroglossal airway space noted on cephalometric radiographs. They postulated the increased EMG activity was due to the effects of gravity on the tongue mass, causing a reflex increase in tone.

Pae et al (1994) also compared lateral cephalometric radiographs taken upright and supine on subjects with obstructive sleep apnoea and a control group. They found significant differences between the two groups, both in terms of which dimensions altered and the degree to which they changed. In obstructive sleep apnoeic subjects the retropalatal anteroposterior dimension of the upper airway is significantly decreased when supine, as reported by Yildirim et al (1991). There was no significant change in this dimension in the control group. Interestingly there was a significant increase in soft palate thickness in the control group when supine which was not evident in the obstructive sleep apnoeic group. Similarly the obstructive sleep apnoeic subjects had a significantly decreased retroglossal (oropharyngeal) airway space when supine. This dimension did not change in the control group when cephalometric radiographs taken upright and supine were compared. Tongue cross-sectional area was correspondingly increased in obstructive sleep apnoeic subjects when supine, but not in controls. Oropharyngeal airway space was decreased significantly in both groups when supine, and did not differ significantly between the two groups. In the control group the hyoid was found to be anterosuperiorly displaced on changing from an upright to supine position. No such movement was demonstrated in the obstructive sleep apnoeic group.

Loube et al (1995) measured soft tissue variables on upright lateral cephalometric radiographs on obstructive sleep apnoea patients and control patients at different phases of the respiratory cycle. They found no significant differences in soft tissue measurements during inspiration or expiration in obstructive sleep apnoea patients or control subjects. They did however note that the length of the soft palate was significantly greater in obstructive sleep apnoea patients than control subjects.

The absence of teeth has also been reported to alter some parameters measured in the assessment of obstructive sleep apnoeic subjects. Tallgren et al (1983) reported the craniocervicofacial changes that occurred in the twelve months, as measured by lateral cephalometric radiograph, following the extraction of all teeth and provision of full dentures. These subjects were not investigated for the presence or absence of symptoms related to obstructive sleep apnoea. They found a mean increase in the distance from the cervical spine to the hyoid bone, suggesting this was due to upward and forward rotation of the mandible. The eighteen patients had no uniform change in head posture or cervical column posture. Individual patients with pronounced autorotation of the mandible did show a corresponding retroclination of the cervical column and decrease in the craniocervical angle.

Many authors have attempted to determine what influence, if any obesity plays in the upper airway structure of patients with obstructive sleep apnoea. Mayer et al (1996) studied 120 consecutive patients investigated for sleep disorders (94 obstructive sleep apnoea and 46 simple snorers) and found for all patients BMI significantly correlated with AHI at the 5% level. They also found the shape of the upper airway in awake subjects is more dependant on BMI than the AHI. When subjects were divided upon the basis of BMI and age, they found only those with a BMI $< 27$ kg/m$^2$ or less than 52 years old had significantly different upper airway anatomy. This study therefore concluded upper airway changes visible on lateral cephalometric radiographs or CT scan, in obstructive sleep apnoea patients, are independent of BMI.

Neck circumference has also been measured in a number of studies at the level of the cricothyroid membrane (Davies et al, 1992; Davies and Stradling, 1990; Katz et al, 1990; and Hoffstein and Mateika, 1992). Neck circumference has been compared to BMI as a predictor of incidence and severity of obstructive sleep apnoea. Davies et al (1992) used neck circumference corrected for height and reported this to be a better predictor of the presence of obstructive sleep apnoea than obesity alone as measured by the BMI. They studied 150 patients by questionnaire, polysomnography, BMI and neck circumference. They found a significant correlation between neck circumference corrected for height and obstructive sleep apnoea ($r2 = 0.38$).

This is in direct contrast to a similar study by Ono et al (1996) who examined 61 obstructive sleep apnoea patients and 10 control subjects and found a significant correlation (at the 5% level) between BMI and AHI. Lowe et al (1995) and Zucconi et al (1993) have also suggested a relationship between BMI and upper airway findings. These authors also found a significant correlation between BMI and neck circumference in all patients. The division of patients into groups according to neck circumference (presumed to be an indicator of obesity) was reported by Ferguson et al (1995). On the basis of their results they proposed three

groups of patients, agreeing with Tsuchiya et al (1992) but adding an intermediate group of patients with some craniofacial abnormalities and intermediate neck circumference (obesity).

The finding of an increased neck circumference in many subjects with an increased AHI and who are obese suggests there may be a relationship between these three variables (Katz et al, 1990).

The use of neck circumference as a measure of obesity has been compared to the use of abdominal obesity. There are conflicting findings over which measure is a more reliable predictor of obesity, particularly related to subjects with obstructive sleep apnoea. Hoffstein and Mateika (1992) found that both measures were significantly increased in subjects with obstructive sleep apnoea compared with non-apnoeic controls. When apnoeic and non-apnoeic subjects were matched exactly for BMI and age neck circumference had a greater predictive capacity for obstructive sleep apnoea. Grunstein et al (1993) reported waist circumference to be a better predictor of obstructive sleep apnoea than neck circumference or BMI. They concluded from their findings that the relationship between obesity and obstructive sleep apnoea cannot be explained by fat deposition in the neck alone.

Neck circumference was also been reported to be increased in a population of snoring subjects without evidence of obstructive sleep apnoea compared to non-snoring control subjects Zamarron et al, 2000). Davies and Stradling (1990) concluded that variation in neck circumference was likely to be of primary importance in determining the relationship between general obesity, hyoid position, soft palate length and OSA. Using multiple stepwise regression analysis they found only neck circumference and retroglossal space were independently significant correlates with decreases in arterial oxygen saturation during sleep.

Nelson and Hans (1997) studied 142 habitual snorers with and without evidence of apnoeic activity. They found obesity to be an independent variable important in increasing apnoeic activity. Furthermore they found the largest predictor of apnoeic activity in nonobese subjects to be tongue length, followed by middle cranial fossa alignment and age. Obese individuals had an increased mandibular plane to hyoid bone distance as the most reliable predictor, followed by tongue length. This paper suggests different variables measured from a lateral cephalometric radiograph may be of importance when considering obese and nonobese subjects.

This realization may necessitate the use of different cephalometric norms when comparing obese and nonobese patients for obstructive sleep apnoea indicators using lateral cephalometric radiographs. This practice is familiar for surgeons and orthodontists involved in the practice of surgical correction of dentofacial deformities. Different norms are used

when comparing, for example, Caucasian and south east Asian people cephalometrically during workup for orthognathic surgery.

Attempts have been made to correlate cephalometric and demographic data to produce a predictive model of patients suffering obstructive sleep apnoea. Battagel et al (1996) derived two four variant discriminant models were derived, both of which accurately predicted the presence of obstructive sleep apnoea. The first model used the whole population and used BMI, S-N, soft palate thickness and soft palate area. The second model used only those subjects suffering obstructive sleep apnoea with a BMI $< 25 kg/m^2$ , soft palate area, soft palate thickness and the intermaxillary space. If this model or one similar were to be developed it would greatly assist the identification of patients suffering obstructive sleep apnoea who may not otherwise be recognised.

From the preceding review of the literature it is obvious there is no consensus on the most appropriate investigation to visualize and identify the point of upper airway obstruction on patients with obstructive sleep apnoea. The conflicting findings between many groups using the same modality also point to possible problems with landmark identification, patient positioning and an understanding of the mechanism of upper airway collapse.

# Chapter 4

# Errors in Lateral Cephalometry

## 4.1 Introduction

Analysis of radiographs to diagnose and plan treatment as well as assessing treatment outcomes underpins a large volume of orthodontic and surgical literature. The ability to reproducibly duplicate radiographic records on the one patient, or on a group of patients allows meaningful comparison to be made between patients based upon the radiographic record.

The lateral cephalometric radiograph is widely used in the orthodontic and oral and maxillofacial surgery specialties. This radiograph is a cost effective way of imaging the dentoskeletal and soft tissue profile of a living subject, unlike other forms of imaging. Cross-sectional studies, such as this study, are possible because of the ability to obtain the radiograph with each patient in an identical, reproducible head position.

Brodie (1955) noted the lateral cephalometric radiograph permitted either cross-sectional or longitudinal (serial) evaluation of cranial changes. Brown (1965) noted the use of measurements based upon the lateral cephalometric radiograph were only useful if errors of estimation do not affect the true angles and distances measured.

Houston (1983) and Buschang (1987) divided errors associated with the measurement and interpretation of information from lateral cephalometric radiographs into two broad categories:

1. Systematic error; and
2. Random error.

Systematic errors can be minimized by standardizing equipment and technique (Brown et al, 1970) as they are introduced by observer and equipment bias. Observer bias most commonly arises because of unconscious weighting of the data obtained from the radiograph in order to support the hypothesis proposed by the researcher.

Random errors arise as chance events. Such events may arise because of incorrect positioning of the patient or the film in the cephalostat or inaccurate landmark identification due to poor film quality or equivocal landmark definition.

Gravely and Murray-Benzies (1974) proposed an alternate classification of cephalometric errors related specifically to cephalometry. Projection errors were said to arise from inaccuracies inherent in recording a three dimensional object (the skull) as a two dimensional image. The projection error varied according to the distance from the landmark to the film.

Tracing errors were said to arise from incorrect landmark identification and errors in the measurement of landmark distances and angles.

Battagel (1993) described the following as potential differences that may arise when comparing two groups of individuals using lateral cephalometric radiographs:

1. Between group – real differences between the samples;
2. Between individual – representing biological variation; and
3. With-in individual – representing the measurement error.

Obviously differences in group one and group two may be differences that are relevant to the investigation being undertaken and therefore are often not a result of error. What must be minimized in these groups is error associated with either concluding there is a difference when in fact no significant difference exists, or conversely failing to identify a significant difference between groups or individuals when one exists.

The third group of errors must be minimized or the study becomes irrelevant. The sources of measurement error when considering lateral cephalometric radiographs can be divided into six broad categories:

1. Errors of projection;
2. Errors of landmark identification;
3. Errors of digitizing;
4. Errors of measurement;
5. Errors attributable to operator variability; and
6. Errors of superimposition.

A variety of methods have been used in an attempt to overcome these inaccuracies, and these will be discussed in the following sections.

## 4.2 Errors of Projection

Projection errors may arise from misalignment of the x-ray source, the cephalostat, the film or the subject (Ahlqvist et al, 1983; Carlsson, 1967; and Eliasson et al, 1982).

Projection errors associated with lateral cephalometric radiography were defined by Carlsson (1967) as "those arising in the projection of the skull, and including the enlargement, departures from parallelity between the median and film planes, especially when the patient is

fitted on the cephalostat, deviations on the position of the focus in relation to an imaginary line through the ear rods, and geometric unsharpness due to the area of focus."

Baumrind and Frantz (1971a) also proposed "foreshortening of distances between points lying in different planes and by radial displacements of all points and structures not on the principal axis" were an addition source of projection error due to film distortion.

Positioning of the patient accurately within the cephalostat and correct loading of the film cassette are thus seen to be critical in minimizing the projection error. Both Carlsson (1967) and Ahlqvist (1986) have quantified the degree of error.

Carlsson (1967) found that whilst errors of projection occurred, these errors were small in relation to the total error of the method.

Ahlqvist et al (1986) concluded from theoretical calculations that head rotation in the cephalostat +\- five degrees results in an error less than 1%. Head positioning greater than five degrees from ideal results in a much greater error, however they noted that rotations of this magnitude should be obvious to the alert radiographer and thus corrected prior to film exposure.

A number of other authors (Houston et al, 1986; Midtgard et al, 1974; and Solow, 1966) also concluded that projection errors should not be statistically significant if the lateral cephalometric radiograph is taken carefully by an experienced radiographer.

## 4.3 Errors of Landmark Identification

Landmark identification has been discussed widely in the literature and may be a source of a number of errors, both systematic and random.

Random errors on lateral cephalometric radiograph interpretation relate to either to inaccurate landmark measurement or poorly defined landmarks leading to ambiguity in their identification (Baumrind and Frantz, 1971a; Brown et al, 1970; Chate, 1987; Houston, 1983; Midtgard et al, 1974; van der Linden, 1971; Vincent and West, 1987).

The source of random error associated with landmark identification has been most commonly found to result from imprecise landmark identification and inaccurate landmark definition (Broch et al, 1981; Chate, 1987; and Houston, 1983). These authors, amongst others (Baumrind and Frantz, 1971a; Miller and Baumrind, 1973; and Savage et al, 1987) have encouraged more precise landmark definition to minimize these errors. When considering the literature of lateral cephalometry related to OSA there is even more variation in landmark definition with respect to the soft tissue of the upper airway than in conventional landmark

definition of lateral cephalometry for orthodontic, anthropologic and orthognathic surgical use.

In conventional lateral cephalometry, the bony landmarks menton and pogonion, despite precise landmark definition, are in fact highly variable in location. This relates to the fact these points are located on a geometric shape (the mandibular symphysis) that may alter according to the horizontal reference plane and the degree of jaw opening (Moyers and Bookstein, 1979).

Inaccurate landmark identification has been reported to result in an average error of magnitude five times that ascribed to errors of measurement (Savara and Takeuchi, 1979). A characteristic pattern of error associated with landmark identification has been described by a number of authors (Baumrind and Frantz, 1971a; Broch et al, 1981; and Richardson, 1966). Broch et al (1981) concluded "the reliability of the landmark identification depends on five factors:

1. Characteristics of the cranial structures;
2. The general quality of the head plate;
3. Blurring of the anatomical structures caused by secondary radiation or movement during exposure;
4. Precise landmark recording; and
5. The accuracy of the operator.

The use of radiopaque liquids swallowed by the patient prior to exposure of the radiographic film has been used to enhance the visibility of soft tissue landmarks.

Cooke and Wei (1991) used retaken radiographs on 22 12 year-old children to determine the error in landmark identification between x number of points. They reported the error percentage for both dentoskeletal and soft tissue measurements was doubled, on average, on retaken radiographs compared with repeat measurements on the same radiograph.

They found, in common with other cephalometric studies, that landmarks in the midsagittal plane had the least variation in error. On remeasured radiographs the landmarks producing the greatest method error (>1 mm or 1°) were variables including Go, incisor long axes and the functional occlusal plane. On retaken radiographs the greatest method errors were for P-ANS and any measurement involving the Frankfort horizontal plane.

Miles et al (1995) reported significant error in the identification of the vertical position of the soft palate resulted in errors of measurement of soft palate length. This study used three separate investigators measuring landmarks commonly identified in the OSA literature one

week apart on 20 randomly selected radiographs and 10 superior quality radiographic films. ANOVA indicated most other landmarks could be reliably identified.

## 4.4 Errors of Digitizing

The digitization of radiographic landmarks is also used in a bid to improve the accuracy of cephalometric radiographs. The only source of error associated with the measurement of landmarks by digitizing is landmark identification (Bergin et al, 1978; Broch et al, 1981 and Richardson, 1981).

Broch et al (1981) replicated the co-ordinate system when digitizing and found the error was no greater than 0.03mm in either the x or y axes. A similar magnitude of error was reported by Savage et al (1987).

Richardson (1981) compared the accuracy of cephalometric measurements using a digitizer and traditional methods. He found little difference in accuracy between the two methods. The major source of recording error has been reported as point identification (Houston et al, 1986; Sandler, 1988), and this remains a constant problem irrespective of the method of recording the points.

Cohen (1994) and Sandler (1988) have found the accuracy of point identification is little affected by direct digitization or the use of an intermediate tracing stage prior to digitization.

## 4.5 Errors of Measurement

Digitizing cephalometric radiographs has almost eliminated measurement errors (Bondevik et al, 1981; and Broch et al, 1981). Double determination of points on a lateral cephalometric radiograph and digitizing both series followed by calculating the mean of the points has been suggested as the best method of eliminating the measurement error (Baumrind and Frantz, 1971a; 1971b).

1. Dahlberg's Statistic

Using this method, proposed in 1940 by Dahlberg, measurements of a single point are repeated on each radiograph for each of a series of patients. This measurement is compared and the standard deviation of each of the repeated measurements from its own pair mean is calculated. The formula for this measure of error is:

$$S(error) = \sqrt{\frac{diff^2}{2N}}$$

Se is the standard deviation of the difference of each of the two measurements from their mean; d is the difference between the first and second measurement; and n is the number of radiographs recorded.

2. Chebib and Burdick's Method

This method describes the error associated with a single variable using a number of repeated measurements. This is represented mathematically as:

$$e = \sqrt{\frac{2s}{k}}$$

e is the expected error associated with each measurement; s is the standard deviation of the error of each measurement; and k is the number of times each point is measured.

The Dahlberg and Chebib and Burdick equations are mathematically related. When radiographs are digitized twice the use of Chebib and Burdick's method approximately halves the error reported compared with the use of Dahlberg's equation.

3. Houston Estimate of Random Error

This estimate is described as the variance of the difference between repeated measurements. Mathematically the equation derived is identical to that described by Dahlberg.

4. Bjork's Quotient

Described in 1947 this method uses repeat measurements to compare the standard error of the mean differences and the mean differences themselves. This method is based upon the assumption that for a difference to be significant it must be three times the standard error of the measurement.

5. Houston's Estimate of Systematic Error

Houston separates systematic and random error, unlike Dahlberg and Chebib and Burdick. Houston used a one sample t test between a number of repeated measurements and examined them at the 10% level of significance.

### 4.5.1 The Co-efficient of Reliability

The reliability of a measurement assessment may also be used to express the accuracy of a measurement. Midtgard et al (1974) related the error between two separate measurements and the variance of the landmark measurement in the sample population.

### 4.5.2 Confidence Limits

A confidence limit within which a measurement lies can be determined by the product of the Dahlberg statistic and a value of t dependent upon the level of probability chosen and the number of the sample. The 95% confidence level is that most frequently chosen, the t value is obtained from the 0.05 level of significance column in a standard t table.

Battagel (1993) analyzed 246 radiographs using the above methods and determined that mathematically the Dahlberg statistic is the most accurate means of evaluating measurement error. The limiting factor in using the Dahlberg statistic only is its inability to take into account the proportionate size of the error in relation to the measurement itself. The use of the coefficient of reliability as proposed by Midtgard was found to be useful, provided its limitations are also recognized. These limitations are most apparent in studies with a small population with large differences in the indices measured. Widening the scope of the study increases the variance and improves the coefficient of reliability. In a study with widely disparate samples this may not be helpful, and breaking the population into subgroups, such as by Angle Classification may improve the relevance of the coefficient of reliability.

Trpkova et al (1997) used meta analysis to identify the accuracy of localization of cephalometric landmarks in the x and y plane. This analysis was also used to determine which landmarks were most easily and accurately identified and therefore which landmarks are most likely to be useful landmarks when comparing results between studies. Fifteen bony landmarks commonly quoted in the literature when referring to the cephalometric radiograph were assessed.

The results showed that an acceptable error when plotting any of these landmarks was 0.59 mm total error for the x co-ordinate and 0.56 mm total error for the y co-ordinate.

Earlier, Midtgard et al (1974) digitized landmarks on tracings of 25 lateral cephalometric films and found the variance of the error as a percent of the total variance was less than three per cent for four of seven distances. Distances from nasion to point A and nasion to point B were found to exceed this variance and it was attributed to difficulty in landmark identification.

Errors of measurement are increased for points not located on the midsagittal plane due to magnification. Bergersen (1980) measured these errors using lateral and frontal cephalometric radiographs. He found errors in all measured planes did not exceed seven per cent and formulated tables to allow compensation for these errors.

## 4.6 Intra-observer and Inter-observer Variability

Differences in landmark identification has been determined by having a single person trace and identify points on a lateral cephalometric radiograph on two separate occasions and two or more people trace and identify points on the same lateral cephalometric radiograph. The former is used to determine intra-observer variability whilst the latter measures inter-observer variability.

Errors have been found both intra-observer (Solow, 1966; Stabrun and Danielsen, 1982) and inter-observer (Baumrind and Frantz, 1971b; Stabrun and Danielsen, 1982; Vincent and West, 1987). These studies suggest that intra-observer error is generally less than inter-observer errors. Savage et al (1987) and most other authors ascribe this finding to different interpretation of landmark location.

# Chapter 5

# Lateral Cephalometric Examination of the Upper Airway – Hard Tissue

## 5.1 Introduction

The use of lateral cephalometric radiographs in the assessment of the skeletal relationships and the soft tissue profile is well established. The lateral cephalometric radiograph is easily obtained and there are well defined landmarks that can be identified and measurements taken to allow comparison of skeletal and soft tissue position. These measurements are easily compared with known norms for a particular population. Oral and Maxillofacial surgeons and Orthodontists routinely utilise these radiographs in diagnosis and planning of procedures for patients undergoing treatment for a skeletal based malocclusion.

One of the difficulties in the surgical management of the obstructive sleep apnoea patient is determining the site of upper airway obstruction. The lateral cephalometric radiograph has been utilised by a large number of investigators in attempts to measure differences in position of reliable skeletal and soft tissue landmarks. If there are significant differences in skeletal or soft tissue position between obstructive sleep apnoea patients and controls with out obstructive sleep apnoea it has been postulated a lateral cephalometric radiograph may identify the areas of significance.

A large number of authors have used lateral cephalometric radiographs to assess the skeletal and soft tissue dimensions in the obstructive sleep apnoeic patient. Unfortunately not all studies have utilised the same anatomic points to measure these dimensions. Direct comparison between many of these studies is therefore difficult.

The literature concerning the assessment of the skeleton and upper airway on subjects with obstructive sleep apnoea has been reviewed. The results of this review will be presented by anatomic area. The skeletal components of the cranium, facial bones and cervical spine that are usually assessed from lateral cephalometric radiographs are the cranial base, the maxilla, mandible and the inter-relationship between these two structures, facial height, bony pharyngeal dimensions, dental measurements and the cervical spine. Linear and angular measures can be taken for many of these structures and will be presented under the appropriate section.

## 5.2 Cranial Base

The cranial base serves as the superior attachment for the muscles of the pharynx posteriorly and anteriorly serves as the upper extent of the facial skeleton. Obstructive sleep apnoea, as mentioned in Chapter 3, has been associated with various craniofacial anomalies and it has been suggested by Shprintzen (1992) and Glander and Cisneros (1992) that the apnoea may be associated with cranial base abnormalities.

The anterior cranial base extends from nasion anteriorly at the junction of the nasal bones and nasal process of the frontal bone to sella, the midpoint of the pituitary fossa in the sphenoid bone. The posterior cranial base extends from sella anteriorly to basion at the anterior margin of foramen magnum. Other points have been identified on the cranial base and used in cephalometric studies of subjects with obstructive sleep apnoea and are fully described in the glossary.

A number of authors have measured the length of the anterior and posterior cranial base and found the length to be significantly shorter in obstructive sleep apnoea subjects. Andersson and Brattstrom (1991); Bacon et al (1989); Battagel and L'Estrange (1996); Sakakibara et al (1999); Tangugsorn et al (1995a); and Zucconi et al (1993) reported obstructive sleep apnoea patients had a significantly shorter anterior cranial base. Sakakibara et al (1999) found no significant difference in the length of the anterior cranial base between obese obstructive sleep apnoea subjects and controls. Zucconi et al (1993) reported no significant difference between obstructive sleep apnoea subjects and non-snoring controls, however there was a significantly shorter anterior cranial base in obstructive sleep apnoea subjects when compared with snoring controls. Other authors have found no significant difference in anterior cranial base length (deBerry-Borowiecki et al, 1988; and Strelzow et al, 1988).

The posterior cranial base is less commonly decreased in length in obstructive sleep apnoea subjects (Andersson and Brattstrom, 1991; and Tangugsorn et al, 1995a). Sakakibara et al (1999) also considered this parameter and found no significant difference in length between any of the groups studied.

Other linear measurements of the cranial base have reported by a few authors. deBerry-Borowiecki et al (1988) measured the length of the anterior portion of the anterior cranial base. They found no significant difference in this length between obstructive sleep apnoea subjects and controls. This group of authors also measured the distance from sella to articulare and found no difference between obstructive sleep apnoea subjects and controls. This dimension was also measured by Sakakibara et al (1999) and no difference was found between any group of subjects.

The total length of the cranial base, from nasion to basion has been found significantly decreased by Sakakibara et al (1999) only between obese and non-obese obstructive sleep apnoea subjects. This length was also significantly shorter in the population studied by Tangugsorn et al (1995a) and Zucconi et al (1993).

Overall the literature indicates the cranial base may be shorter anteroposteriorly in obstructive sleep apnoea subjects when compared to control populations.

Table 5.2-1 records the authors and the linear measurements of the cranial base. Definitions of the landmarks used may be found in the glossary.

**Table 5.2-1: Cranial Base – Linear Measurements**

| | | | S-N | S-Ba | N-SC | Ar-S | N-Ba |
|---|---|---|---|---|---|---|---|
| Andersson et al (1991) | A vs C | U | 0.001 | 0.01 | | | |
| Bacon et al (1989) | A vs C | U | 0.001 | | | | |
| Battagel et al (1996) | A vs C | U | 0.01 | | | | |
| deBerry-Borowiecki et al (1988) | A vs C | U | NS | | NS | NS | |
| Sakakibara et al (1999) | A1 vs C | U | 0.001 | NS | | NS | NS |
| Sakakibara et al (1999) | A2 vs C | U | NS | NS | | NS | NS |
| Sakakibara et al (1999) | A1 vs A2 | U | 0.001 | NS | | NS | 0.001 |
| Strelzow et al (1988) | A vs C | U | NS | | | | |
| Tangugsorn et al (1995a) | A vs C | U | 0.001 | 0.001 | | | 0.001 |
| Zucconi et al (1993) | A vs C | U | NS | | | | 0.01 |
| Zucconi et al (1993) | A vs S | U | 0.05 | | | | 0.01 |

A = obstructive sleep apnoea subject; C = non-snoring, non-apnoeic subject; S = snoring, non-apnoeic subject; A1 = non-obese obstructive sleep apnoea subject; A2 = Obese obstructive sleep apnoea subject; A3 High apnoea index / Low BMI; A4 Low apnoea index / High BMI.

The angular measurements of the cranial base are determined by the angle between three landmarks, a plane with true horizontal or vertical planes or by the angle between two planes. The angular measures formed by landmarks are the angle between nasion – sella – basion (N-S-Ba) or the angle between nasion – sella – articulare (N-S-Ar). The plane sella – nasion may be compared to a true vertical plane (SN/Ver) whilst the Frankfort Horizontal may be compared to a true horizontal plane (FH/Hor). The angle formed by the intersection of the planes sella – nasion and Frankfort Horizontal are also compared. A final angle formed by the planes of the middle cranial fossa can be measured from a lateral cepahalometric radiograph.

The cranial base angle (N-S-Ba) has been found to be significantly more acute in patients with obstructive sleep apnoea compared with control subjects (Andersson and Brattstrom, 1991; Battagel and L'Estrange, 1996; Steinberg and Fraser, 1995). This finding however, has not been duplicated in all populations studied (Bacon et al, 1989; deBerry-Borowiecki et al, 1988; Hochban and Brandenburg, 1994; Johns et al, 1998; Lyberg et al, 1989a; Pracharktam et al, 1994; Pracharktam et al, 1996; Sakakibara et al, 1999; Tangugsorn et al, 1995a; and Zucconi et al, 1993).

A divergent facial skeleton, as measured by the angle between the cranial base and Frankfort Horizontal has been reported in some studies (deBerry-Borowiecki et al, 1988; and Lyberg et al, 1995a). Mayer and Meier-Ewert (1995) and Strelzow et al (1988) did not find this comparison significantly different in their population. The angulation of the Frankfort horizontal compared with true horizontal was not found significantly different in obstructive sleep apnoea subjects studied by Pracharktam et al (1994).

Pracharktam et al (1994) reported obstructive sleep apnoea subjects had a retruded mandible as measured by MCF, a relationship between the jaws and the nasion-sella line. This was not supported by their research conducted in 1996 (Pracharktam et al, 1996).

The inclination of the anterior cranial base relative to true vertical has been measured by Sakakibara et al (1999) and Tangugsorn et al (1995a). The latter authors found a significantly steeper anterior cranial base inclination as measured by this angle in obstructive sleep apnoea subjects. The former group reported no significant difference in anterior cranial base inclination as measured by this parameter.

Table 5.2-2 records the authors and the angular measurements of the cranial base. Definitions of the landmarks used may be found in the glossary. Figure 5.2-1 shows the measurements of the cranial base diagrammatically.

**Table 5.2-2: Cranial Base – Angular Measurements**

| | | | Ba-S-N | SN/FH | N-S-Ar | MCF | FH/Hor | SN/Ver |
|---|---|---|---|---|---|---|---|---|
| Andersson et al (1991) | A vs C | U | 0.01 | | | | | |
| Battagel et al (1996) | A vs C | U | 0.01 | | | | | |
| deBerry-Borowiecki et al (1988) | A vs C | U | | 0.045 | NS | | | |
| Hochban et al (1994) | A vs C | U | NS | | | | | |
| Johns et al (1998) | A vs S | U | NS | | | | | |
| Lyberg et al (1995a) | A vs C | U | NS | 0.05 | | | | |
| Mayer et al (1995) | A vs C | U | | NS | | | | |
| Pracharktam et al (1994) | A vs S | U | NS | | | 0.01 | | |
| Pracharktam et al (1996) | A vs S | U | NS | | | NS | NS | |
| Sakakibara et al (1999) | A1 vs C | U | NS | | | | | NS |
| Sakakibara et al (1999) | A2 vs C | U | NS | | | | | NS |
| Sakakibara et al (1999) | A1vs A2 | U | NS | | | | | NS |
| Steinberg et al (1995) | A vs C | U | 0.0001 | | | | | |
| Strelzow et al (1988) | A vs C | U | | NS | | | | |
| Tangugsorn et al (1995a) | A vs C | U | NS | | | | | 0.01 |
| Zucconi et al (1993) | A vs C | U | NS | | | | | |
| Zucconi et al (1993) | A vs S | U | NS | | | | | |

A = obstructive sleep apnoea subject; C = non-snoring, non-apnoeic subject; S = snoring, non-apnoeic subject; A1 = non-obese obstructive sleep apnoea subject; A2 = Obese obstructive sleep apnoea subject; A3 High apnoea index / Low BMI; A4 Low apnoea index / High BMI.

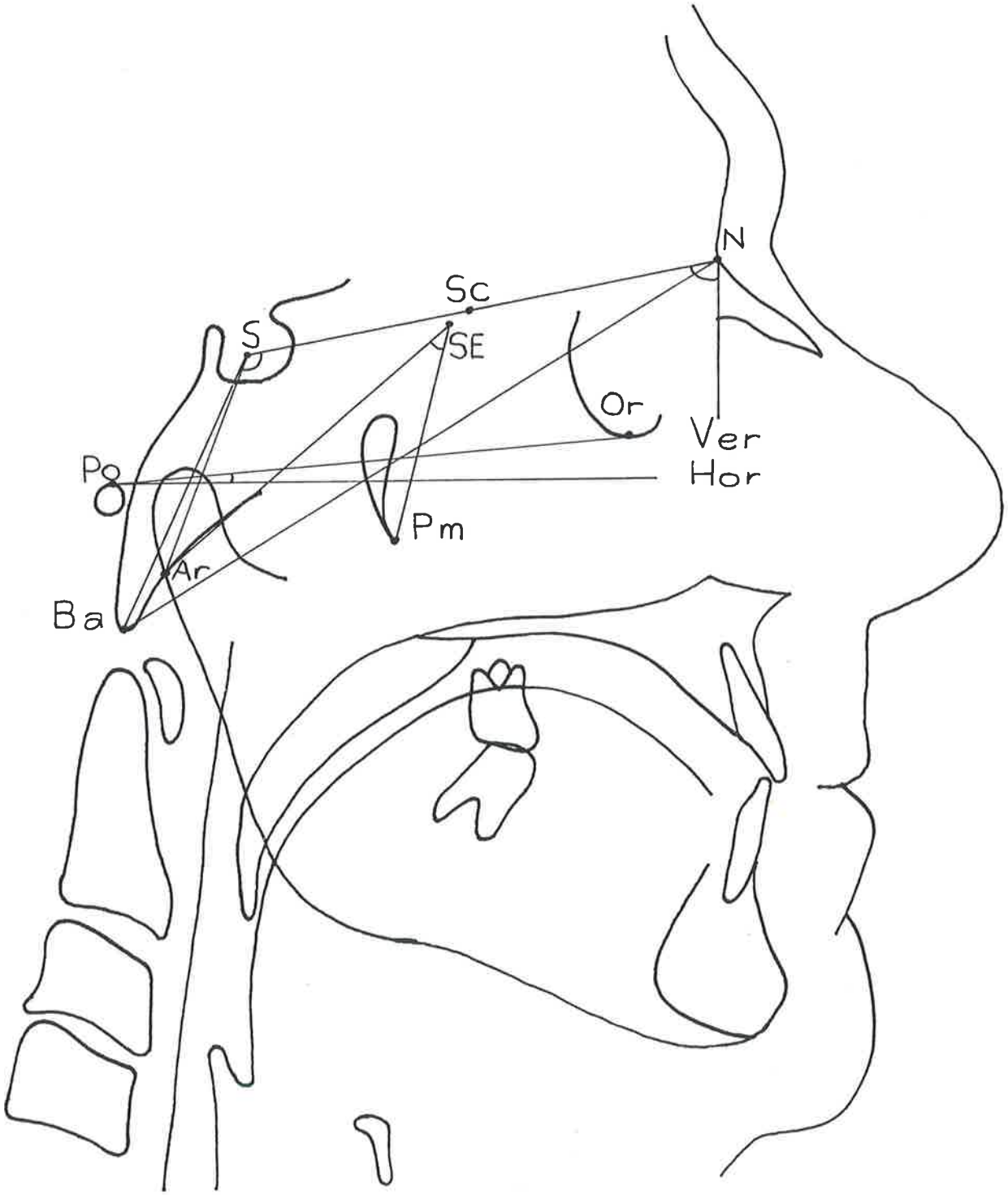


**Figure 5.2-1 Cranial base cephalometric measures**

## 5.3 Maxilla

The maxilla is a paired bone consisting of a body and four processes. The body is hollow, forming the maxillary antrum and roughly pyramidal in shape. The base of the pyramid lies anteriorly and the apex lies caudally. The superior surface of the body of the maxilla forms part of the floor of the orbit. The medial surface of the maxilla forms part of the lateral nasal wall. The anterior surface forms the external surface of the maxilla whilst the posterior surface of the maxilla forms the anterior wall of the infratemporal fossa.

The maxilla has four processes that arise from the body. The frontal process projects upwards to articulate with the frontal bone. These processes form part of the medial orbital wall, the lateral nose and the nasal bridge behind the nasal bones. The zygomatic process of the maxilla projects laterally to form the anterior portion of the zygomatic arch. This process articulates with the zygomatic bone, which forms the central portion of the zygomatic arch. The palatine process is a horizontal bony shelf that projects medially to articulate with the palatine process of the contralateral maxilla. The two palatine processes form the anterior two thirds of the palate. The maxilla, like the mandible, has an alveolar process that develops with the eruption of teeth and is resorbed following the loss of teeth. Thus the vertical height of the maxilla will vary depending upon the presence or absence of teeth.

The anterior nasal spine is a midline structure formed by the left and right maxillae. It is located immediately inferior to the anterior nasal aperture. The posterior nasal spine is a similar structure located at the posterior extent of the bony palatine processes of the maxilla. Both these structures provide landmarks useful in cephalometry.

The absolute length of the bony maxilla can be measured from the posterior nasal spine to the anterior nasal spine or to point A, the point of maximum concavity on the anterior maxilla below the anterior nasal spine. The cephalometric studies that consider this measurement are not conclusive regarding a difference between obstructive sleep apnoea subjects and controls. Andersson and Brattstrom (1991); deBerry-Borowiecki et al (1988); Lowe et al (1996); Pracharktam et al (1994); Sakakibara et al (1999); Strelzow et al (1988); Tangugsorn et al (1995a); and Tsuchiya et al (1992) found obstructive sleep apnoea subjects had a decreased anteroposterior length of the maxilla. In contrast Johns et al (1998); Lowe et al (1995); Lyberg et al (1989a); Mochizuki et al (1996); Pracharktam et al (1996); and Zucconi et al (1993) found no difference in maxillary length. Interestingly obstructive sleep apnoea subjects who had a lateral cephalometric radiograph taken supine did not have a decreased maxillary length, but did when the same subject was radiographed upright (Lowe et al, 1996). No explanation or theory was offered as to the aetiology of this finding. The most likely

explanation would be an observer error in location of the landmarks on the maxilla used to define the length of the maxilla. All obstructive sleep apnoea subjects in this study with a Class III dental malocclusion had no significant difference in maxillary length when measured from a supine or upright radiograph. Sakakibara et al (1999) found no significant difference between obese obstructive sleep apnoea subjects and controls, or between obese and nonobese obstructive sleep apnoea subjects. Tsuchiya et al (1992) reported low AHI/high BMI subjects did not have a significantly shorter maxilla than a control population, and there was no significant difference in maxillary length between the two obstructive sleep apnoea groups.

Bacon et al (1989) found obstructive sleep apnoea subjects in their population had a significantly shorter face measured anteroposteriorly compared with a nonsnoring control population. Table 5.3-1 records the authors and the linear measurements of maxillary position. Definitions of the landmarks used may be found in the glossary.

**Table 5.3-1: Maxilla – Linear Measurements**

| | | | ANS-PNS | Dc - A | PNS – A |
|---|---|---|---|---|---|
| Andersson et al (1991) | A vs C | U | 0.01 | | |
| Bacon et al (1989) | A vs C | U | | 0.01 | |
| deBerry-Borowiecki et al (1988) | A vs C | U | 0.001 | | |
| Johns et al (1998) | A vs S | U | NS | | |
| Lowe et al (1995) | A vs C | U | NS | | |
| Lowe et al (1996) I | A vs C | U | 0.002 | | |
| Lowe et al (1996) I | A vs C | S | NS | | |
| Lowe et al (1996) II | A vs C | U | 0.031 | | |
| Lowe et al (1996) II | A vs C | S | NS | | |
| Lowe et al (1996) III | A vs C | U | NS | | |
| Lowe et al (1996) III | A vs C | S | NS | | |
| Lyberg et al (1995a) | A vs C | U | NS | | |
| Mochizuki et al (1996) | A vs S | U | | | NS |
| Pracharktam et al (1994) | A vs S | U | | | 0.05 |
| Pracharktam et al (1996) | A vs S | U | | | NS |
| Sakakibara et al (1999) | A1 vs C | U | 0.001 | | |
| Sakakibara et al (1999) | A2 vs C | U | NS | | |
| Sakakibara et al (1999) | A1vs A2 | U | NS | | |
| Strelzow et al (1988) | A vs C | U | 0.05 | | |
| Tangugsorn et al (1995a) | A vs C | U | 0.01 | | |
| Tsuchiya et al (1992) | A3 vs C | U | 0.05 | | |
| Tsuchiya et al (1992) | A4 vs C | U | NS | | |
| Tsuchiya et al (1992) | A3 vs A4 | U | NS | | |
| Zucconi et al (1992) | A vs C | U | NS | | NS |
| Zucconi et al (1992) | A vs S | U | NS | | 0.05 |

A = obstructive sleep apnoea subject; C = non-snoring, non-apnoeic subject; S = snoring, non-apnoeic subject; A1 = non-obese obstructive sleep apnoea subject; A2 = Obese obstructive sleep apnoea subject; A3 High apnoea index / Low BMI; A4 Low apnoea index / High BMI.

The position of the maxilla, particularly in its anteroposterior dimension is of interest because of the postulate that a retrognathic maxilla may contribute to obstruction of the upper airway at the level of the soft palate. The simplest measure of the anteroposterior position of the maxilla is in relation to the cranial base. The maxilla was found to be retrognathic in obstructive sleep apnoea subjects by deBerry-Borowiecki et al (1988); Hochban and Brandenburg (1994); Strelzow et al (1988) and Tsuchiya et al (1992). The majority of authors who have considered this parameter have not demonstrated significant retrognathia (Bacon et al, 1989; Johns et al, 1998; Lowe et al, 1995; Lyberg et al, 1989a; Maltais et al, 1991; Mayer and Meier-Ewert, 1995; Mochizuki et al, 1996; Ono et al, 1996; Pracharktam et al, 1996; Sakakibara et al, 1999; Tangugsorn et al, 1995a; Zucconi et al, 1992; and Zucconi et al, 1993). Tsuchiya et al (1992) reported a significantly retrognathic maxilla in a subgroup of obstructive sleep apnoea subjects with a high AHI/low BMI. The maxillary depth, measured by comparing the inclination of a line from nasion to point A relative to Frankfort horizontal, was not significantly different between obstructive sleep apnoea subjects and controls (Hochban and Brandenburg, 1994).

The effective anteroposterior dimension of the maxilla can be quantified by measuring the angle between the palatal plane and the vertical line from the sphenoethmoidal junction through the pterygomaxillary fissure (Pracharktam et al, 1994). The palatal plane was significantly rotated counter clockwise in obstructive sleep apnoea subjects compared with a control population. This indicates a significantly shorter effective dimension of the maxilla in obstructive sleep apnoea subjects.

The maxillary angulation has also been considered relative to horizontal planes, either the cranial base (sella – nasion) or Frankfort horizontal. Hochban and Brandenburg (1994) have reported a dorsocaudal rotation of the palatal plane relative to the cranial base in obstructive sleep apnoea patients. This means the maxillary length is effectively decreased. Bacon et al (1989) and Lowe et al (1996) did not find this angle to differ significantly in obstructive sleep apnoea populations studied. Interestingly Hochban and Brandenburg (1994) did not find a difference in palatal plane inclination relative to Frankfort horizontal.

The angulation between the posterior wall of the maxilla and the constructed cranial base Ar-N was considered by Pracharktam et al (1994) and Pracharktam et al (1996). In the first study this angle was more obtuse in obstructive sleep apnoea patients, indicating a more caudal position of the posterior maxilla in this group. The second study failed to demonstrate a significant difference in this dimension between obstructive sleep apnoea subjects and controls.

Table 5.3-2 records the authors and the angular measurements of maxillary position. Definitions of the landmarks used may be found in the glossary. Figure 5.3-1 shows the cephalometric measurements of the maxilla.

**Table 5.3-2: Maxilla – Angular Measurements**

| | | | SNA | A – P | SN/ANS-PNS | Inclination |
|---|---|---|---|---|---|---|
| Bacon et al (1989) | A vs C | U | NS | | NS | |
| deBerry-Borowiecki et al (1988) | A vs C | U | 0.018 | | | |
| Hochban et al (1994) | A vs C | U | 0.05 | NS[1] | 0.02 | NS[2] |
| Johns et al (1998) | A vs S | U | NS | | | |
| Lowe et al (1995) | A vs C | U | NS | | | |
| Lowe et al (1996) I | A vs C | U | | | NS | |
| Lowe et al (1996) I | A vs C | S | | | NS | |
| Lowe et al (1996) II | A vs C | U | | | NS | |
| Lowe et al (1996) II | A vs C | S | | | NS | |
| Lowe et al (1996) III | A vs C | U | | | NS | |
| Lowe et al (1996) III | A vs C | S | | | NS | |
| Lyberg et al (1995a) | A vs C | U | NS | | | |
| Maltais et al (1991) | A vs C | U | NS | | | |
| Mayer et al (1995) | A vs C | U | NS | | | |
| Mochizuki et al (1996) | A vs S | U | NS | | | |
| Ono et al (1996) | A vs C | U | NS | | | |
| Pracharktam et al (1994) | A vs S | U | | 0.05[3] | | 0.01[4] |
| Pracharktam et al (1996) | A vs S | U | NS | | | NS[4] |
| Sakakibara et al (1999) | A1 vs C | U | NS | | | |
| Sakakibara et al (1999) | A2 vs C | U | NS | | | |
| Sakakibara et al (1999) | A1vs A2 | U | NS | | | |
| Strelzow et al (1988) | A vs C | U | 0.05 | | | |
| Tangugsorn et al (1995a) | A vs C | U | NS | | | |
| Tsuchiya et al (1992) | A3 vs C | U | 0.05 | | | |
| Tsuchiya et al (1992) | A4 vs C | U | NS | | | |
| Tsuchiya et al (1992) | A3 vs A4 | U | NS | | | |
| Zucconi et al (1992) | A vs C | U | NS | | | |
| Zucconi et al (1993) | A vs S | U | NS | | | |

A = obstructive sleep apnoea subject; C = non-snoring, non-apnoeic subject; S = snoring, non-apnoeic subject; A1 = non-obese obstructive sleep apnoea subject; A2 = Obese obstructive sleep apnoea subject; A3 High apnoea index / Low BMI; A4 Low apnoea index / High BMI.

1 FH/NA; 2 ANS-PNS/FH; 3 ANS-PNS/PM; 4 Ar-N/PM

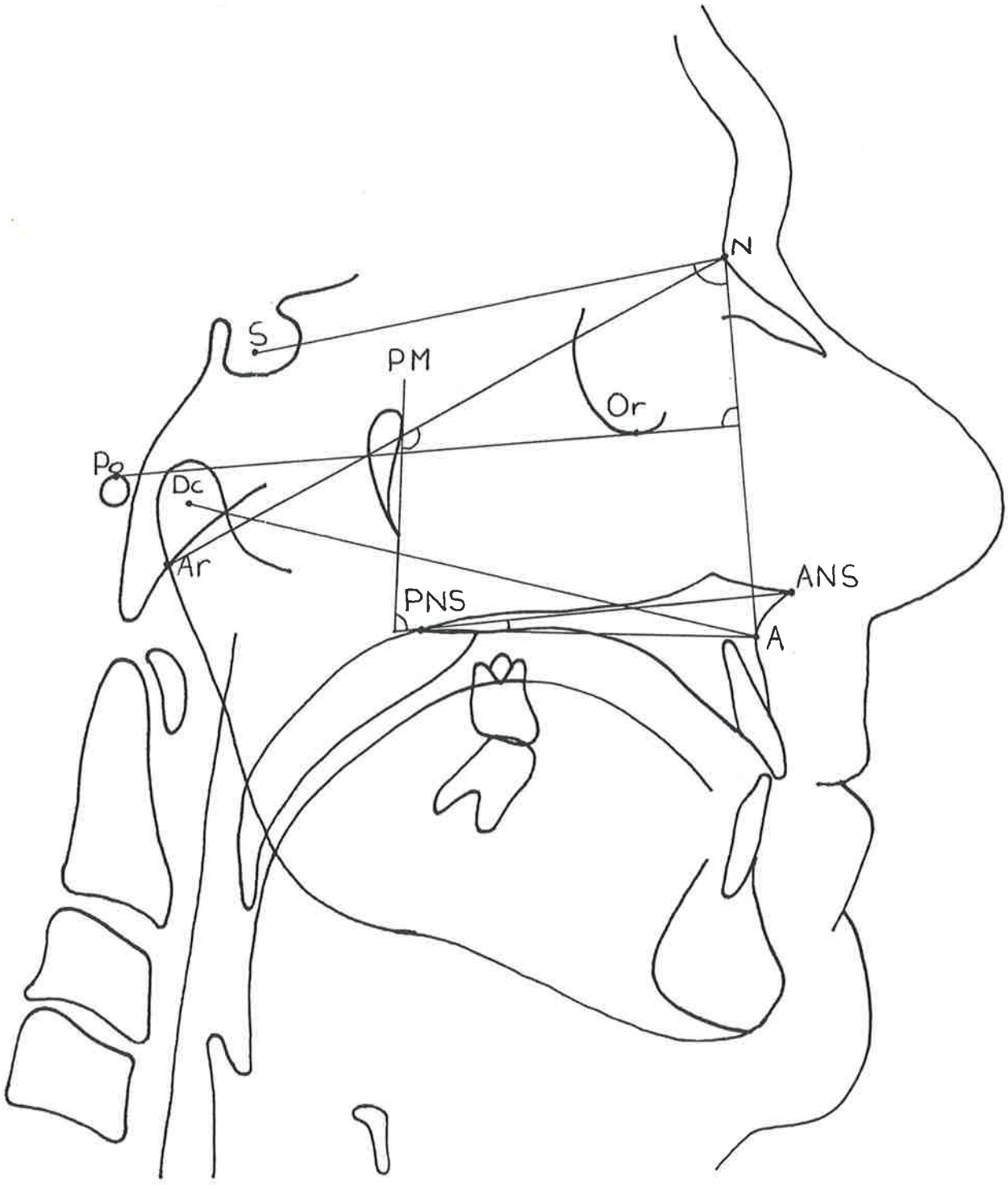


**Figure 5.3-1 Maxillary cephalometric measures**

## 5.4 Mandible

The mandible is an unpaired bone consisting of a horizontal body and two vertical rami. The body is U-shaped when viewed axially whilst the rami are flared laterally and longer anteroposteriorly than mediolaterally. A projection superiorly from the ramus, the condylar neck is crowned with the articular element of the mandible, the condyle. A bone projection from the anterior superior border of the ramus, named the coronoid process, serves as the mandibular insertion of the temporalis muscle. Similar to the maxilla, the mandible also has an alveolar process that forms the bony support for the dentition.

Investigators seeking a possible anatomic pathologic basis for obstructive sleep apnoea have extensively studied the size of the mandible and its position relative to other craniofacial structures.

The length of the mandible has been considered in totality, measuring from the mandibular condyle to the mandibular symphseal region. Andersson and Brattstrom (1991) report the total length of the mandible is significantly shorter in patients with obstructive sleep apnoea compared with control subjects. This finding was not reported in other studies that measured total mandibular length (Bacon et al, 1989; Johns et al, 1998; Lyberg et al, 1989a; and Tangugsorn et al, 1995a).

The conventional understanding of mandibular length from a cephalometric radiograph is in an anteroposterior direction, measured from the gonion, or angle of the mandible to the symphysis anteriorly. This length has been measured significantly shorter in obstructive sleep apnoea subjects by a number of authors (Andersson and Brattstrom, 1991; Battagel and L'Estrange, 1996; Lowe et al, 1996; Sakakibara et al, 1999; Strelzow et al, 1988; Tangugsorn et al, 1995a; Zucconi et al, 1992; and Zucconi et al, 1993). Other obstructive sleep apnoea populations have not had a significantly shorter mandibular body when compared with a control group (deBerry-Borowiecki et al, 1988; Lowe et al, 1995; Lyberg et al, 1989a; and Tsuchiya et al, 1992). A number of different landmarks have been used as anterior and posterior limits for this linear measurement on the mandibular body.

Lowe et al (1996) found the mandibular body was significantly shorter in only one group of obstructive sleep apnoea patients – those with a class I dental malocclusion who had an upright cephalometric radiograph. Sakakibara et al (1999) found no significant decrease in mandibular length in any obstructive sleep apnoea patient when measured from gonion to menton. Using the same population and measuring mandibular length the same study reported a decrease in mandibular length when measured from gonion to retrognathion. This decreased mandibular length was found in nonobese obstructive sleep apnoea subjects

compared to controls and to obese obstructive sleep apnoea subjects. Tangugsorn et al (1995a) reported no significant decrease in mandibular body length from gonion to prognathion, however pogonion was significantly more caudal in obstructive sleep apnoea subjects than controls when measured from nasion perpendicular.

The vertical height of the ramus of the mandible is increased in some populations (deBerry-Borowiecki et al, 1988; and Strelzow et al, 1988). Lowe et al (1995) and Sakakibara et al (1999) did not find this dimension significantly different in any of the groups studied. The height of the anterior mandible has also been reported to be significantly increased in subjects with obstructive sleep apnoea when measured from the tip of the lower central incisor to the lower border of the mandibular symphysis (Andersson and Brattstrom, 1991).

Pracharktam et al (1994) and Pracharktam et al (1996) considered the ratio between the ramus width anteroposteriorly and the length of the middle cranial fossa. They found the ramus width to be significantly smaller in the obstructive sleep apnoea population in the earlier study and suggested this measurement confirmed a smaller dimension of the oropharyngeal airway at this level.

Table 5.4-1 records the authors and the linear measurements of mandibular position. Definitions of the landmarks used may be found in the glossary.

**Table 5.4-1: Mandible – Linear Measurements**

| | | | Total Length | AP Length | Vertical - Anterior | Ramus Height | Proportion |
|---|---|---|---|---|---|---|---|
| Andersson et al (1991) | A vs C | U | 0.01[1] | 0.001[2] | | | |
| Andersson et al (1991) | A vs S | U | | | 0.05[3] | | |
| Battagel et al (1996) | A vs C | U | | 0.002[4] | | | |
| Battagel et al (1996) | A vs C | U | | 0.002[5] | | | |
| deBerry-Borowiecki et al (1988) | A vs C | U | | NS[2] | | 0.020[6] | |
| deBerry-Borowiecki et al (1988) | A vs C | U | | NS[5] | | | |
| Johns et al (1998) | A vs S | U | NS[1] | | | | |
| Lowe et al (1995) | A vs C | U | | NS[2] | | NS | |
| Lowe et al (1996) I | A vs C | U | | 0.004[2] | | | |
| Lowe et al (1996) I | A vs C | S | | NS[2] | | | |
| Lowe et al (1996) II | A vs C | U | | NS[2] | | | |
| Lowe et al (1996) II | A vs C | S | | NS[2] | | | |
| Lowe et al (1996) III | A vs C | U | | NS[2] | | | |
| Lowe et al (1996) III | A vs C | S | | NS[2] | | | |
| Lyberg et al (1995a) | A vs C | U | NS[7] | NS[8] | | | |
| Pracharktam et al (1994) | A vs C | U | | | | | 0.01[9] |
| Pracharktam et al (1996) | A vs C | U | | | | | NS[9] |
| Sakakibara et al (1999) | A1 vs C | U | | NS[4] | | NS[6] | |
| Sakakibara et al (1999) | A2 vs C | U | | NS[4] | | NS[6] | |
| Sakakibara et al (1999) | A1 vs A2 | U | | NS[4] | | NS[6] | |
| Sakakibara et al (1999) | A1 vs C | U | | 0.001[10] | | | |
| Sakakibara et al (1999) | A2 vs C | U | | NS[10] | | | |
| Sakakibara et al (1999) | A1 vs A2 | U | | 0.001[10] | | | |
| Strelzow et al (1988) | A vs C | U | | 0.05[2] | | 0.05[6] | |
| Strelzow et al (1988) | A vs C | U | | 0.05[5] | | | |
| Tangugsorn et al (1995a) | A vs C | U | NS[7] | NS[8] | | | |
| Tangugsorn et al (1995a) | A vs C | U | | 0.05[11] | | | |
| Tsuchiya et al (1992) | A3 vs C | U | | NS[2] | | | |
| Tsuchiya et al (1992) | A4 vs C | U | | NS[2] | | | |
| Tsuchiya et al (1992) | A3 vs A4 | U | | NS[2] | | | |
| Zucconi et al (1992) | A vs C | U | | 0.05[2] | | | |
| Zucconi et al (1992) | A vs C | U | | 0.05[4] | | | |
| Zucconi et al (1993) | A vs S | U | | 0.01[2] | | | |
| Zucconi et al (1992) | A vs C | U | | 0.05[4] | | | |

A = obstructive sleep apnoea subject; C = non-snoring, non-apnoeic subject; S = snoring, non-apnoeic subject; A1 = non-obese obstructive sleep apnoea subject; A2 = Obese obstructive sleep apnoea subject; A3 High apnoea index / Low BMI; A4 Low apnoea index / High BMI.

1 Cd-Gn; 2Go-Gn; 3 Id-Gn; 4 Go-Me; 5 Go-B; 6 Ar-Go; 7 Ar-Pgn; 8 Go-Pgn; 9 Ram/MCF; 10 G-VL; 11 Pg

Angular measurements of mandibular landmarks can be used to describe the anteroposterior relationship of the mandible to other craniofacial structures. The simplest of these is an

angular measure between the cranial base (sella – nasion) and point B on the anterior mandible. This angle may be influenced by the angulation of the cranial base, but is nevertheless commonly used when analysing mandibular position from cephalometric radiographs.

Angle SNB has been found by a number of authors to be more acute in obstructive sleep apnoea patients than control populations, indicating a retrognathic mandibular position (Hochban and Brandenburg, 1994; Lowe et al, 1995; Lowe et al, 1996; Tangugsorn et al, 1995a; and Tsuchiya et al, 1992). Other authors have not found a significant difference in this angular measure of mandibular position between obstructive sleep apnoea and control populations (Battagel and L'Estrange, 1996; deBerry-Borowiecki et al, 1988; Johns et al, 1998; Lyberg et al, 1989a; Maltais et al, 1991; Mayer and Meier-Ewert, 1995; Mochizuki et al, 1996; Pracharktam et al, 1996; Strelzow et al, 1988; Zucconi et al, 1992; and Zucconi et al, 1993). Lowe et al (1996) found a more acute angle SNB in patients with a Class I dental malocclusion, and this was a significant finding whether the cephalometric radiograph was taken upright or supine. There was no significant difference between obstructive sleep apnoea subjects and controls with other patterns of dental malocclusion.

The angle between the cranial base (sella – nasion) and pogonion is similar to angle SNB but is more greatly influenced by the development or not of the bony chin. Tangugsorn et al (1995) is the only author to have found a significant decrease in this angle in obstructive sleep apnoea subjects. Lyberg et al (1989a) and Sakakibara et al (1999) who also used this angular measurement have found no significant difference between obstructive sleep apnoea subjects and controls.

The angulation of the mandibular plane relative to either the cranial base (sella – nasion) or Frankfort horizontal may indicate the relative prognathism of the mandible. Those subjects with a steep mandibular plane angle have a clockwise rotation of the mandible, and thus a more caudal positioned jaw. This might be expected in patients who have a tendency to obstruct their upper airway. A number of authors have indeed found this to be the case, with obstructive sleep apnoea subjects more likely to have a steep mandibular plane angle (Andersson and Brattstrom, 1991; Bacon et al, 1989; Johns et al, 1998; Lowe et al, 1995; Lowe et al, 1996; Strelzow et al, 1988; Tangugsorn et al, 1995a; and Tsuchiya et al, 1992). Other authors have not found a steeper mandibular plane angle in obstructive sleep apnoea subjects (deBerry-Borowiecki et al, 1988; Hochban and Brandenburg, 1994; Maltais et al, 1991; and Ono et al, 1996).

Lowe et al (1996) found obstructive sleep apnoea subjects with a class I dental malocclusion had an increased mandibular plane angle on cephalometric radiographs taken upright, but

there was no difference if the radiograph was taken supine. There was no significant difference in this measurement for obstructive sleep apnoea subjects with other patterns of dental malocclusion. Interestingly Strelzow et al (1988) found an increased mandibular plane angle in obstructive sleep apnoea subjects when compared to the cranial base, but not when compared to Frankfort horizontal. This finding cannot be explained alone on divergent cranial base and Frankfort horizontal as there was no significant difference between the two groups when the angle between these reference planes was compared. Tsuchiya et al (1992) reported an increased mandibular plane angle compared to the cranial base in obstructive sleep apnoea subjects with a high AHI and low BMI when compared to non-apnoeic controls and obstructive sleep apnoea subjects with a low AHI and high BMI. This latter obstructive sleep apnoea group did not have a significantly increased mandibular plane angle when compared with non-apnoeic controls.

The gonial angle is the angle between the vertical component of the mandible (ramus) and the horizontal component (body). An increased gonial angle indicates a clockwise rotation of the mandibular body relative to the ramus. An increase in this angle might be expected to have the same effect as an increase in the mandibular plane angle relative to a horizontal cranial reference line. Tangugsorn et al (1995a) found an increased gonial angle in obstructive sleep apnoea subjects, but no other author has found this to be true in their population (Battagel and L'Estrange, 1996; deBerry-Borowiecki et al, 1988; Hochban and Brandenburg, 1994; Lyberg et al, 1989a; Strelzow et al, 1988; Tsuchiya et al, 1992; and Zucconi et al, 1992).

Table 5.4-2 records the authors and the angular measurements of mandibular position. Definitions of the landmarks used may be found in the glossary. Figure 5.4-1 shows the parameters of the mandible measured from a lateral cephalometric radiograph.

**Table 5.4-2: Mandible – Angular Measurements**

| | | | Mandibular Plane Angle | S-N-B | S-N-Pg | Gonial Angle |
|---|---|---|---|---|---|---|
| Andersson et al (1991) | A vs C | U | 0.05[1] | | | |
| Bacon W et al (1989) | A vs C | U | 0.05[1] | | | |
| Battagel et al (1996) | A vs C | U | | NS | | NS[2] |
| deBerry-Borowiecki et al (1988) | A vs C | U | NS[1] | NS | | NS[3] |
| deBerry-Borowiecki et al (1988) | A vs C | U | NS[4] | | | |
| Hochban et al (1994) | A vs C | U | NS[1] | 0.02 | | NS[2] |
| Hochban et al (1994) | A vs C | U | NS[4] | | | |
| Johns et al (1998) | A vs S | U | 0.01[4] | NS | | |
| Lowe et al (1995) | A vs C | U | 0.02[1] | 0.01 | | |
| Lowe et al (1996) I | A vs C | U | 0.033[1] | 0.003 | | |
| Lowe et al (1996) I | A vs C | S | NS[1] | 0.006 | | |
| Lowe et al (1996) II | A vs C | U | NS[1] | NS | | |
| Lowe et al (1996) II | A vs C | S | NS[1] | NS | | |
| Lowe et al (1996) III | A vs C | U | NS[1] | NS | | |
| Lowe et al (1996) III | A vs C | S | NS[1] | NS | | |
| Lyberg et al (1995a) | A vs C | U | | NS | NS | NS[5] |
| Maltais et al (1991) | A vs C | U | NS[6] | NS | | |
| Mayer et al (1995) | A vs C | U | | NS | | |
| Mochizuki et al (1996) | A vs S | U | | NS | | |
| Ono et al (1996) | A vs C | U | NS[1] | | | |
| Pracharktam et al (1996) | A vs C | U | | NS | | |
| Sakakibara et al (1999) | A1 vs C | U | | | NS | |
| Sakakibara et al (1999) | A2 vs C | U | | | NS | |
| Sakakibara et al (1999) | A1 vs A2 | U | | | NS | |
| Strelzow et al (1988) | A vs C | U | 0.05[1] | NS | | NS[3] |
| Strelzow et al (1988) | A vs C | U | NS[4] | | | |
| Tangugsorn et al (1995a) | A vs C | U | 0.001[1] | 0.01 | 0.01 | 0.01[5] |
| Tsuchiya et al (1992) | A3 vs C | U | 0.01[1] | 0.01 | | NS[7] |
| Tsuchiya et al (1992) | A4 vs C | U | NS[1] | 0.05 | | NS[7] |
| Tsuchiya et al (1992) | A3 vs A4 | U | 0.05[1] | 0.05 | | NS[7] |
| Zucconi et al (1992) | A vs C | U | | NS | | |
| Zucconi et al (1993) | A vs S | U | | NS | | NS[3] |

A = obstructive sleep apnoea subject; C = non-snoring, non-apnoeic subject; S = snoring, non-apnoeic subject; A1 = non-obese obstructive sleep apnoea subject; A2 = Obese obstructive sleep apnoea subject; A3 High apnoea index / Low BMI; A4 Low apnoea index / High BMI.

1 SN/Go-Gn; 2 Ar-Go-Me; 3 Ar-Go-Gn; 4 FH/Go-Gn; 5 RL/ML; 6 SN/Go-B; 7 Not defined

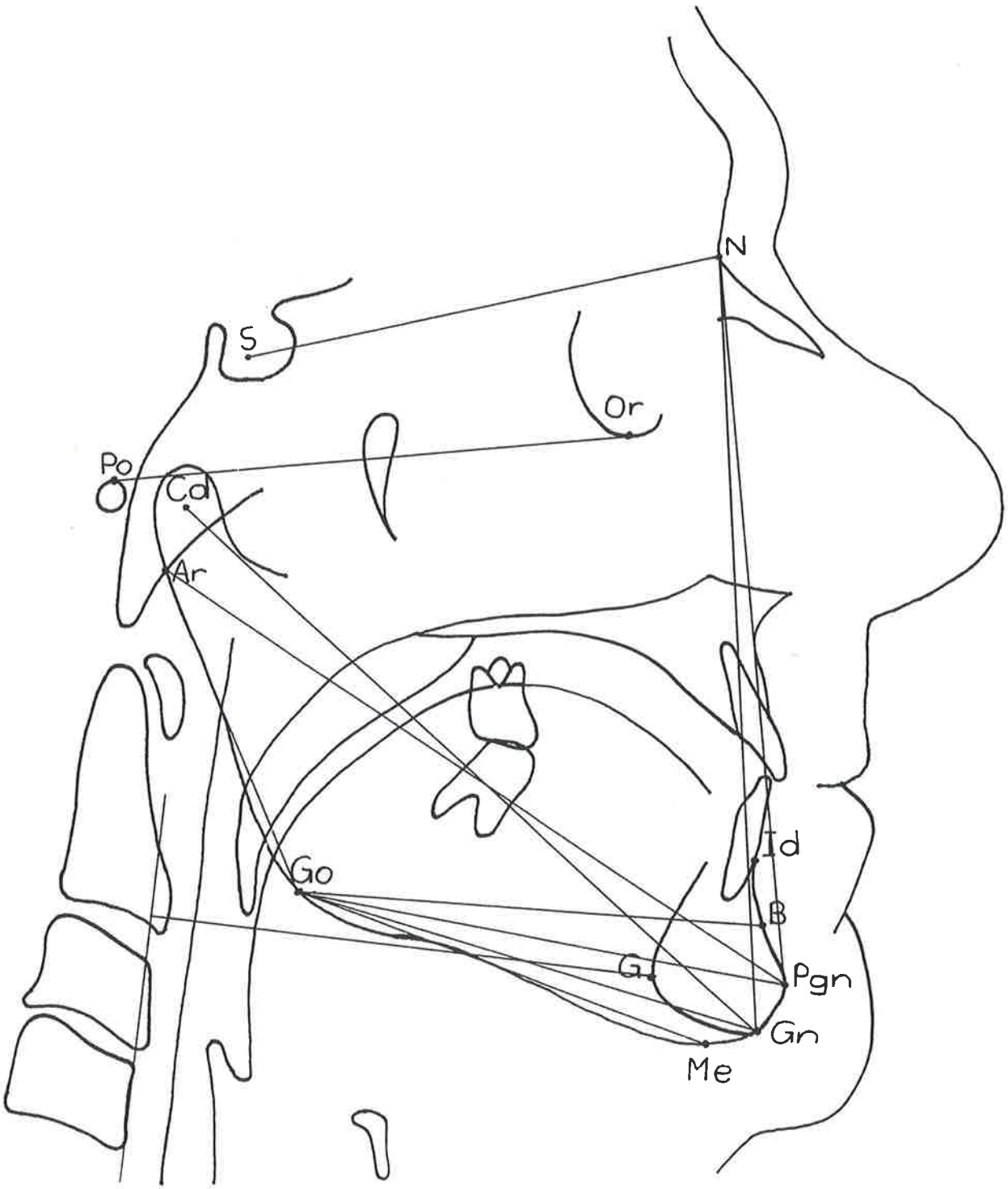


**Figure 5.4-1 Mandibular cephalometric measures**

## 5.5 Maxilla and Mandible Inter-relationship

There have been relatively few efforts to measure differences in length between the maxilla and mandible using a linear scale. The linear measures used have included the difference between the maxillary and mandibular length, the length from the pharyngeal wall to the lower incisors or tongue tip, or the distance from the posterior nasal spine to point B. Battagel and L'Estrange (1996) found a significantly decreased intermaxillary space length measured from the pharyngeal wall to the lingual aspect of the lower incisor tooth in obstructive sleep apnoea patients when compared to control subjects. Pracharktam et al (1994) and Pracharktam et al (1996) used an almost identical reference line and found no significant difference in length between obstructive sleep apnoea subjects and controls. Strelzow et al (1988) found a significant decrease in the distance PNS – B in obstructive sleep apnoea subjects, however deBerry-Borowiecki et al (1988) did not.

The area between the maxilla and mandible has been measured using a variety of landmarks. Only two authors have found a significantly decreased area (Battagel and L'Estrange, 1996; and Tangugsorn et al, 1995b).

Table 5.5-1 records the authors and the linear measurements of the relationship between the maxilla and the mandible. Definitions of the landmarks used may be found in the glossary.

**Table 5.5-1: Maxilla and Mandible Inter-relationship – Linear Measurements**

| | | | Length | Area |
|---|---|---|---|---|
| Battagel et al (1996) | A vs C | U | 0.001[1] | 0.035 |
| deBerry-Borowiecki et al (1988) | A vs C | U | NS[2] | |
| Lowe et al (1995) | A vs C | U | NS[3] | |
| Pracharktam et al (1994) | A vs S | U | NS[4] | NS |
| Pracharktam et al (1996) | A vs S | U | NS[4] | |
| Pracharktam et al (1996) | A vs S | U | NS[5] | |
| Sakakibara et al (1999) | A1 vs C | U | | NS |
| Sakakibara et al (1999) | A2 vs C | U | | NS |
| Sakakibara et al (1999) | A1 vs A2 | U | | NS |
| Strelzow et al (1988) | A vs C | U | 0.05[2] | NS |
| Tangugsorn et al (1995b) | A vs C | U | | 0.01 |

A = obstructive sleep apnoea subject; C = non-snoring, non-apnoeic subject; S = snoring, non-apnoeic subject; A1 = non-obese obstructive sleep apnoea subject; A2 = Obese obstructive sleep apnoea subject; A3 High apnoea index / Low BMI; A4 Low apnoea index / High BMI.

1 PhW-L1; 2 PNS-B; 3 Go-Gn subtract ANS-PNS; 4 PhW-TT; 5 Ar-A/Ar-B

Comparison of maxillary and mandibular position relative to each other is most easily done using the angle A-N-B. This measures the anteroposterior relationship of the maxilla and

mandible. A skeletal class I relationship results in a positive A-N-B angle whilst a skeletal class III relationship results in a negative A-N-B angle.

The majority of authors who have considered angle A-N-B have found no significant difference between obstructive sleep apnoea subjects and controls (Andersson and Brattstrom, 1991; Battagel and L'Estrange, 1996; Bacon et al, 1989; deBerry-Borowiecki et al, 1988; Hochban and Brandenburg, 1994; Lyberg et al, 1989a; Maltais et al, 1991; Ono et al, 1996; Sakakibara et al, 1999; Tangugsorn et al, 1995a; and Tsuchiya et al, 1992). Lowe et al (1995) found a significantly increased angle in obstructive sleep apnoea subjects, indicative of a retrognathic mandible relative to the maxilla in this population. Subjects with a dental class I malocclusion also had an increased A-N-B angle if they suffered obstructive sleep apnoea. This finding was significant for both upright and supine cephalometric radiographs (Lowe et al, 1996). This paper reported no significant difference in this angle for subjects with a dental class II or dental class III malocclusion.

The angle between the maxillary plane and mandibular plane has been measured to determine the divergence of the facial skeleton. Andersson and Brattstrom (1991) and Strelzow et al (1988) found obstructive sleep apnoea subjects had a more divergent facial pattern than non-apnoeic controls. Other authors have found no significant difference in this measure of facial type (deBerry-Borowiecki et al, 1988; and Hochban and Brandenburg, 1994).

The angulation of the occlusal plane to the cranial base was found to be less acute in obstructive sleep apnoea subjects than controls by Bacon et al (1989). Lowe et al (1996) found no significant difference in this angle for obstructive sleep apnoea subjects unless they had an upright cephalometric radiograph and a dental class I relationship in which case this angle was significantly increased. This indicates a divergent facial profile.

Table 5.5-2 records the authors and the angular measurements of the relationship between the maxilla and the mandible. Definitions of the landmarks used may be found in the glossary. Figure 5.5-1 shows the cephalometric measures of maxillary-mandibular inter-relationship.

**Table 5.5-2: Maxilla and Mandible Inter-relationship – Angular Measurements**

| | | | ANS-PNS/Go-Gn | SN/OP | A-N-B |
|---|---|---|---|---|---|
| Andersson et al (1991) | A vs C | U | 0.05 | | NS |
| Bacon et al (1989) | A vs C | U | | 0.05 | NS |
| Battagel et al (1996) | A vs C | U | | | NS |
| deBerry-Borowiecki et al (1988) | A vs C | U | NS | | NS |
| Hochban et al (1994) | A vs C | U | NS | | NS |
| Lowe et al (1995) | A vs C | U | | | 0.02 |
| Lowe et al (1996) I | A vs C | U | | 0.044 | 0.027 |
| Lowe et al (1996) I | A vs C | S | | NS | 0.011 |
| Lowe et al (1996) II | A vs C | U | | NS | NS |
| Lowe et al (1996) II | A vs C | S | | NS | NS |
| Lowe et al (1996) III | A vs C | U | | NS | NS |
| Lowe et al (1996) III | A vs C | S | | NS | NS |
| Lyberg et al (1995a) | A vs C | U | | NS | NS |
| Maltais et al (1991) | A vs C | U | | | NS |
| Ono et al (1996) | A vs C | U | | | NS |
| Sakakibara et al (1999) | A1 vs C | U | | | NS |
| Sakakibara et al (1999) | A2 vs C | U | | | NS |
| Sakakibara et al (1999) | A1 vs A2 | U | | | NS |
| Strelzow et al (1988) | A vs C | U | 0.05 | | |
| Tangugsorn et al (1995b) | A vs C | U | | | NS |
| Tsuchiya et al (1992) | A3 vs C | U | | | NS |
| Tsuchiya et al (1992) | A4 vs C | U | | | NS |
| Tsuchiya et al (1992) | A3 vs A4 | U | | | NS |

A = obstructive sleep apnoea subject; C = non-snoring, non-apnoeic subject; S = snoring, non-apnoeic subject; A1 = non-obese obstructive sleep apnoea subject; A2 = Obese obstructive sleep apnoea subject; A3 High apnoea index / Low BMI; A4 Low apnoea index / High BMI.

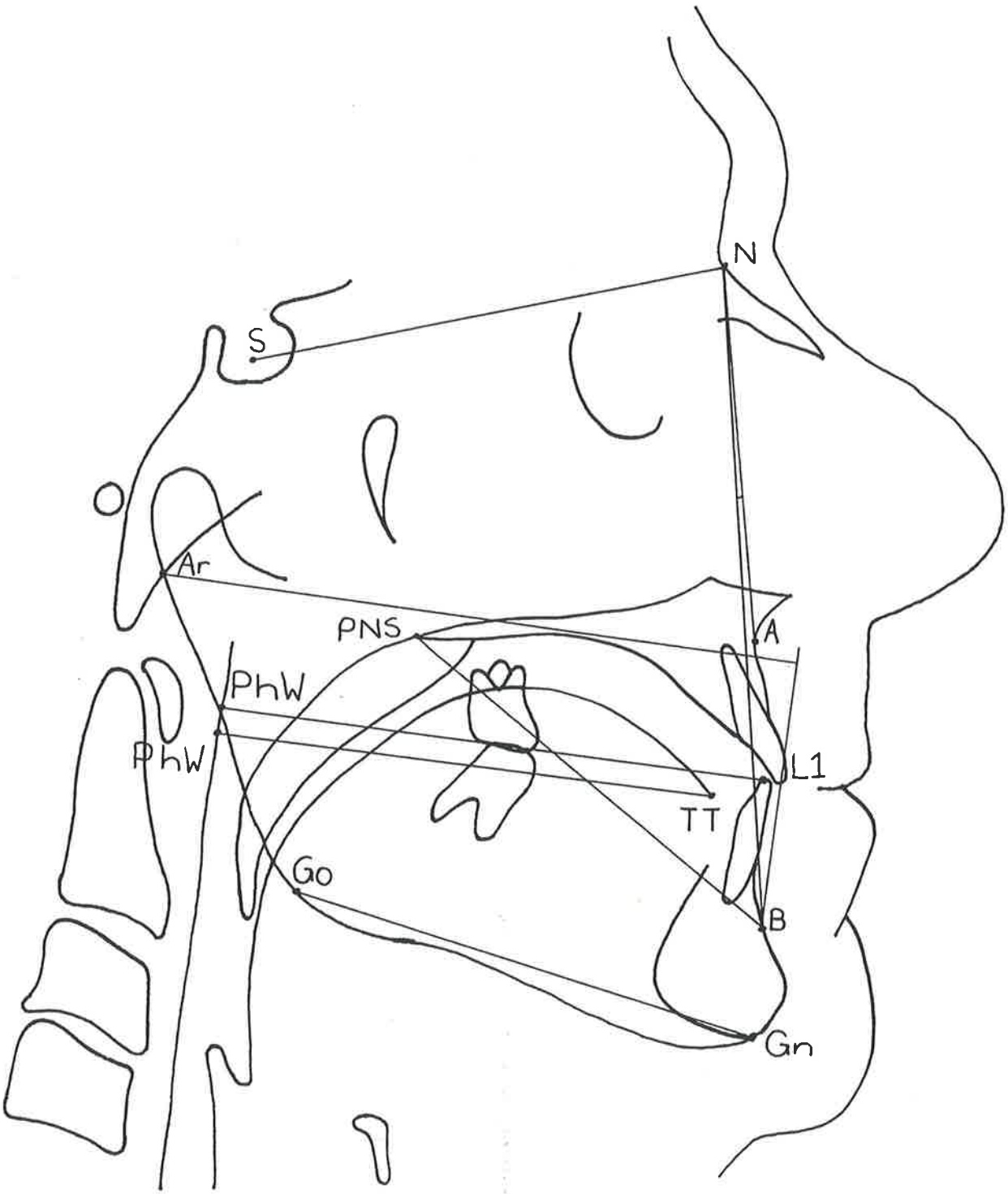


**Figure 5.5-1 Maxilla and manidible inter-relationship cephalometric measures**

## 5.6 Facial Height

Facial height may be considered anteriorly and posteriorly using a number of landmarks and reference lines. The anterior facial height may be considered as total face height from the anterior cranium to the lower border of the mandible. This length may be divided into upper facial height from the anterior cranium to the maxillary plane (essentially this measures the dimensions of the nasal cavity). The lower facial height is measured from the maxillary plane to the lower border of the anterior mandible. Many authors have considered the anterior facial height of obstructive sleep apnoea subjects and compared them with control subjects. There is no standard measure of anterior facial height and a large number of landmarks have been used.

Total anterior face height in obstructive sleep apnoea subjects was increased in all studies (Andersson and Brattstrom, 1991; deBerry-Borowiecki et al, 1988; Lowe et al, 1995; Lyberg et al, 1989a; and Strelzow et al, 1988) except (Sakakibara et al, 1999) who found no significant difference compared with a control population.

Upper anterior facial height has not been consistently increased or decreased in obstructive sleep apnoea populations. A smaller upper facial height in obstructive sleep apnoea subjects was found in one population (Lowe et al, 1995). In contrast deBerry-Borowiecki et al (1988); Johns et al (1998); and Strelzow et al (1988) measured an increase in upper anterior facial height in their obstructive sleep apnoea subjects. Most other authors have not found any significant difference in upper anterior facial height between obstructive sleep apnoea subjects and controls (Andersson and Brattstrom, 1991; Bacon et al, 1989; Lowe et al, 1995; Lyberg et al, 1989a; Maltais et al, 1991; Pracharktam et al, 1994; Pracharktam et al, 1996; Sakakibara et al, 1999; and Tangugsorn et al, 1995a).

Lower anterior facial height has also produced conflicting results. Some studies (Bacon et al, 1989; deBerry-Borowiecki et al, 1988; Lowe et al, 1995; Strelzow et al, 1988; and Tangugsorn et al, 1995a) have found an increased lower anterior facial height in obstructive sleep apnoea subjects. Whilst other populations have not differed significantly between obstructive sleep apnoea subjects and controls (Andersson and Brattstrom, 1991; Johns et al, 1998; Lyberg et al, 1989a; and Sakakibara et al, 1999). Strelzow et al (1988) did not find any increase in distance A – B or A – Gn, however distance ANS – B was increased in obstructive sleep apnoea subjects. This suggests the increased anterior facial height in this group of obstructive sleep apnoea subjects was in the maxillary basal bone.

The height of the palatal vault was measured by Johns et al (1998) and found not to differ significantly between obstructive sleep apnoea subjects and controls. The ratio of the

distance of the upper facial height and lower facial height was measured by Tangugsorn et al (1995a). They found this ratio to be significantly smaller in obstructive sleep apnoea subjects, principally because of an increase in their anterior lower facial height.

Table 5.6-1 records the authors and the linear measurements of facial height. Definitions of the landmarks used may be found in the glossary.

**Table 5.6-1: Facial Height – Linear Measurements**

| | | | UFH | LFH | TFH | Palate | UFH/LFH |
|---|---|---|---|---|---|---|---|
| Andersson et al (1991) | A vs S | U | NS[7] | NS[11] | 0.05[1] | | |
| Bacon et al (1989) | A vs C | U | NS | 0.01 | | | |
| deBerry-Borowiecki et al (1988) | A vs C | U | 0.000[2] | 0.000[3] | 0.008[4] | | |
| deBerry-Borowiecki et al (1988) | A vs C | U | | 0.001[5] | | | |
| deBerry-Borowiecki et al (1988) | A vs C | U | | 0.021[6] | | | |
| Johns et al (1998) | A vs S | U | 0.04[7] | NS[8] | | NS[9] | |
| Lowe et al (1995) | A vs C | U | 0.01 | 0.03 | 0.001 | | |
| Lyberg et al (1989a) | A vs C | U | NS[10] | NS[11] | 0.051 | | |
| Maltais et al (1991) | A vs C | U | NS[7] | NS[8] | | | |
| Pracharktam et al (1994) | A vs C | U | NS[12] | | | | |
| Pracharktam et al (1996) | A vs C | U | NS[12] | | | | |
| Sakakibara et al (1999) | A1 vs C | U | NS[7] | NS[8] | NS[13] | | |
| Sakakibara et al (1999) | A2 vs C | U | NS[7] | NS[8] | NS[13] | | |
| Sakakibara et al (1999) | A1 vs A2 | U | NS[7] | NS[8] | NS[13] | | |
| Strelzow et al (1988) | A vs C | U | 0.05[2] | NS[3] | 0.05[4] | | |
| Strelzow et al (1988) | A vs C | U | | 0.05[5] | | | |
| Strelzow et al (1988) | A vs C | U | | NS[6] | | | |
| Tangugsorn et al (1995a) | A vs C | U | NS[7] | 0.001[11] | | | 0.001 |
| Zucconi et al (1993) | A vs C | U | | NS[8] | | | |
| Zucconi et al (1993) | A vs S | U | | NS[8] | | | |

A = obstructive sleep apnoea subject; C = non-snoring, non-apnoeic subject; S = snoring, non-apnoeic subject; A1 = non-obese obstructive sleep apnoea subject; A2 = Obese obstructive sleep apnoea subject; A3 High apnoea index / Low BMI; A4 Low apnoea index / High BMI.

1 N-Gn; 2 A-N; 3 A-B; 4 B-N; 5 ANS-B; 6 A-Gn; 7 ANS-N; 8 ANS-Me; 9 Ocl-Pal 6; 10 N (ANS-PNS; 11 Gn (ANS-PNS; 12 Me (ANS-PNS; 13 N-Me.

Posterior facial height is measured from the cranial base to the lower border of the mandible. This may be divided into upper posterior facial height, which extends from the cranial base to the maxilla, and lower posterior facial height, which extends from the maxilla to the lower border of the mandible. As with most cephalometric distances the choice of landmark and reference lines varies between studies, as indicated in Table 5.6-2.

Total posterior facial height was significantly decreased in the obstructive sleep apnoea population studied by Andersson and Brattstrom (1991); Tangugsorn et al (1995a). In

contrast, Hochban and Brandenburg (1994); and Strelzow et al (1988) measured a significantly increased total posterior facial height in obstructive sleep apnoea subjects. Other authors (deBerry-Borowiecki et al, 1988; and Sakakibara et al, 1999) have measured no significant difference in this dimension between obstructive sleep apnoea subjects and a control population.

Posterior upper facial height has been measured in three cephalometric studies with conflicting results.

Strelzow et al (1988) measured a significantly increased distance from the sphenoidal rostrum to the posterior nasal spine in obstructive sleep apnoea subjects; measured a significantly decreased distance from sella to the maxillary plane whilst Lyberg et al (1989a) found no significant difference in upper posterior facial height between obstructive sleep apnoea subjects and controls.

Lower posterior facial height has been measured by a larger number of authors, but with no greater consensus of result. Obstructive sleep apnoea subjects had a significantly increased lower posterior facial height in studies by Hochban and Brandenburg (1994); Pracharktam et al (1996); and Strelzow et al (1988). A decreased lower posterior facial height in obstructive sleep apnoea subjects has been reported by Tangugsorn et al (1995a). Other authors (deBerry-Borowiecki et al, 1988; Lowe et al, 1995; Lyberg et al, 1989a; Pracharktam et al, 1994; Zucconi et al, 1992; and Zucconi et al, 1993) have found no significant difference in lower posterior facial height between obstructive sleep apnoea subjects and controls.

The ratio between posterior lower and middle facial height was decreased in obstructive sleep apnoea subjects, indicating a decreased lower facial height in obstructive sleep apnoea subjects. Lyberg et al (1989a) and Tangugsorn et al (1995a) found no significant difference in ratio between upper and lower posterior facial height. This latter group of authors did find a significant increased ratio in obstructive sleep apnoea subjects which was attributed to the increased lower anterior facial height of their obstructive sleep apnoea population.

Table 5.6-2 records the authors and the angular measurements of facial height. Definitions of the landmarks used may be found in the glossary. Figure 5.6-1 shows the cephalometric measures of facial height.

**Table 5.6-2: Facial Height – Angular Measurements**

| | | | UFH | LFH | TFH | UFH/LFH | AFH/PFH |
|---|---|---|---|---|---|---|---|
| Andersson et al (1991) | A vs S | U | | | 0.05[1] | | |
| deBerry-Borowiecki et al (1988) | A vs C | U | | NS[2] | NS[1] | | |
| Hochban et al (1994) | A vs C | U | | 0.005[2] | 0.05[1] | | |
| Lowe et al (1995) | A vs C | U | | NS | | | |
| Lyberg et al (1989a) | A vs C | U | NS[3] | NS[4] | | NS | |
| Pracharktam et al (1994) | A vs C | U | | NS[5] | | 0.01[6] | |
| Pracharktam et al (1996) | A vs C | U | | 0.045[5] | | | |
| Sakakibara et al (1999) | A1 vs C | U | | | NS[1] | | |
| Sakakibara et al (1999) | A2 vs C | U | | | NS[1] | | |
| Sakakibara et al (1999) | A1 vs A2 | U | | | NS[1] | | |
| Strelzow et al (1988) | A vs C | U | 0.05[7] | 0.05[2] | 0.05[1] | | |
| Tangugsorn et al (1995a) | A vs C | U | 0.05[3] | 0.05[4] | 0.01 | NS | 0.001 |
| Zucconi et al (1993) | A vs C | U | | NS[2] | | | |
| Zucconi et al (1993) | A vs S | U | | NS[2] | | | |

A = obstructive sleep apnoea subject; C = non-snoring, non-apnoeic subject; S = snoring, non-apnoeic subject; A1 = non-obese obstructive sleep apnoea subject; A2 = Obese obstructive sleep apnoea subject.

1S-Go; 2 PNS-Go; 3 S (ANS-PNS; 4 Go (ANS-PNS; 5 Post. In. Mx. Ht.; 6 LFH/MFH; 7 SR-PNS.

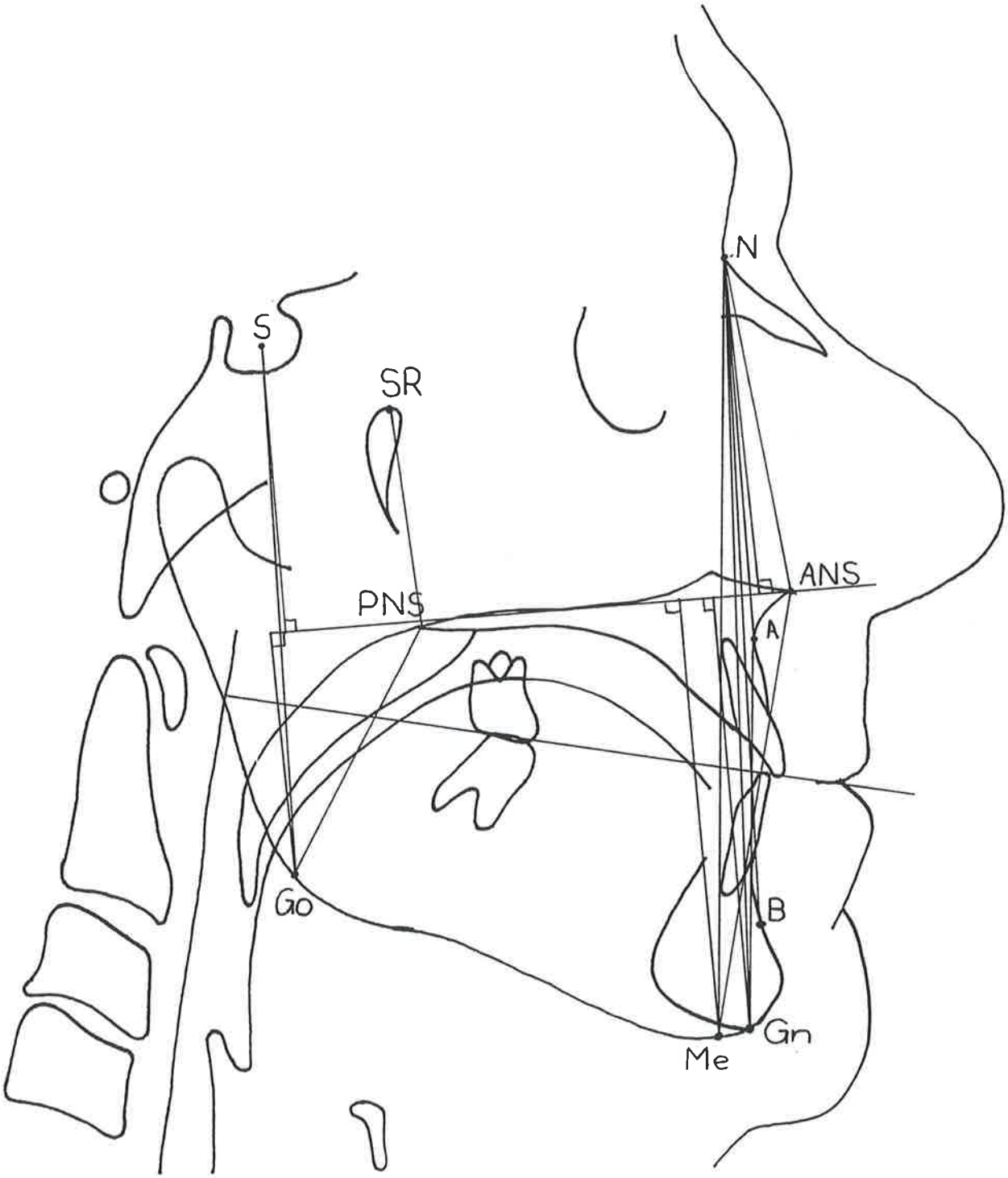


**Figure 5.6-1 Facial height cephalometric measures**

## 5.7 Bony Pharynx

The dimensions of the bony pharynx have also been considered at various levels. The soft tissue drape is influenced by the position of the underlying skeletal tissue, and consideration of skeletal pharyngeal position is therefore justified.

The most commonly assessed distances are those between the most posterior point on the bony hard palate and the anterior inferior margin of foramen magnum (Ba) or the most anterior superior point on the first cervical vertebra (AA). A fairly consistent finding in most studies of the bony pharynx is a decrease in distance PNS – Ba in obstructive sleep apnoea subjects (Bacon et al, 1989; Hochban and Brandenburg, 1994; Lyberg et al, 1989a; Sakakibara et al, 1999; Tangugsorn et al, 1995a; and Zucconi et al, 1993). Sakakibara et al (1999) did not find a significant decrease in pharyngeal width at this level for obese obstructive sleep apnoea subjects, however non-obese obstructive sleep apnoea subjects had a significantly narrowed airway at this level both compared to nonapnoeic controls and obese obstructive sleep apnoea subjects. Zucconi et al (1993) found no significant difference between obstructive sleep apnoea subjects and snoring controls, however nonsnoring controls had a significantly larger distance at this level.

Mochizuki et al (1996); Pracharktam et al (1994); and Pracharktam et al (1996) did not find a significant difference in this dimension between obstructive sleep apnoea subjects and control subjects.

Andersson and Brattstrom (1991); Sakakibara et al (1999); Strelzow et al (1988); and Tangugsorn et al (1995a) found the linear distance PNS – AA significantly decreased in obstructive sleep apnoea patients compared with controls. This is in contrast to other authors who have measured no significant difference in this distance between obstructive sleep apnoea and control subjects (deBerry-Borowiecki et al, 1988; Hochban and Brandenburg, 1994; Johns et al, 1998; and Mayer and Meier-Ewert, 1995).

Other isolated measurements of the bony pharynx have been made, principally at the nasopharyngeal level of the pharynx. Table 5.7-1 records the authors and the measures of the bony pharynx. Definitions of the landmarks used may be found in the glossary. Figure 5.7-1 shows diagrammatically the cephalometric measures of the bony pharynx.

**Table 5.7-1: Bony Pharyngeal Measurements**

| | | | PNS-Ba | PNS-AA | PNS-SR | PNS-Ar | Ba-PhW | Ba-PM | Go-PhW |
|---|---|---|---|---|---|---|---|---|---|
| Andersson et al (1991) | A vs S | U | | 0.05 | | | | | |
| Bacon et al (1989) | A vs S | U | 0.01 | | | | | | |
| deBerry-Borowiecki et al (1988) | A vs S | U | | NS | 0.05 | NS | | | NS |
| Hochban et al (1994) | A vs C | U | 0.001 | NS | | | NS | | |
| Johns et al (1998) | A vs S | U | | NS | | | | | |
| Lyberg et al (1989a) | A vs C | U | 0.01 | | | | | | |
| Mayer et al (1995) | A vs C | U | | NS | | | | | |
| Mochizuki et al (1996) | A vs S | U | NS | | | | | | |
| Pracharktam et al (1994) | A vs S | U | NS | | | | | | |
| Pracharktam et al (1996) | A vs S | U | NS | | | | | | |
| Sakakibara et al (1999) | A1 vs C | U | 0.001 | 0.001 | | | | | |
| Sakakibara et al (1999) | A2 vs C | U | NS | 0.001 | | | | | |
| Sakakibara et al (1999) | A1 vs A2 | U | 0.001 | 0.02 | | | | | |
| Strelzow et al (1988) | A vs C | U | | 0.05 | 0.05 | | | | |
| Tangugsorn et al (1995a) | A vs C | U | 0.001 | 0.01 | | | | | |
| Zucconi et al (1993) | A vs C | U | 0.01 | | | | | | |
| Zucconi et al (1993) | A vs S | U | NS | | | | | | |

A = obstructive sleep apnoea subject; C = non-snoring, non-apnoeic subject; S = snoring, non-apnoeic subject; A1 = non-obese obstructive sleep apnoea subject; A2 = Obese obstructive sleep apnoea subject.

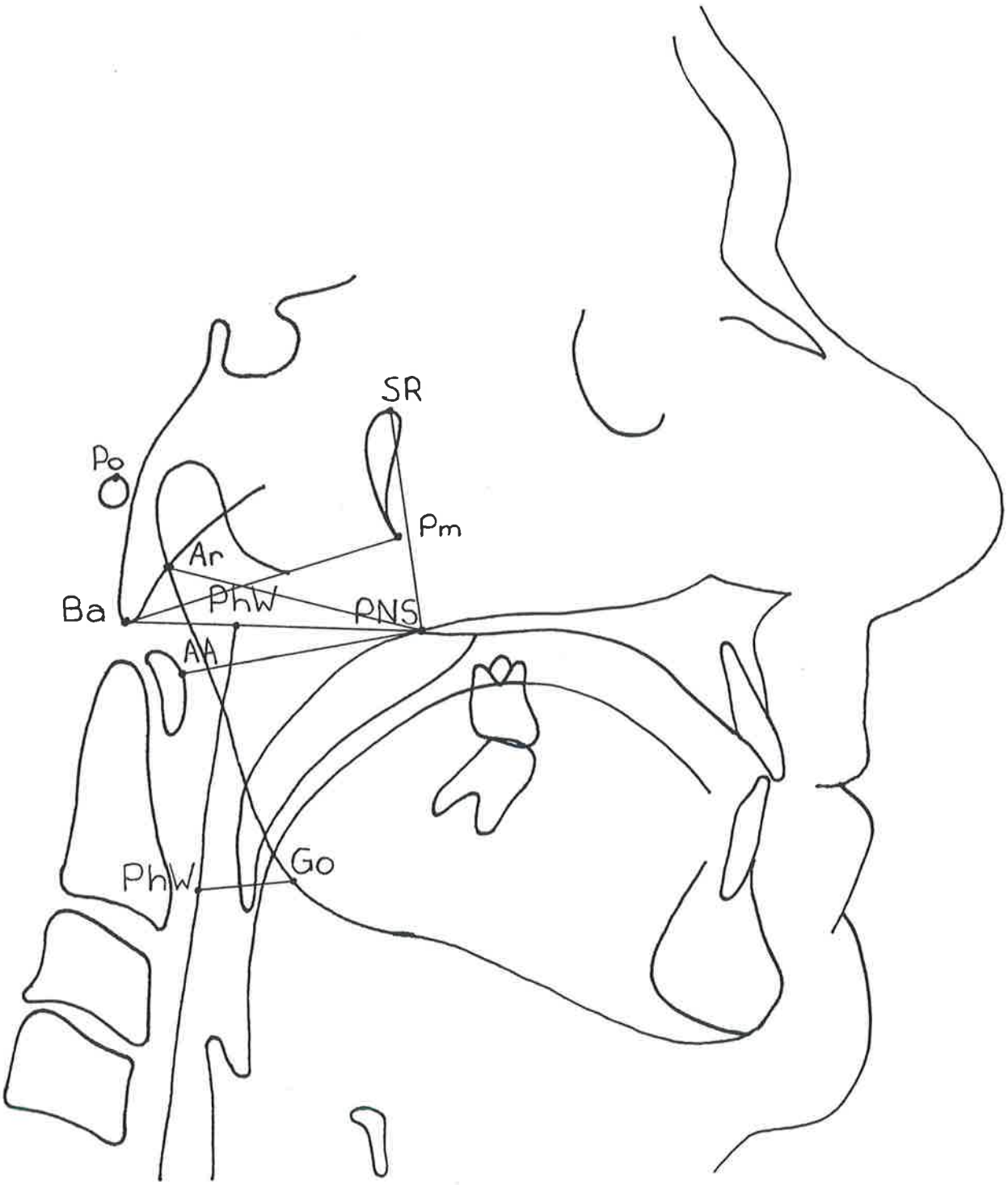


**Figure 5.7-1: Bony pharynx cephalometric measures.**

## 5.8 Dental Measurements

There are no consistent findings on lateral cephalometric radiographs from dental measurements alone that are significant for obstructive sleep apnoea patients compared with controls. The dental anomalies reported by some authors are probably related to dental compensation for skeletal abnormalities in the obstructive sleep apnoea population.

When considering the overbite or overjet relationship of the central incisors only Lowe et al (1996) found a significant difference between obstructive sleep apnoea subjects and controls. They found obstructive sleep apnoea subjects with a dental Class II malocclusion had a significantly increased overjet when compared with non-apnoeic controls with the same dental malocclusion. Battagel and L'Estrange (1996); Lowe et al (1995); Lowe et al (1996); Lyberg et al (1995a); Ono et al (1996); Tangugsorn et al (1995) and Tsuchiya et al (1992) found no significant difference in central incisor overbite or overjet relationships.

Lowe et al (1995) measured the length of the upper and lower central incisors and molars. They found a significantly increased length of the upper central incisor in obstructive sleep apnoea subjects compared with controls. There were no other significant differences for the parameters measured.

Although not measured from lateral cephalometric radiographs there is a suggestion from one group of authors that a constricted maxilla may contribute to the development of obstructive sleep apnoea. This group used models of 40 patients with obstructive sleep apnoea and 21 nonsnoring nonapnoeic controls. They found 20/40 (50%) of obstructive sleep apnoea patients had a posterior crossbite, compared with only 1/21(5%) of the control subjects ($p<0.001$). There was significantly smaller intercanine, interpremolar, intermolar and maxillary lengths in obstructive sleep apnoea patients compared with control subjects ($p<0.05$).

Table 5.8-1 records the authors and the linear measurements of the dentition. Definitions of the landmarks used may be found in the glossary.

**Table 5.8-1: Dentition – Linear Measurements**

| | | | OB | OJ | ADH | MxM | MdMH |
|---|---|---|---|---|---|---|---|
| Battagel et al (1996) | A vs C | U | NS | NS | | | |
| Lowe et al (1995) | A vs C | U | | NS | 0.01 | NS | NS |
| Lowe et al (1996) I | A vs C | U | | NS | | | |
| Lowe et al (1996) I | A vs C | S | | NS | | | |
| Lowe et al (1996) II | A vs C | U | | 0.042 | | | |
| Lowe et al (1996) II | A vs C | S | | NS | | | |
| Lowe et al (1996) III | A vs C | U | | NS | | | |
| Lowe et al (1996) III | A vs C | S | | NS | | | |
| Lyberg et al (1995a) | A vs C | U | NS | NS | | | |
| Ono et al (1996) | A vs C | U | NS | NS | | | |
| Tangugsorn et al (1995b) | A vs C | U | NS | NS | | | |
| Tsuchiya et al (1992) | A3 vs C | U | NS | NS | | | |
| Tsuchiya et al (1992) | A4 vs C | U | NS | NS | | | |
| Tsuchiya et al (1992) | A3 vs A4 | U | NS | NS | | | |

A = obstructive sleep apnoea subject; C = non-snoring, non-apnoeic subject; S = snoring, non-apnoeic subject; A1 = non-obese obstructive sleep apnoea subject; A2 = Obese obstructive sleep apnoea subject; A3 High apnoea index / Low BMI; A4 Low apnoea index / High BMI.

The angular dental measurements that may be taken from lateral cephalometric radiographs determine the inclination of the central incisors relative to horizontal or vertical planes of reference. The angulation between the upper and lower incisors may also be measured.

Lowe et al (1996) found the upper incisors of obstructive sleep apnoea subjects were more upright than those of non-apnoeic controls. This was only true for obstructive sleep apnoea subjects with a class I dental malocclusion. Battagel and L'Estrange (1996); Ono et al (1996) and Tsuchiya et al (1992) found no difference in upper incisor angulation. The lower incisors were significantly proclined in obstructive sleep apnoea subjects with a dental class I malocclusion (Lowe et al, 1996) or those obstructive sleep apnoea subjects with a high apnoea index and low BMI (Tsuchiya et al, 1992). Battagel and L'Estrange (1996) found no significant difference in incisor angulation.

Bacon et al (1989) found no significant difference in the angulation between the upper and lower incisors of obstructive sleep apnoea or control subjects.

The significance of dental measurements in obstructive sleep apnoea is to show dental compensation for underlying skeletal abnormalities.

Table 5.8-2 records the authors and the angular measurements of the dentition. Definitions of the landmarks used may be found in the glossary. Figure 5.8-1 shows diagrammatically the dental measurements from a lateral cephalometric radiograph.

**Table 5.8-2: Dentition – Angular Measurements**

| | | | U1/L1 | U1/ANS-PNS | U1/S-N | U1/N-A | L1 to MP | L1/Go-Gn | L1/N-B |
|---|---|---|---|---|---|---|---|---|---|
| Bacon et al (1989) | A vs C | U | NS | | | | | | |
| Battagel et al (1996) | A vs C | U | | NS | | | | NS | |
| Lowe et al (1995) | A vs C | U | | | NS | | NS | NS | |
| Lowe et al (1996) I | A vs C | U | | | 0.037 | | | 0.002 | |
| Lowe et al (1996) I | A vs C | S | | | 0.035 | | | NS | |
| Lowe et al (1996) II | A vs C | U | | | NS | | | NS | |
| Lowe et al (1996) II | A vs C | S | | | NS | | | NS | |
| Lowe et al (1996) III | A vs C | U | | | NS | | | NS | |
| Lowe et al (1996) III | A vs C | S | | | NS | | | NS | |
| Ono et al (1996) | A vs C | U | | | NS | | | | |
| Tsuchiya et al (1992) | A3 vs C | U | | | | NS | | | 0.05 |
| Tsuchiya et al (1992) | A4 vs C | U | | | | NS | | | NS |
| Tsuchiya et al (1992) | A3 vs A4 | U | | | | NS | | | NS |

A = obstructive sleep apnoea subject; C = non-snoring, non-apnoeic subject; S = snoring, non-apnoeic subject; A1 = non-obese obstructive sleep apnoea subject; A2 = Obese obstructive sleep apnoea subject; A3 High apnoea index / Low BMI; A4 Low apnoea index / High BMI.

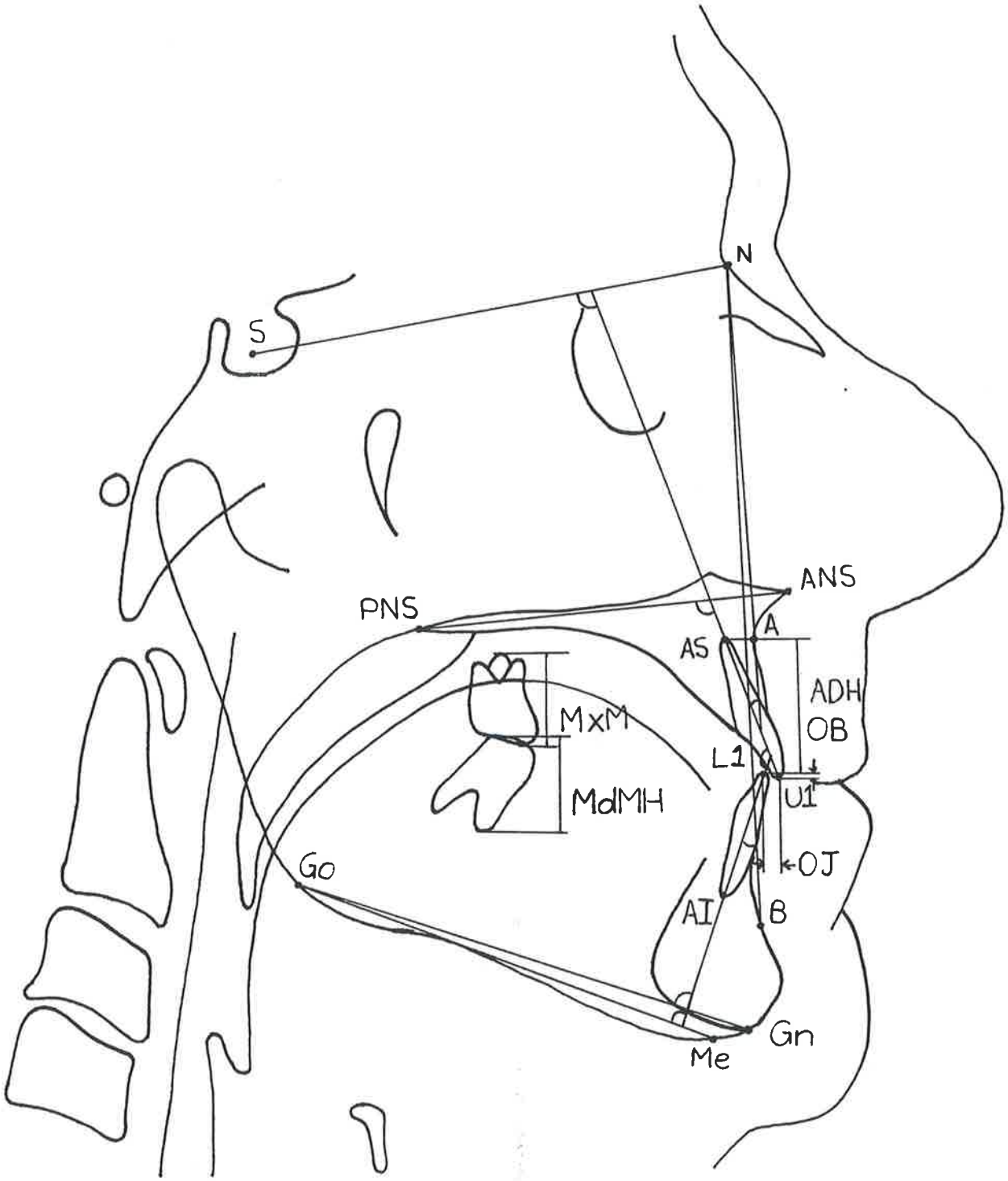


**Figure 5.8-1 Dentition cephalometric measures**

## 5.9 Cervical Spine

The measurement of natural head posture has been reported using cephalometric radiographs in populations with obstructive sleep apnoea. The literature suggests head posture is maintained to ensure a patent airway and is influenced by sight, hearing, vestibular orientation, and mass and contour of the head.

Only one linear measure involving the cervical spine posture has been reported. The distance from C2 to a perpendicular dropped from sella was found significantly smaller in obstructive sleep apnoea subjects (Battagel and L'Estrange, 1996).

Table 5.9-1 records the authors and the linear measurements of the cervical spine. Definitions of the landmarks used may be found in the glossary.

**Table 5.9-1: Cervical Spine – Linear Measurements**

| | | | C2-S |
|---|---|---|---|
| Battagel and L'Estrange (1996) | A vs C | U | 0.003 |

A = obstructive sleep apnoea subject; C = non-snoring, non-apnoeic subject; S = snoring, non-apnoeic subject; A1 = non-obese obstructive sleep apnoea subject; A2 = Obese obstructive sleep apnoea subject; A3 High apnoea index / Low BMI; A4 Low apnoea index / High BMI.

Angulation of the cranium and facial skeleton to the cervical spine is used as the measure of head posture. The reference lines may be a tangent to the second cervical vertebra (OPT); a tangent to the second and fourth cervical vertebrae (CVT); or arbitrary horizontal and vertical reference lines.

The differences reported by all authors who found a significant difference between obstructive sleep apnoea subjects and controls was a more upright head posture. Sakakibara et al (1999) reported significant differences for most measures of head posture between obese and non-obese obstructive sleep apnoea subjects, but no significant differences between either obstructive sleep apnoea group and matched controls.

The horizontal plane did not prove effective in differentiating between obstructive sleep apnoea subjects and controls in any of the studies included, despite other parameters indicating a more upright head posture in obstructive sleep apnoea subjects.

Table 5.9-2 records the authors and the angular measurements of the dentition. Definitions of the landmarks used may be found in the glossary. Figure 5.9-1 shows diagrammatically the cephalometric measurements of the cervical spine.

**Table 5.9-2: Cervical Spine – Angular Measurements**

| | | | /Ver | /OPT | /CVT | /Hor |
|---|---|---|---|---|---|---|
| Battagel et al (1996) | A vs C | U | | | 0.038[1] | |
| Ozbek et al (1998) | A vs C | U | NS1 | 0.001[1] | 0.001[1] | 0.05[2] |
| Ozbek et al (1998) | A vs C | U | | | | 0.05[3] |
| Pracharktam et al (1996) | A vs S | U | | 0.05[1] | 0.002[1] | |
| Sakakibara et al (1999) | A1 vs C | U | NS[1] | NS[1] | NS[2] | NS[2] |
| Sakakibara et al (1999) | A1 vs C | U | NS[4] | NS[4] | | NS[3] |
| Sakakibara et al (1999) | A1 vs C | U | | NS[5] | | |
| Sakakibara et al (1999) | A2 vs C | U | NS[1] | NS[1] | NS[2] | NS[2] |
| Sakakibara et al (1999) | A2 vs C | U | NS[4] | NS[4] | | NS[3] |
| Sakakibara et al (1999) | A2 vs C | U | | NS[5] | | |
| Sakakibara et al (1999) | A1vs A2 | U | NS[1] | 0.02[1] | NS[2] | 0.02[2] |
| Sakakibara et al (1999) | A1vs A2 | U | NS[4] | 0.02[4] | | 0.02[3] |
| Sakakibara et al (1999) | A1vs A2 | U | | 0.02[5] | | |
| Tangugsorn et al (1995a) | A vs C | U | NS[5] | 0.001[5] | 0.001[4] | |
| Tangugsorn et al (1995a) | A vs C | U | NS[4] | 0.001[4] | 0.001[5] | |

A = obstructive sleep apnoea subject; C = non-snoring, non-apnoeic subject; S = snoring, non-apnoeic subject; A1 = non-obese obstructive sleep apnoea subject; A2 = Obese obstructive sleep apnoea subject; A3 High apnoea index / Low BMI; A4 Low apnoea index / High BMI. 1 S-N; 2 OPT; 3 CVT; 4 ANS-PNS; 5 FH

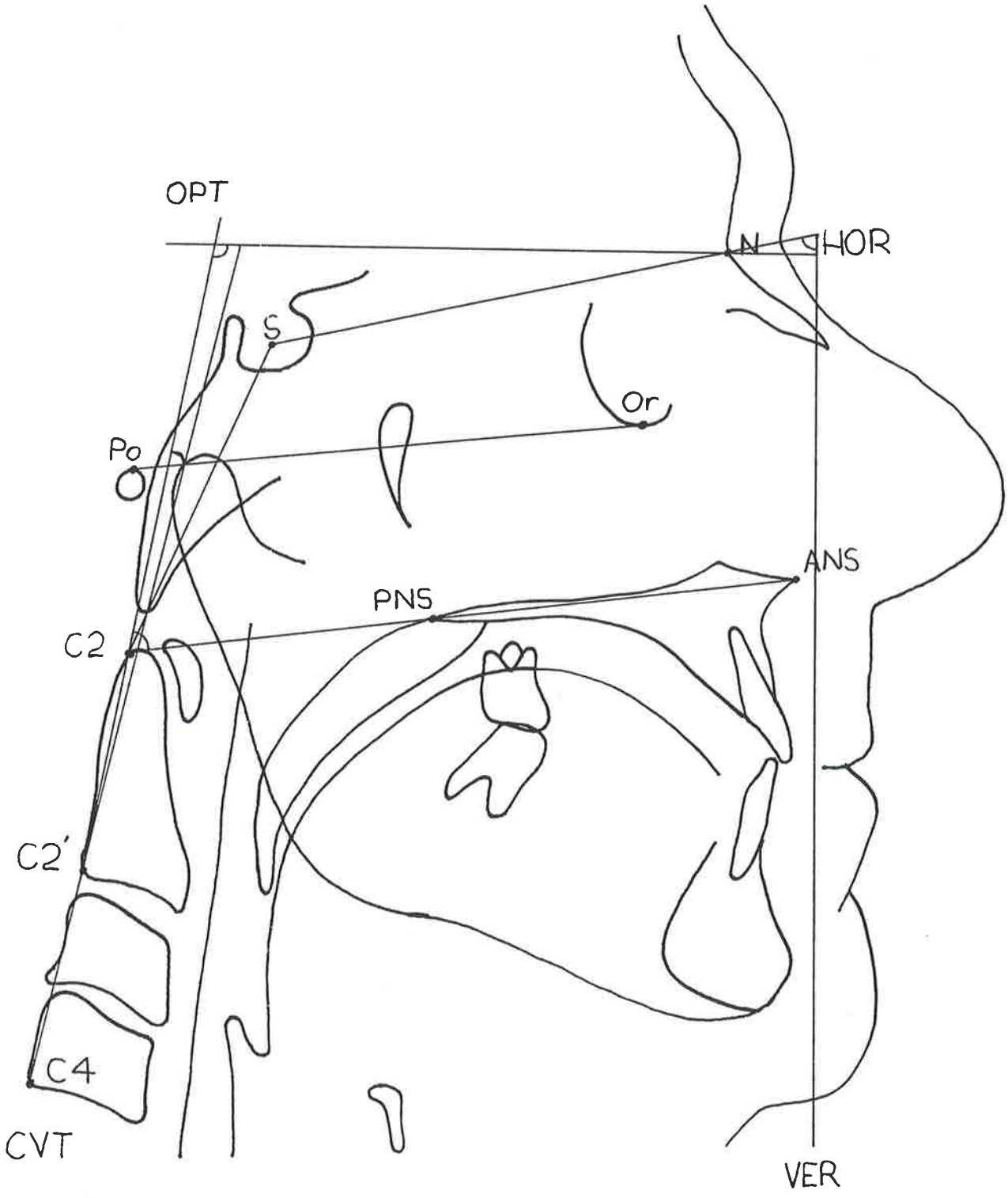


**Figure 5.9-1 Cervical spine cephalometric measures.**

# Chapter 6

# Lateral Cephalometric Examination of the Upper Airway – Soft Tissue

## 6.1 Introduction

The soft tissues of the upper airway can be visualized on a lateral cephalometric radiograph. They present as softer opacities than skeletal landmarks and may therefore be more difficult to identify accurately.

The literature concerning the assessment of the soft tissue and its relationship to the upper airway on subjects with obstructive sleep apnoea has been reviewed. The results of this review will be presented by anatomic area. The anatomic areas to be considered in this chapter are shown at diagrammatically at Figure 6.1-1 as they would appear on a lateral cephalometric radiograph. The soft tissue structures whose position can be determined from a lateral cephalometric radiograph include the soft palate, the tongue, the hyoid bone and the pharynx. Linear, angular and some sagittal cross-sectional area measures can be taken for many of these structures and will be presented under the appropriate section.

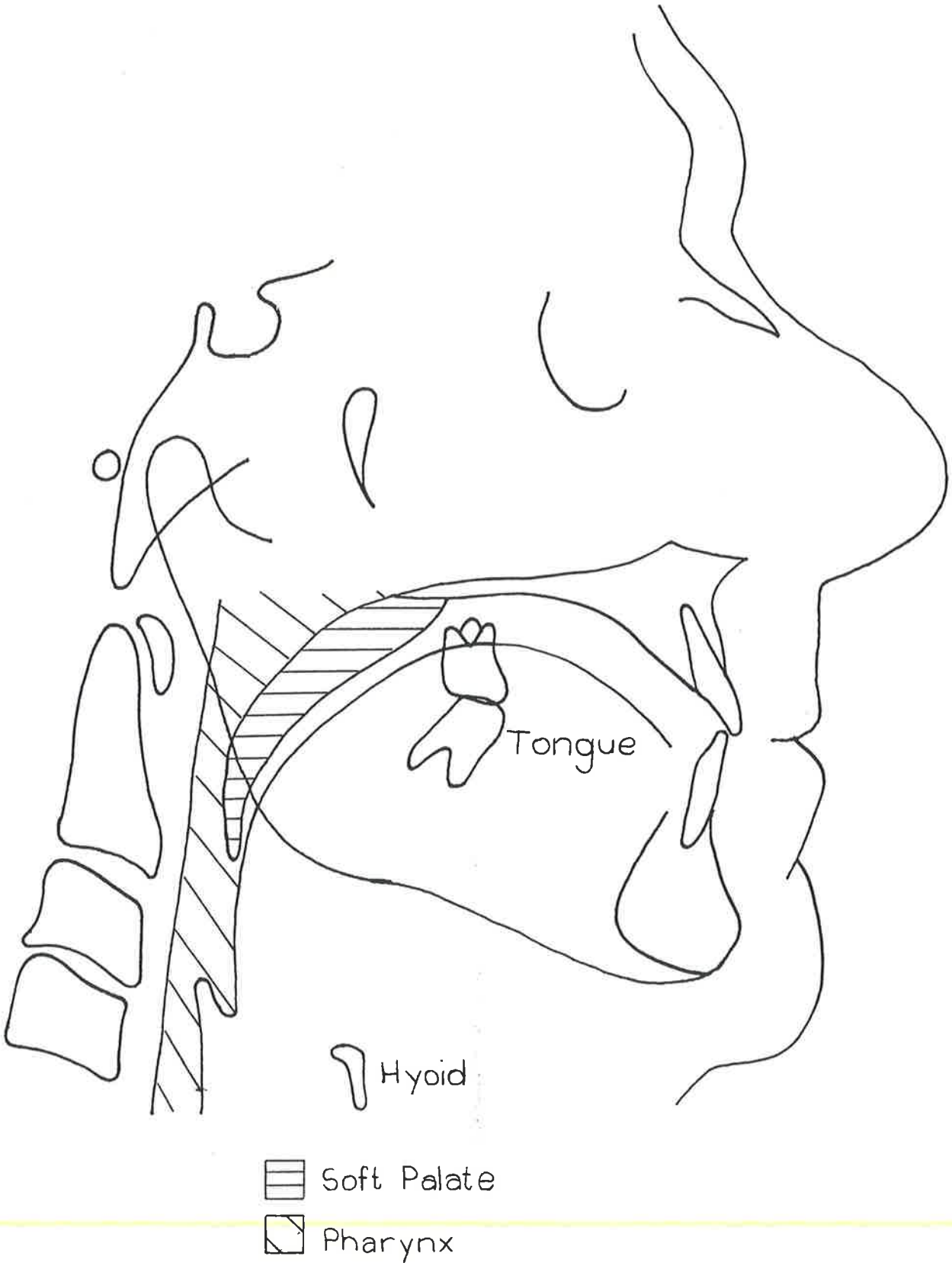


**Figure 6.1-1 Soft Tissue Cephalometric Measurements**

## 6.2 Soft Palate

The soft palate is visible on lateral cephalometric radiographs as a radiopacity extending from the posterior nasal spine caudally and often lying in contact with the pharyngeal surface of the tongue. The soft palate demarcates the nasopharynx inferiorly and the oropharynx superiorly.

The soft palate consists of an aponeurosis to which is attached several muscles. The tensor veli palatini muscle is the main muscle of the soft palate and acts to tense the palatine aponeurosis so the other muscles can act. The levator veli palatini inserts into the palatine aponeurosis and acts to elevate the soft palate ventrally. The palatoglossus and palatopharyngeus constitute little of the soft palate image on the lateral cephalometric radiograph.

The palatopharyngeal sphincter is a separate group of muscle fibres arising from the posterior border of the hard palate and run horizontally backwards encircling the pharynx. They form a ridge (Passavant's ridge) on the anterior aspect of the pharyngeal wall and when the soft palate is elevated these fibres contract, allowing the soft palate to more easily contact the posterior pharyngeal wall.

The nerve supply to the soft palate musculature arises from the pharyngeal plexus (CN XI), with the exception of tensor veli palatini which is innervated by the mandibular division of CN V.

The width of the soft palate is arbitrarily measured as the greatest width of the soft tissue shadow of the soft palate. deBerry-Borowiecki et al (1988); Hochban and Brandenburg (1994); Lowe et al (1996); Lyberg et al (1989b); Ono et al (1996); Strelzow et al (1988); and Tangugsorn et al (1995b) have all reported the greatest width of the soft palate is significantly greater in patients with obstructive sleep apnoea than in their respective control groups. In contrast to these studies Andersson and Brattstrom (1991); Battagel and L'Estrange (1996); Bacon et al (1989); Mayer et al (1996); and Sakakibara et al (1999) did not find a wider soft palate in obstructive sleep apnoea patients.

Lowe et al (1996) divided their obstructive sleep apnoea patients into groups according to their dental malocclusion and the position of the patient when the lateral cephalometric radiograph was taken. They found subjects with a class III dental malocclusion did not have a greatly increased width of the soft palate and that soft palate width varied according to patient position.

Ono et al (1996) did not find a significant difference in soft palate thickness between obstructive sleep apnoea and control subjects on lateral cephalometric radiographs taken in a supine position.

Bacon et al (1989); deBerry-Borowiecki et al (1988); Hochban and Brandenburg (1994); Lowe et al (1996); Lyberg et al (1989b); Maltais et al (1991); Mayer et al (1996); Mochizuki et al (1996); Ono et al (1996); Pracharktam et al (1996); Sakakibara et al (1999); Strelzow et al (1988); Tangugsorn et al (1995b); and Zucconi et al (1993) found the distance from the posterior nasal spine to the tip of the uvula to be significantly greater in patients with obstructive sleep apnoea than control patients. Lowe et al (1996) found this difference was significant only for patients with a Class I or Class II div 1 malocclusion, and only on the lateral cephalometric radiograph taken with the patient upright. Sakakibara et al (1999) found no relationship to obesity and soft palate length in obstructive sleep apnoea patients.

In contrast to these studies Andersson and Brattstrom et al (1991); Battagel and L'Estrange (1996); Johns et al (1998); Mayer and Meier-Ewert (1995); and Pracharktam et al (1994) did not find a significant difference in this parameter when comparing obstructive sleep apnoea subjects with controls.

The angle of the soft palate to the maxillary plane was significantly decreased in the obstructive sleep apnoea patients studied by Lyberg et al (1989b) and Tangugsorn et al (1995b). This partially explains the increased contact length between the tongue and soft palate in obstructive sleep apnoea patients in this study. This angle was not significantly increased in the populations of Battagel and L'Estrange (1996) or Hochban and Brandenburg (1994).

Battagel and L'Estrange (1996); deBerry-Borowiecki et al (1988); Lowe et al (1996); Lyberg et al (1989b); Mochizuki et al (1996); Ono et al (1996); Sakakibara et al (1999); Strelzow et al (1988); and Tangugsorn et al (1995b) reported the area of the soft palate to be significantly greater in patients with obstructive sleep apnoea compared with control groups. Sakakibara et al (1999) found soft palate area was not related to obesity in obstructive sleep apnoea patients. This was not found in populations studied by Pracharktam et al (1994). Lowe et al (1996) found in patients with a Class III dental malocclusion no significant difference in palatal area.

The limits of the soft palate shadow on the lateral cephalometric radiograph were not specified by Battagel and L'Estrange (1996); deBerry-Borowiecki et al (1988); Lowe et al (1996); Sakakibara et al (1999); Strelzow et al (1988) and Pracharktam et al (1994).

Lyberg et al (1989b) determined soft palate area by measuring along the anterior and posterior contour of the soft palate. The superior outline was a line through point pterygomaxillare

(pm) perpendicular to the line joining point pm and point U. Ono et al (1996) measured the area confined by the outline of the soft plate that starts and ends at PNS through P. Mochizuki et al (1996) measured the area of the soft tissue shadow behind the posterior nasal spine to determine the soft palate area. Tangugsorn et al (1995b) defined the outline of the soft palate as lines along the anterior and posterior contour of the soft palate, the superior border was a line through pterygomaxillare (pm) perpendicular to the pm-U line.

The contact length between the dorsum of the tongue and the soft palate was increased in some populations (Lyberg et al, 1989b and Tangugsorn et al, 1995b).

Table 6.2-1 records the authors, the linear and angular measurements of the soft palate. Definitions of the landmarks used may be found in the glossary. Figure 6.2-1 shows diagrammatically the measurements of the soft palate on a lateral cephalometric radiograph.

**Table 6.2-1: Soft Palate – Linear and Angular Measurements**

| | | | UD | UL | UV | ANS-PNS-UT | CL |
|---|---|---|---|---|---|---|---|
| Andersson et al (1991) | A vs C | U | | NS | | | |
| Battagel et al (1996) | A vs C | U | | NS | 0.014 | NS | |
| Bacon et al (1989) | A vs C | U | | | 0.05 | NS | |
| deBerry-Borowiecki et al (1988) | A vs C | U | 0.000 | 0.010 | 0.000 | | |
| Hochban et al (1994) | A vs C | U | 0.005 | 0.0001 | | NS | |
| Johns et al (1998) | A vs S | U | | NS | | | |
| Lowe et al (1996) I | A vs C | U | 0.007 | 0.002 | 0.000 | | |
| Lowe et al (1996) I | A vs C | S | NS | NS | NS | | |
| Lowe et al (1996) II | A vs C | U | 0.023 | 0.009 | 0.001 | | |
| Lowe et al (1996) II | A vs C | S | 0.034 | NS | 0.031 | | |
| Lowe et al (1996) III | A vs C | U | NS | NS | NS | | |
| Lowe et al (1996) III | A vs C | S | NS | NS | NS | | |
| Lyberg et al (1989b) | A vs C | U | 0.001 | 0.001 | 0.001 | 0.01 | 0.01 |
| Maltais et al (1991) | A vs C | U | | 0.01 | | | |
| Mayer et al (1995) | A vs C | U | | NS | | | |
| Mayer et al (1996) | A vs S | U | NS | 0.01 | | | |
| Mochizuki et al (1996) | A vs C | U | | 0.01 | 0.01 | | |
| Ono et al (1996) | A vs C | U | 0.05 | 0.05 | 0.05 | | |
| Ono et al (1996) | A vs C | S | NS | 0.05 | 0.05 | | |
| Pracharktam et al (1994) | A vs S | U | | NS | NS | | |
| Pracharktam et al (1996) | A vs S | U | | 0.013 | | | |
| Sakakibara et al (1999) | A1 vs C | U | NS | 0.02 | 0.001 | | |
| Sakakibara et al (1999) | A2 vs C | U | NS | 0.001 | 0.001 | | |
| Sakakibara et al (1999) | A1 vs A2 | U | NS | NS | NS | | |
| Strelzow et al (1988) | A vs C | U | 0.05 | 0.05 | 0.05 | | |
| Tangugsorn et al (1995b) | A vs C | U | 0.001 | 0.001 | 0.001 | 0.05 | 0.001 |
| Zucconi et al (1992) | A vs S | U | | 0.05 | | | |

A = obstructive sleep apnoea subject; C = non-snoring, non-apnoeic subject; S = snoring, non-apnoeic subject; A1 = non-obese obstructive sleep apnoea subject; A2 = Obese obstructive sleep apnoea subject.

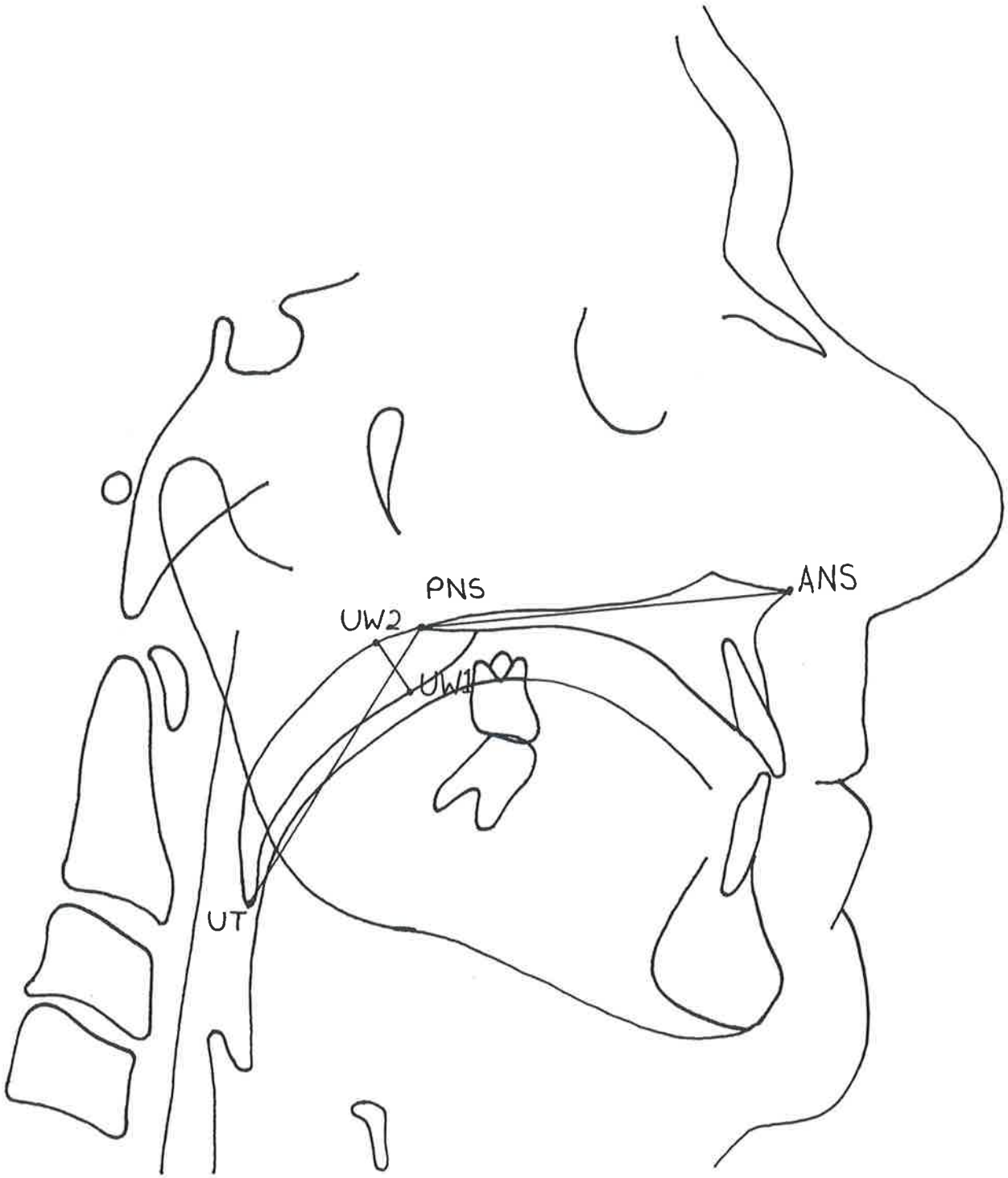


**Figure 6.2-1 Soft Palate cephalometric measures.**

## 6.3 Tongue

The tongue is a skeletal muscle that is very mobile and occupies much of the space of the oral cavity and a substantial volume in the hypopharynx. The so-called anterior two thirds of the tongue in the oral cavity is in fact only approximately one third of the volume of the tongue. The tongue is necessary for swallowing and speech. The muscles of the tongue are broadly categorised as intrinsic or extrinsic muscles. The intrinsic muscles act to change the shape of the tongue whist the extrinsic muscles act to alter the position of the tongue within the oral cavity.

The extrinsic muscles may arise from the hyoid bone inferiorly (hyoglossus, chondroglossus), the genial tubercles on the lingual aspect of the mandibular symphysis anteriorly (genioglossus), the styloid process of the temporal bone superolaterally (styloglossus) or from the palatine aponeurosis superomedially (palatoglossus).

The bulk of the tongue that is distinguished on a lateral cephalometric radiograph consists of the intrinsic muscles, the genioglossus and the geniohyoid. The tongue base, consisting primarily of genioglossus extends caudally into the oropharynx and forms the vallecula at its most caudal point.

The outline of the tongue in the oral cavity may be difficult to distinguish on a lateral cephalometric radiograph and consequently various radiopaque solutions have been used to demarcate the surface of the tongue (Battagel and L'Estrange, 1996; Lowe et al, 1995; Lowe et al, 1996; Ono et al, 1996; Ozbek et al, 1998; Pae et al, 1994; Pracharktam et al, 1994; Pracharktam et al, 1996; and Tsuchiya et al, 1992).

The length of the tongue from its apex to base has been measured from the tongue tip to the vallecula, or the tongue base on an extension of the mandibular plane or to the tip of the epiglottis. The length of the tongue was increased when measured to the tongue base by deBerry-Borowiecki et al (1988); Hochban and Brandenburg (1994); Lowe et al (1996); Pracharktam et al (1994); Pracharktam et al (1996); Sakakibara et al (1999); and Strelzow et al (1988). The length of the tongue was increased when measured to the vallecula by deBerry-Borowiecki et al (1988) also found an increase in tongue length when measured to the tip of the epiglottis. This is in contrast to Hochban and Brandenburg (1994) who found no difference in this dimension.

Lowe et al (1996) found no significant difference in tongue length between obstructive sleep apnoea subjects with a Class I or Class II dental malocclusion on supine radiographs. Interestingly they did find a significant difference for obstructive sleep apnoea subjects with a Class III dental malocclusion on supine cephalometric radiographs, but not on upright

radiographs. Sakakibara et al (1999) found non-obese obstructive sleep apnoea subjects did not have a significantly increased tongue length. Other authors have also reported no increase in tongue length for obstructive sleep apnoea subjects (Lyberg et al, 1989b; and Tangugsorn et al, 1995a).

Tongue depth is a measure of the caudal extension of the tongue base or vallecula to skeletal reference points on the maxilla, mandible or cranium. A majority of authors who have measured this relationship have found the tongue lying more caudally in obstructive sleep apnoea subjects than control subjects (deBerry-Borowiecki et al, 1988; Hochban and Brandenburg, 1994; Johns et al, 1998; Lyberg et al, 1989b; Sakakibara et al, 1999; Strelzow et al, 1988; and Tangugsorn et al, 1995b). This increase in depth of the vallecula places the tongue in a more inferior and posterior position compared with control subjects. This may allow obstruction of the upper airway to occur more readily in these patients than control subjects by placing the bulk of the tongue closer to the relatively more narrow hypopharynx.

deBerry-Borowiecki et al (1988) did not find a significant difference in the position of the tongue base with respect to gonion. Mayer and Meier-Ewert (1995) found no significant decrease in distance PNS-TB between obstructive sleep apnoea subjects and controls.

Tongue height measures the "thickness" or "bulk" of the tongue. A perpendicular line to the reference line TT-V is taken such that the perpendicular is maximum length. Hochban and Brandenburg (1994); Lowe et al (1996) and Sakakibara et al (1999) all found this perpendicular to be significantly longer in obstructive sleep apnoea patients. Lowe et al (1996) reported no significant difference on supine radiographs or for obstructive sleep apnoea subjects with a Class I dental malocclusion in the upright position. Sakakibara et al (1999) reported no difference in this tongue dimension between obstructive sleep apnoea sub-groups. Johns et al (1998) and Lyberg et al (1989b) found no significant increase in this dimension for obstructive sleep apnoea subjects.

A measure of the position of the inferior tongue with respect to the anterior mandible has also been considered. This measurement indicates how posterior the inferior tongue lies. Hochban and Brandenburg (1994) and Strelzow et al (1988) found this length significantly increased in obstructive sleep apnoea patients. deBerry-Borowiecki et al (1988) and Hochban and Brandenburg (1994) found no difference in other measures of this relationship of the tongue to the mandible in obstructive sleep apnoea subjects. Lyberg et al (1989b) considered the proximity of the vallecula to the cervical spine and found no significant difference between obstructive sleep apnoea subjects and controls.

Tongue area has been measured by a number of investigators, most of whom found a significantly increased area in obstructive sleep apnoea subjects compared to control subjects

(deBerry-Borowiecki et al, 1988; Mochizuki et al, 1996; Pracharktam et al, 1996; Sakakibara et al, 1999; Strelzow et al, 1988; and Tangugsorn et al, 1995b).

The area of the tongue was defined by deBerry-Borowiecki et al (1988) as the area determined by measuring the superior limit of the tongue and the line contained between the apex of the tongue (TT), the genial tubercle (G), the hyoid bone (H) and a line parallel to Frankfort Horizontal (FH) up to the epiglottic apex (EA). Lowe et al (1996) defined the area of the tongue as the area enclosed by the dorsal configuration of the tongue surface and lines that connect tongue tip (TT), retrognathion (RGN), the hyoid (H) and the epiglottic base (Eb). Mochizuki et al (1996) defined the area of the tongue as the soft tissue shadow above the line connecting the vallecula, the upper edge of the hyoid bone and the mental spine. The area of the tongue defined by Pracharktam et al (1994) enclosed the lines joining the tongue tip (Tt) along retrognathion surface to menton, the base of the epiglottis (Eb) and dorsum of the tongue. Sakakibara et al (1999) measured tongue area in a similar manner to deBerry-Borowiecki et al (1988), replacing point EA with vallecula (V). Lyberg et al (1989b) and Tangugsorn et al (1995b) defined the upper outline by the dorsal contour of the tongue from vallecula (V) through point H to the tongue tip (T). The lower outline was defined by lines connecting vallecula (V), point AH on the hyoid, the genial tubercle (GE) and the tongue tip (T).

Strelzow et al (1988) did not clearly define the soft tissue shadow measured to determine the tongue area.

Battagel and L'Estrange (1996); Lowe et al (1996); Lyberg et al (1989b); and Pracharktam et al (1994) reported no significant difference in tongue area between obstructive sleep apnoea subjects and controls.

The proportion of the oropharyngeal area occupied by the tongue was measured by two authors, both finding the tongue occupying a greater proportion of this area in obstructive sleep apnoea subjects (Battagel and L'Estrange, 1996; and Tangugsorn et al, 1995b).

Nelson and Hans (1997) found tongue length to be a predictor of obstructive sleep apnoca severity in both obese and nonobese subjects.

Table 6.3-1 records the authors and the linear measurements of the tongue. Definitions of the landmarks used may be found in the glossary.

**Table 6.3-1: Tongue – Linear Measurements**

| | | | V - TT | Depth | Height | Length | Area | Proportion |
|---|---|---|---|---|---|---|---|---|
| Battagel et al (1996) | A vs C | U | | | | | NS | 0.01[9] |
| deBerry-Borowiecki et al (1988) | A vs C | U | 0.003 | 0.000[1] | | NS[2] | 0.002 | |
| deBerry-Borowiecki et al (1988) | A vs C | U | 0.0083 | NS[4] | | NS[5] | | |
| Hochban et al (1994) | A vs C | U | 0.000[1] | 0.002[6] | 0.000[1] | 0.01[7] | | |
| Hochban et al (1994) | A vs C | U | 0.005[8] | 0.0001[9] | | NS[10] | | |
| Hochban et al (1994) | A vs C | U | NS[3] | 0.0001[11] | | | | |
| Hochban et al (1994) | A vs C | U | | 0.01[12] | | | | |
| Johns et al (1998) | A vs S | U | | 0.01[13] | NS | | | |
| Lowe et al (1996) I | A vs C | U | 0.045 | | NS | | NS | |
| Lowe et al (1996) I | A vs C | S | NS | | NS | | NS | |
| Lowe et al (1996) II | A vs C | U | 0.045 | | 0.015 | | NS | |
| Lowe et al (1996) II | A vs C | S | NS | | NS | | NS | |
| Lowe et al (1996) III | A vs C | U | NS | | 0.030 | | NS | |
| Lowe et al (1996) III | A vs C | S | 0.041 | | NS | | NS | |
| Lyberg et al (1995b) | A vs C | U | NS | 0.011[6] | NS | NS[14] | NS | |
| Mayer et al (1995) | A vs C | U | | NS[6] | | | | |
| Mochizuki et al (1996) | A vs S | U | | | | | 0.05 | |
| Pracharktam et al (1994) | A vs S | U | 0.01[15] | | | | NS | |
| Pracharktam et al (1996) | A vs S | U | 0.006[15] | | | | 0.004 | 0.01 |
| Sakakibara et al (1999) | A1 vs C | U | NS | 0.001 | 0.02 | | 0.001 | |
| Sakakibara et al (1999) | A2 vs C | U | 0.001 | 0.001 | 0.001 | | 0.001 | |
| Sakakibara et al (1999) | A1 vs A2 | U | 0.001 | NS | NS | | 0.001 | |
| Strelzow et al (1988) | A vs C | U | 0.05[15] | 0.05[12] | | 0.05[10] | 0.05 | |
| Tangugsorn et al (1995b) | A vs C | U | NS | 0.05[15] | | | 0.001 | |

A = obstructive sleep apnoea subject; C = non-snoring, non-apnoeic subject; S = snoring, non-apnoeic subject; A1 = non-obese obstructive sleep apnoea subject; A2 = Obese obstructive sleep apnoea subject.
1 PNS-TB; 2 B-TB; 3 TT – ET; 4 Go-TB; 5 Gn-TB; 6 TB – PNS; 7 V – Me; 8 TT – TB; 9 V – S; 10 TB – B; 11 V – ANS; 12 TB – ANS; 13 PNS – EB; 14 V – C spine parallel to FH; 15 TT – EB; 16 V – FH.

The tongue assumed a more upright position, as measured by V-TT/FH in the obstructive sleep apnoea populations of Lyberg et al (1989b); and Tangugsorn et al (1995b), however this was not the case for the population studied by Hochban and Brandenburg (1994).

Table 6.3-2 records the authors and the angular measurements of the tongue. Definitions of the landmarks used may be found in the glossary. Figure 6.3-1 shows diagrammatically the tongue measurements taken on a lateral cephalometric radiograph.

**Table 6.3-2: Tongue – Angular Measurements**

| | | | V-TT/Go-Gn | V-TT/FH |
|---|---|---|---|---|
| Hochban et al (1994) | A vs C | U | NS | NS |
| Lyberg et al (1995b) | A vs C | U | | 0.001 |
| Tangugsorn et al (1995b) | A vs C | U | | 0.05 |

A = obstructive sleep apnoea subject; C = non-snoring, non-apnoeic subject

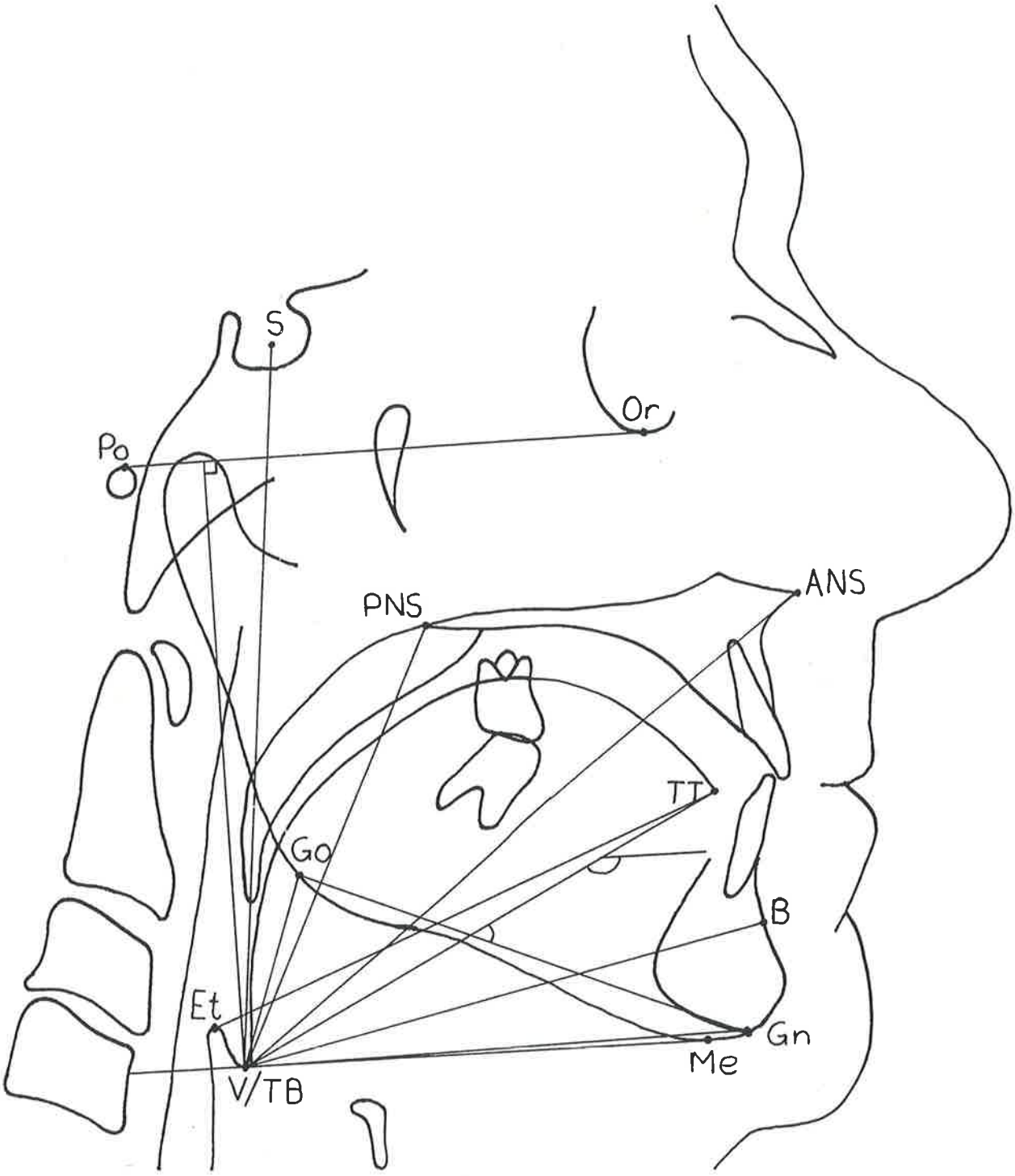


**Figure 6.3-1 Tongue cephalometric measures**

## 6.4 Hyoid Bone

The hyoid is a thin U-shaped bone suspended below the lower border of the mandible, above the larynx. The hyoid consists of a central body and two posterolateral bony projections, the greater cornu. The lesser cornu is a bony prominence at the junction of the body and greater cornu.

The body of the hyoid serves as the attachment of genioglossus, geniohyoid, mylohyoid and part of hyoglossus (suprahyoid) muscles as well as sternohyoid and omohyoid (infrahyoid) muscles. The fibrous sling of the digastric muscle and the attachment of stylohyoid lie on the greater cornu near its origin from the body of the hyoid. The middle constrictor and posterior part of hyoglossus are attached to the upper border of the greater cornu. The medial surface of the greater cornu serves as the attachment of the thyrohyoid membrane. The lesser cornu serves as the inferior attachment of the stylohyoid ligament.

The linear distance between the mandibular plane and a perpendicular to the most anterior superior point on the body of the hyoid has been measured by a large number of authors. This distance has was increased in populations studied by Andersson and Brattstrom (1991); Hochban and Brandenburg (1994); Lowe et al (1996); Lyberg et al (1989b); Maltais et al (1991); Mayer et al (1996); Mochizuki et al (1996); Pracharktam et al (1994); Pracharktam et al (1996); Sakakibara et al (1999); Tangugsorn et al (1995a) and Tsuchiya et al (1992). No difference between obstructive sleep apnoea subjects and controls was found for patients with a Class II dental malocclusion or Class III dental malocclusion if the radiograph was taken supine (Lowe et al, 1996). Sakakibara et al (1999) found no difference in this dimension between obese and non-obese obstructive sleep apnoea subjects. Patients with a high AHI and low BMI did not have a significant increase in this distance compared with a control population (Tsuchiya et al, 1992), however this group had a significant decreased distance compared with the low AHI/high BMI group. Other authors reported no significant difference between obstructive sleep apnoea subjects and controls for this measurement (Battagel and L'Estrange, 1996; and Mayer and Meier-Ewert, 1995).

The vertical position of the hyoid is determined by its relationship to a point on the skeleton lying superiorly or a constructed reference line. A number of different landmarks have been used to enable this vertical relationship of the hyoid to be determined. All authors who found a significant difference in vertical position of the hyoid between obstructive sleep apnoea subjects and controls found the hyoid was placed more caudally in the obstructive sleep apnoea subject (deBerry-Borowiecki et al, 1988; Hochban and Brandenburg, 1994; Johns et al, 1998; Lowe et al, 1996; Lyberg et al, 1989a; Pracharktam et al, 1996; Strelzow et al, 1988;

Tangugsorn et al, 1995a; and Tsuchiya et al, 1992). Lowe et al (1996) found the hyoid was not caudally placed in obstructive sleep apnoea subjects with a Class II or Class III dental malocclusion on either upright or supine cephalometric radiographs. Lyberg et al (1989) found the hyoid was not caudally placed in relation to the cranial base, but obstructive sleep apnoea subjects had a caudally placed hyoid in relation to a perpendicular line to Frankfort horizontal. Tsuchiya et al (1992) found the high AHI/low BMI group had a normally placed hyoid, but the low AHI/high BMI group had a caudally placed hyoid with respect to controls and the high AHI/low BMI group. Battagel and L'Estrange (1996) found no significant difference in the vertical position of the hyoid.

The vertical position of the hyoid has been measured in relationship to C3 by Tangugsorn et al (1995a) who found a significantly caudal relationship of the hyoid in obstructive sleep apnoea subjects.

The anteroposterior position of the hyoid (or horizontal position) has been considered. One of the problems with the assessment of this parameter is the tangential nature of many of the distances measured. There is no true horizontal assessment of hyoid position using these measures because most relate to a landmark on the anterior maxilla or mandible. All these constructed lines have a significant vertical component and may therefore diminish the sensitivity of the horizontal position of the hyoid because of the vectors involved. A better estimate of the anteroposterior position of the hyoid is by its relationship to the cervical spine. The horizontal distance to a point or reference line on the cervical spine allows a true horizontal measurement to be taken, with little or no vertical component.

The anteroposterior position of the hyoid varies markedly between populations studied. Some authors have reported a more posteriorly placed hyoid in obstructive sleep apnoea subjects (Hochban and Brandenburg, 1994; Lowe et al, 1996; Pracharktam et al, 1994; and Strelzow et al, 1988). Others have found no difference between obstructive sleep apnoea subjects and controls for this parameter (Battagel and L'Estrange, 1996; deBerry-Borowiecki et al, 1988; Hochban and Brandenburg, 1994; Lowe et al, 1996; Strelzow et al, 1988; and Tsuchiya et al, 1992). Battagel and L'Estrange (1996) even found a more anteriorly place hyoid in obstructive sleep apnoea subjects with respect to point B.

The horizontal relationship to the cervical spine has usually been considered with respect to C3 as this is the vertebrae at which the hyoid usually lies in closest vertical proximity. Variable results have been found in this relationship. Tangugsorn et al (1995a) measured a significantly decreased distance from the hyoid to C3 for obstructive sleep apnoea subjects. Lowe et al (1996) reported a similar result for patients with a Class I dental malocclusion on upright cephalometric radiographs and for subjects with a Class III dental malocclusion on

supine radiographs. They found no significant differences in this relationship for all other groups studied. Sakakibara et al (1999) found the hyoid to be significantly closer to the cervical spine in obese obstructive sleep apnoea subjects compared to normal controls and non-obese obstructive sleep apnoea subjects. They found no significant difference between non-obese obstructive sleep apnoea subjects and controls for this dimension. Tsuchiya et al (1992) found this dimension was significantly larger in low AHI/high BMI subjects compared with controls. There was no significant difference between the high AHI/low BMI group and controls or between the two obstructive sleep apnoea groups.

Table 6.4-1 records the authors and the linear measurements of the hyoid bone. Definitions of the landmarks used may be found in the glossary.

**Table 6.4-1: Hyoid Bone – Linear Measurements**

| | | | H - MP | Vertical | Horizontal | C-Spine |
|---|---|---|---|---|---|---|
| Andersson et al (1991) | A vs C | U | 0.05 | | | |
| Andersson et al (1991) | A vs S | U | 0.01 | | | |
| Battagel et al (1996) | A vs C | U | NS | NS[1] | 0.042[2] | |
| Battagel et al (1996) | A vs C | U | | | NS[3] | |
| deBerry-Borowiecki et al (1988) | A vs C | U | | 0.009[4] | NS[5] | |
| deBerry-Borowiecki et al (1988) | A vs C | U | | 0.001[6] | NS[7] | |
| deBerry-Borowiecki et al (1988) | A vs C | U | | 0.043[8] | | |
| Hochban et al (1994) | A vs C | U | 0.000[1] | 0.0001[4] | 0.0005[2] | |
| Hochban et al (1994) | A vs C | U | | 0.05[9] | 0.05[10] | |
| Hochban et al (1994) | A vs C | U | | | NS[7] | |
| Hochban et al (1994) | A vs C | U | | | 0.005[11] | |
| Johns et al (1998) | A vs S | U | 0.01 | 0.01[12] | | |
| Lowe et al (1996) I | A vs C | U | 0.003 | 0.005[13] | NS[14] | 0.031[15] |
| Lowe et al (1996) I | A vs C | S | 0.001 | 0.005[13] | NS[14] | NS[15] |
| Lowe et al (1996) II | A vs C | U | 0.031 | NS[13] | NS[14] | NS[15] |
| Lowe et al (1996) II | A vs C | S | NS | NS[13] | 0.032[14] | NS[15] |
| Lowe et al (1996) III | A vs C | U | 0.047 | NS[13] | NS[14] | 0.001[15] |
| Lowe et al (1996) III | A vs C | S | NS | NS[13] | NS[14] | NS[15] |
| Lyberg et al (1995a) | A vs C | U | 0.001 | NS[4] | | NS[15] |
| Lyberg et al (1995a) | A vs C | U | | 0.001[16] | | |
| Maltais et al (1991) | A vs C | U | 0.05 | | | |
| Mayer et al (1995) | A vs C | U | NS | | | |
| Mayer et al (1996) | A vs S | U | 0.01 | | | |
| Mochizuki et al (1996) | A vs S | U | 0.01 | | | |
| Pracharktam et al (1994) | A vs S | U | 0.001 | | 0.05[15] | |
| Pracharktam et al (1996) | A vs S | U | 0.000 | 0.01[17] | | |
| Sakakibara et al (1999) | A1 vs C | U | 0.001 | | | NS[19] |
| Sakakibara et al (1999) | A2 vs C | U | 0.001 | | | 0.001[19] |
| Sakakibara et al (1999) | A1vs A2 | U | NS | | | 0.001[19] |
| Strelzow et al (1988) | A vs C | U | | 0.05[6] | 0.05[18] | |
| Strelzow et al (1988) | A vs C | U | | 0.05[4] | NS[5] | |
| Strelzow et al (1988) | A vs C | U | | 0.05[8] | | |
| Tangugsorn et al (1995a) | A vs C | U | 0.001 | 0.001[4] | | NS[20] |
| Tangugsorn et al (1995a) | A vs C | U | | 0.001[16] | | 0.001[21] |
| Tsuchiya et al (1992) | A3 vs C | U | NS | NS[13] | NS[15] | NS[22] |
| Tsuchiya et al (1992) | A4 vs C | U | 0.01 | 0.01[13] | NS[15] | 0.01[22] |
| Tsuchiya et al (1992) | A3 vs A4 | U | 0.05 | 0.01[13] | 0.05[15] | NS[22] |
| Zucconi et al (1992) | A vs C | U | 0.001 | | | |

A = obstructive sleep apnoea subject; C = non-snoring, non-apnoeic subject; S = snoring, non-apnoeic subject; A1 = non-obese obstructive sleep apnoea subject; A2 = Obese obstructive sleep apnoea subject; High apnoea index/Low BMI; A4 Low apnoea index/High BMI.

1 H/ANS-PNS; 2 H – B; 3 ANS-H; 4 S – H; 5 Gn-H; 6 Ar-H; 7 H-PhW; 8 Go-H; 9 H-AA; 10 H–Me; 11 H-PhW (Me-H); 12 PNS-H; 13 H-H1; 14 H-RGn; 15 C3 – H; 16 H (FH; 17 H-Ver; 18 H-PhW (Go-H); 19 H-VL; 20 AH-C3 Hor; 21 AH-C3 Ver; 22 C3-H.

The angular position of the hyoid can be related principally to its vertical position in relation to other reference lines of the cranial or facial skeleton.

The authors who have considered the angular relationship between the hyoid and mandibular plane, and the cranial base and the line from the hyoid passing through articulare are in agreement (deBerry-Borowiecki et al, 1988; Hochban and Brandenburg, 1994; and Strelzow et al, 1988). They have all found an increase in the angular relationship of the hyoid and therefore agree it is inferiorly placed in respect to the cranium and the mandible. Strelzow et al (1988) did not find an increase in the angle between the cranial base and the body of the hyoid.

The angle between the hyoid and a line joining the third cervical vertebrae and point retrognathion was measured by Pracharktam et al (1994). They found an increase in this angle in obstructive sleep apnoea patients and concluded that this showed an inferiorly placed hyoid in this group.

Table 6.4-2 records the authors and the angular measurements of the hyoid bone. Definitions of the landmarks used may be found in the glossary. Figure 6.4-1 shows diagrammatically the measurements of hyoid bone position on a lateral cephalometric radiograph.

**Table 6.4-2: Hyoid Bone – Angular Measurements**

| | | | Go-Gn-H | N-S/Ar-H | N-S-H | C3-RGn-H |
|---|---|---|---|---|---|---|
| deBerry-Borowiecki et al (1988) | A vs C | U | 0.000 | 0.017 | 0.042 | |
| Hochban et al (1994) | A vs C | U | 0.005 | 0.0001 | 0.0001 | |
| Pracharktam et al (1994) | A vs S | U | | | | 0.001 |
| Strelzow et al (1998) | A vs C | U | 0.05 | 0.05 | NS | |

A = obstructive sleep apnoea subject; C = non-snoring, non-apnoeic subject; S = snoring, non-apnoeic subject.

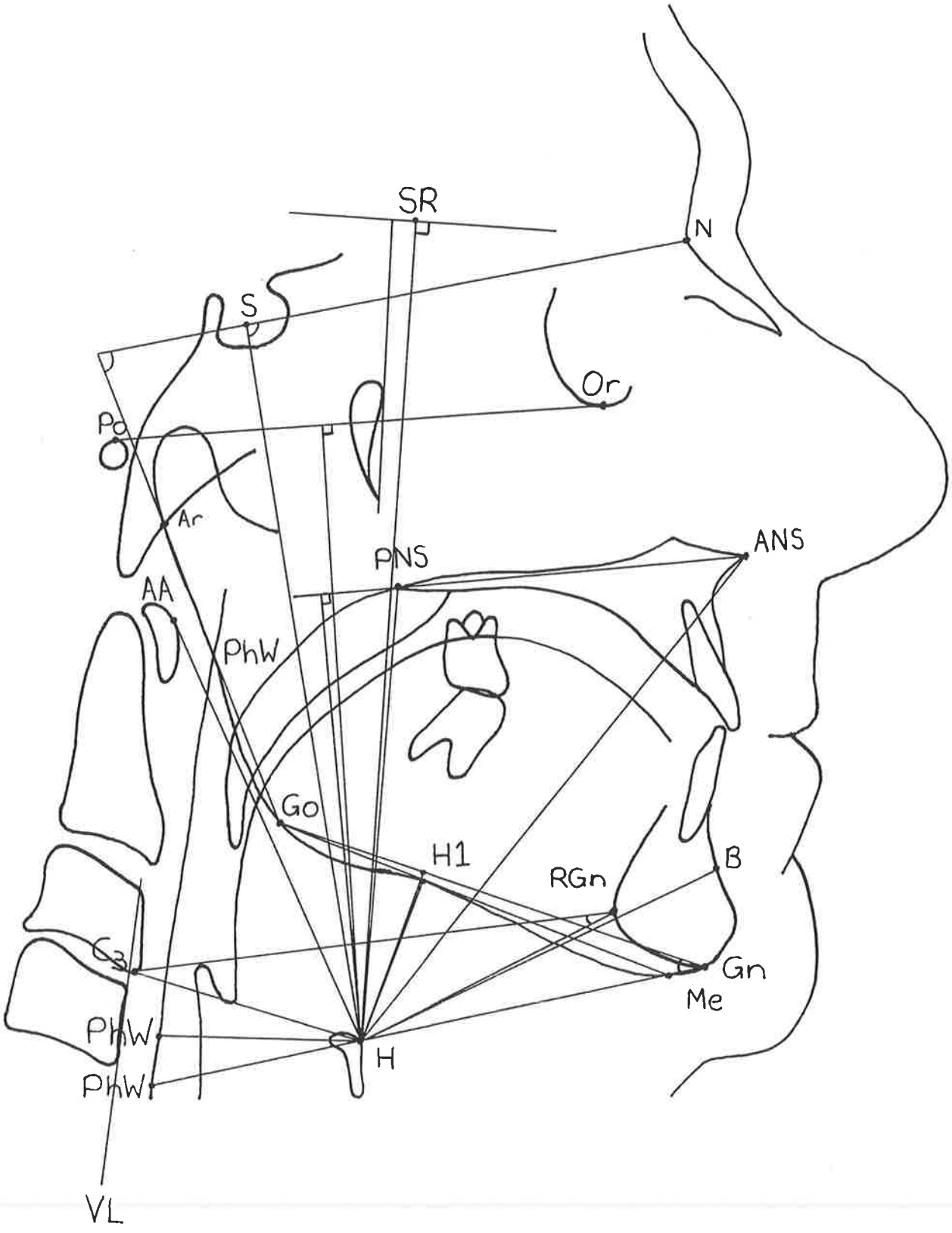


**Figure 6.4-1 Hyoid Bone cephalometric measures.**

## 6.5 Nasopharyngeal Airway

The upper airway is a radiolucent area on the lateral cephalometric radiograph that extends superiorly to the skull base, inferiorly to the pharyngoesophageal inlet, anteriorly to the posterior surface of the soft palate and tongue and posteriorly to the posterior wall of the pharynx.

The upper airway is divided into the nasopharynx (the area above the level of the soft palate), oropharynx (the area below the soft palate and above the hyoid) and the hypopharynx (the area below the hyoid and above the pharyngoesophageal inlet). The area bounded by lines connecting the cephalometric landmarks pharyngeal tubercle (PhT), sphenoidal rostrum (SR), posterior nasal spine (PNS), anterior tubercle of atlas (ATA) and the pharyngeal tubercle (PhT) is defined as the nasopharyngeal space by deBerry-Borowiecki et al (1988). Lowe et al (1996) and Ono et al (1996) defined this area as being outlined by a line between point R, the posterior nasal spine (PNS), the point of intersection on the posterior pharyngeal wall of an extension of the palatal plane and the shadow of the posterior pharyngeal wall superiorly to point R. Strelzow et al (1988) defined the nasopharyngeal boundaries as lines connecting the posterior nasal spine (PNS), sphenoidal rostrum (Sr), articulare (Ar) and inferiorly along the posterior pharyngeal wall to a line from the atlas to the posterior nasal spine (PNS).

The measurements of the upper airway differ in most studies by using different landmarks or reference lines, making direct comparison difficult. Authors have generally considered the nasopharyngeal airway at the level of the posterior nasal spine, the tip of the uvula and a point on the dorsum of the soft palate, usually the most dorsal and superior point. The distance is measured from these three points to the posterior wall of the pharynx along various reference planes.

Andersson and Brattstrom (1991); Hochban and Brandenburg (1994); Lyberg et al (1989b); Sakakibara et al (1999); Solow et al (1996); and Tangugsorn et al (1995b) found a significantly decreased distance between the PNS and pharyngeal wall in obstructive sleep apnoea patients. Sakakibara did not find a significant difference in this space between obese and non-obese obstructive sleep apnoea subjects. Other authors have not found this dimension to be significantly less in their obstructive sleep apnoea population compared to a control group (deBerry-Borowiecki et al, 1988; and Strelzow et al, 1988).

Battagel and L'Estrange (1996); deBerry-Borowiecki et al (1988); Johns et al (1998); Lowe et al (1996); Pracharktam et al (1994); Sakakibara et al (1999); Solow et al (1996); and Strelzow et al (1988) measured a significant difference in the distance between the soft palate and the pharyngeal wall (nasopharynx) in obstructive sleep apnoea subjects compared with controls.

Lowe et al (1996) did not find a significantly decreased space between the soft palate and the pharyngeal wall in obstructive sleep apnoea subjects with a Class III dental malocclusion. Sakakibara et al (1999) found no significant difference in this measurement between obese and non-obese obstructive sleep apnoea subjects.

Mochizuki et al (1996); Pracharktam et al (1996) found no significant decrease in the pharyngeal airway space at the level of the soft palate between obstructive sleep apnoea and control subjects.

The distance from the tip of the soft palate (uvula) and the posterior pharyngeal wall was significantly less in some obstructive sleep apnoea populations compared with either snoring or non-snoring controls (Battagel and L'Estrange 1996; deBerry-Borowiecki et al, 1988; Hochban and Brandenburg, 1994; Lowe et al 1996; Lyberg et al, 1989b; Sakakibara et al, 1999; Solow et al, 1996; and Tangugsorn et al, 1995b).

Lowe et al (1996) found no significant decrease in this airway dimension in patients with a Class III dental malocclusion, or in patients with a Class II dental malocclusion if the lateral cephalometric radiograph was taken in a supine position. found no significant difference in this dimension between obese and non-obese obstructive sleep apnoea subjects.

Several other authors (Johns et al, 1998; and Mayer and Meier-Ewert, 1995) did not find a significant decrease in distance between the uvula and pharyngeal wall in obstructive sleep apnoea subjects.

Few authors have measured the area of the nasopharynx from lateral cephalometric radiographs. deBerry-Borowiecki et al (1988); Lowe et al (1996) and Ono et al (1996) found no significant difference in this area for obstructive sleep apnoea subjects, whereas Strelzow et al (1988) found obstructive sleep apnoea subjects had a significantly decreased nasopharyngeal area compared with a control population.

Table 6.5-1 records the authors, the linear and angular measurements of the nasopharyngeal airway. Definitions of the landmarks used may be found in the glossary. Figure 6.5-1 shows diagrammatically nasopharyngeal cephalometric airway measurements.

**Table 6.5-1: Nasopharyngeal Airway Measurements**

| | | | PNS – PhW | UP-PhW | UT-PhW | Area |
|---|---|---|---|---|---|---|
| Andersson et al (1991) | A vs C | U | 0.001[1] | | | |
| Battagel et al (1991) | A vs C | U | | 0.000[2] | 0.005[2] | |
| Battagel et al (1991) | A vs C | U | | 0.000[2,3] | | |
| Battagel et al (1991) | A vs C | U | | 0.000[4] | | |
| deBerry-Borowiecki et al (1988) | A vs C | U | NS[5] | 0.002[5] | 0.005[5] | NS |
| Hochban et al (1994) | A vs C | U | 0.0001[1] | | 0.001[6] | |
| Hochban et al (1994) | A vs C | U | 0.0001[7] | | | |
| Johns et al (1998) | A vs S | U | | 0.01[8] | NS[5] | |
| Lowe et al (1996) I | A vs C | U | | 0.000[9] | 0.000[9] | NS |
| Lowe et al (1996) I | A vs C | S | | 0.035[9] | 0.027[9] | NS |
| Lowe et al (1996) II | A vs C | U | | 0.000[9] | 0.01[9] | NS |
| Lowe et al (1996) II | A vs C | S | | 0.001[9] | NS[9] | NS |
| Lowe et al (1996) III | A vs C | U | | NS[9] | NS[9] | NS |
| Lowe et al (1996) III | A vs C | S | | NS[9] | NS[9] | NS |
| Lyberg et al (1989b) | A vs C | U | 0.001[9] | | 0.001[9] | |
| Mayer et al (1995) | A vs C | U | | | NS[9] | |
| Mochizuki et al (1996) | A vs S | U | | NS[7] | | |
| Ono et al (1996) | A vs C | U | | | | NS |
| Pracharktam et al (1994) | A vs S | U | | 0.05[9] | | |
| Pracharktam et al (1996) | A vs S | U | | NS[9] | | |
| Sakakibara et al (1999) | A1 vs C | U | 0.001[7] | 0.001[7] | 0.001[7] | |
| Sakakibara et al (1999) | A2 vs C | U | 0.001[7] | 0.001[7] | 0.001[7] | |
| Sakakibara et al (1999) | A1 vs A2 | U | NS[7] | NS[7] | NS[7] | |
| Solow et al (1996) | A vs C | U | 0.01[10] | 0.001[11] | 0.001[11] | |
| Solow et al (1996) | A vs C | U | 0.001[7] | | | |
| Strelzow et al (1988) | A vs C | U | NS[11] | 0.05[11] | 0.05[11] | 0.05 |
| Tangugsorn et al (1995b) | A vs C | U | 0.001[7] | | 0.001[7] | |

A = obstructive sleep apnoea subject; C = non-snoring, non-apnoeic subject; S = snoring, non-apnoeic subject; A1 = non-obese obstructive sleep apnoea subject; A2 = Obese obstructive sleep apnoea subject.

1 Distance along extension of line ANS-PNS; 2 Distance along a horizontal line; 3 The most posterior point on the dorsum of the soft palate; 4 Distance along a horizontal line through the lower incisor tip; 5 Distance along a line parallel to FH; 6 Distance along extension of the occlusal plane; 7 Distance along line PNS-Ba (or parallel) between soft palate and pharyngeal wall; 8 Distance along line parallel to FH at midpoint of soft palate; 9 Distance along line parallel to Go-B; 10 Distance along line parallel to Go-B; 11 Reference line not defined.

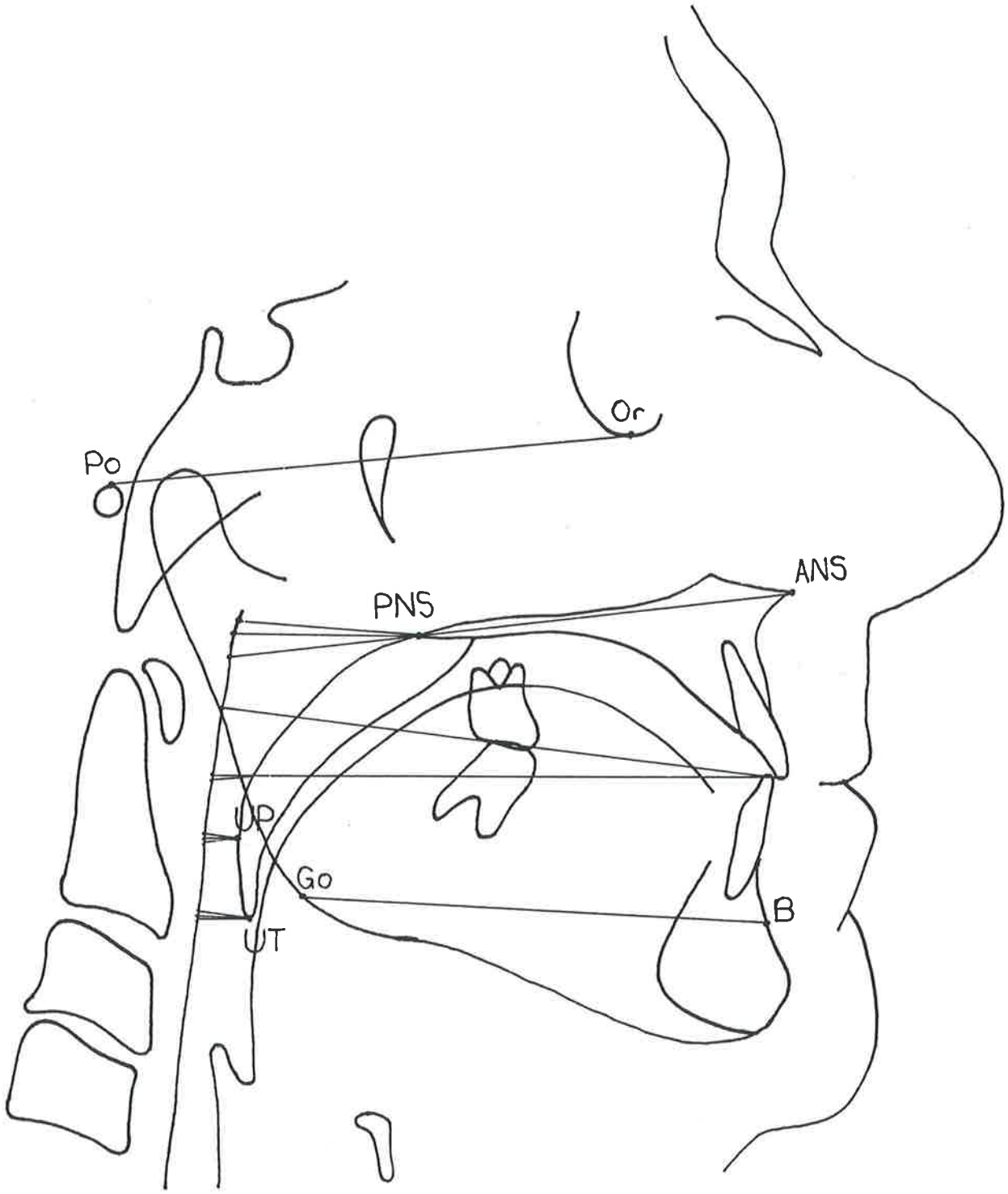


**Figure 6.5-1 Nasopharyngeal Airway cephalometric measures.**

## 6.6 Oropharyngeal Airway

The definition of the borders of the oropharyngeal airway differs in much the same way as definitions vary for the nasopharyngeal and hypopharyngeal airways. Lowe et al (1996) and Ono et al (1996) defined the oropharyngeal area as bounded by the inferior border of the nasopharynx, posterior surface of the soft palate, line parallel to palatal plane from point P to dorsal surface of tongue, posterior inferior surface of tongue and a line parallel to the palatal plane through the tip of the epiglottis (Et) and the posterior pharyngeal wall. The inferior border of the nasopharynx is a line extending the palatal plane to intersect with the posterior pharyngeal wall. Tangugsorn et al (1995b) defined the oropharyngeal area to include the oral area (including the tongue), the soft palate area and the area defined by lines joining pterygomaxillare (pm), point UPW, point LPW and vallecula (V) along the posterior pharyngeal wall and the dorsum of the tongue.

The oropharyngeal airway sagittal dimension between the dorsal surface of the tongue and the posterior pharyngeal wall has been measured by a number of investigators. Essentially three lines of reference have been used to orientate these measurements vertically. They are an extension of the occlusal plane, an extension of a line passing through Go-B and an extension of the lower border of the mandible.

Along an extension of the occlusal plane Hochban and Brandenburg (1994) found a significant decrease in oropharyngeal airway sagittal dimension in obstructive sleep apnoea subjects.

Battagel and L'Estrange (1996); Lowe et al (1996); Lyberg et al (1989b); and Maltais et al (1991) measured a significant decrease in the distance between the tongue and the pharyngeal wall at the level of an extension of Go-B.

The study comparing the obstructive sleep apnoea and control patients with both upright and supine lateral cephalometric radiographs (Lowe et al, 1996) found no significant difference in this dimension measured from supine radiographs. They also considered subjects according to dental malocclusion and found no significant difference in this oropharyngeal airway measurement from upright or supine radiographs in subjects with a Class III dental malocclusion.

The oropharyngeal airway at the level of Go-B was not significantly different between obese and non-obese obstructive sleep apnoea subjects, however in both obstructive sleep apnoea groups there was a significant decrease compared with a nonapnoeic control (Sakakibara et al, 1999).

Johns et al (1998); Mayer and Meier-Ewert (1995); Mayer et al (1996); Mochizuki et al (1996); Pracharktam et al (1994) and Solow et al (1996) found no significant differences in the oropharyngeal dimensions of obstructive sleep apnoea subjects and control subjects along an extension of line Go-B.

The oropharyngeal airway space was decreased in obstructive sleep apnoea subjects at the level of the mandibular plane in two studies (Hochban and Brandenburg, 1994; and Tangugsorn et al, 1995b). Zucconi et al (1992) did not find a significant difference for this measurement.

The area of the oropharynx has been measured and found to be significantly smaller in some groups of obstructive sleep apnoea patients. Lowe et al (1996) found obstructive sleep apnoea patients with a Class I or Class II dental malocclusion had a significantly decreased oropharyngeal area compared with controls only on radiographs taken in the upright position. Interestingly there was no difference in this dimension for supine radiographs, or for patients with a Class III dental malocclusion in either position. Tangugsorn et al (1992b) also found this area significantly decreased in obstructive sleep apnoea patients upright lateral cephalometric radiographs.

In contrast to these findings, Ono et al (1996) did not find a significant decrease in this area between obstructive sleep apnoea subjects and nonapnoeic control subjects.

Table 6.6-1 records the authors, the linear and angular measurements of the oropharyngeal airway. Definitions of the landmarks used may be found in the glossary. Figure 6.6-1 shows diagrammatically the oropharyngeal airway measurements on a lateral cephalometric radiograph.

**Table 6.6-1: Oropharyngeal Airway Measurements**

| | | | PAS (OP) | PAS(Go-B) | PAS (Go-Gn) | Area |
|---|---|---|---|---|---|---|
| Battagel et al (1991) | A vs C | U | | 0.036[1] | | |
| Hochban et al (1994) | A vs C | U | 0.002 | | 0.005 | |
| Johns et al (1998) | A vs S | U | | NS | | |
| Lowe et al (1996) I | A vs C | U | | 0.001 | | 0.000 |
| Lowe et al (1996) I | A vs C | S | | NS | | NS |
| Lowe et al (1996) II | A vs C | U | | 0.015 | | 0.007 |
| Lowe et al (1996) II | A vs C | S | | NS | | NS |
| Lowe et al (1996) III | A vs C | U | | NS | | NS |
| Lowe et al (1996) III | A vs C | S | | NS | | NS |
| Lyberg et al (1989b) | A vs C | U | | 0.051 | | NS |
| Maltais et al (1991) | A vs C | U | | 0.051 | | |
| Mayer et al (1995) | A vs C | U | | NS | | |
| Mayer et al (1996) | A vs S | U | | NS | | |
| Mochizuki et al (1996) | A vs S | U | | NS | | |
| Ono et al (1996) | A vs C | U | | | | NS |
| Pracharktam et al (1994) | A vs S | U | | NS | | |
| Sakakibara et al (1999) | A1 vs C | U | | 0.0011 | | |
| Sakakibara et al (1999) | A2 vs C | U | | 0.001[1] | | |
| Sakakibara et al (1999) | A1 vs A2 | U | | NS | | |
| Solow et al (1996) | A vs C | U | | NS[1] | | |
| Tangugsorn et al (1995b) | A vs C | U | | | 0.001 | 0.01 |
| Zucconi et al (1992) | A vs S | U | | | NS | |

A = obstructive sleep apnoea subject; C = non-snoring, non-apnoeic subject; S = snoring, non-apnoeic subject; A1 = non-obese obstructive sleep apnoea subject; A2 = Obese obstructive sleep apnoea subject.

1 The narrowest distance between the tongue and the posterior oropharyngeal wall.

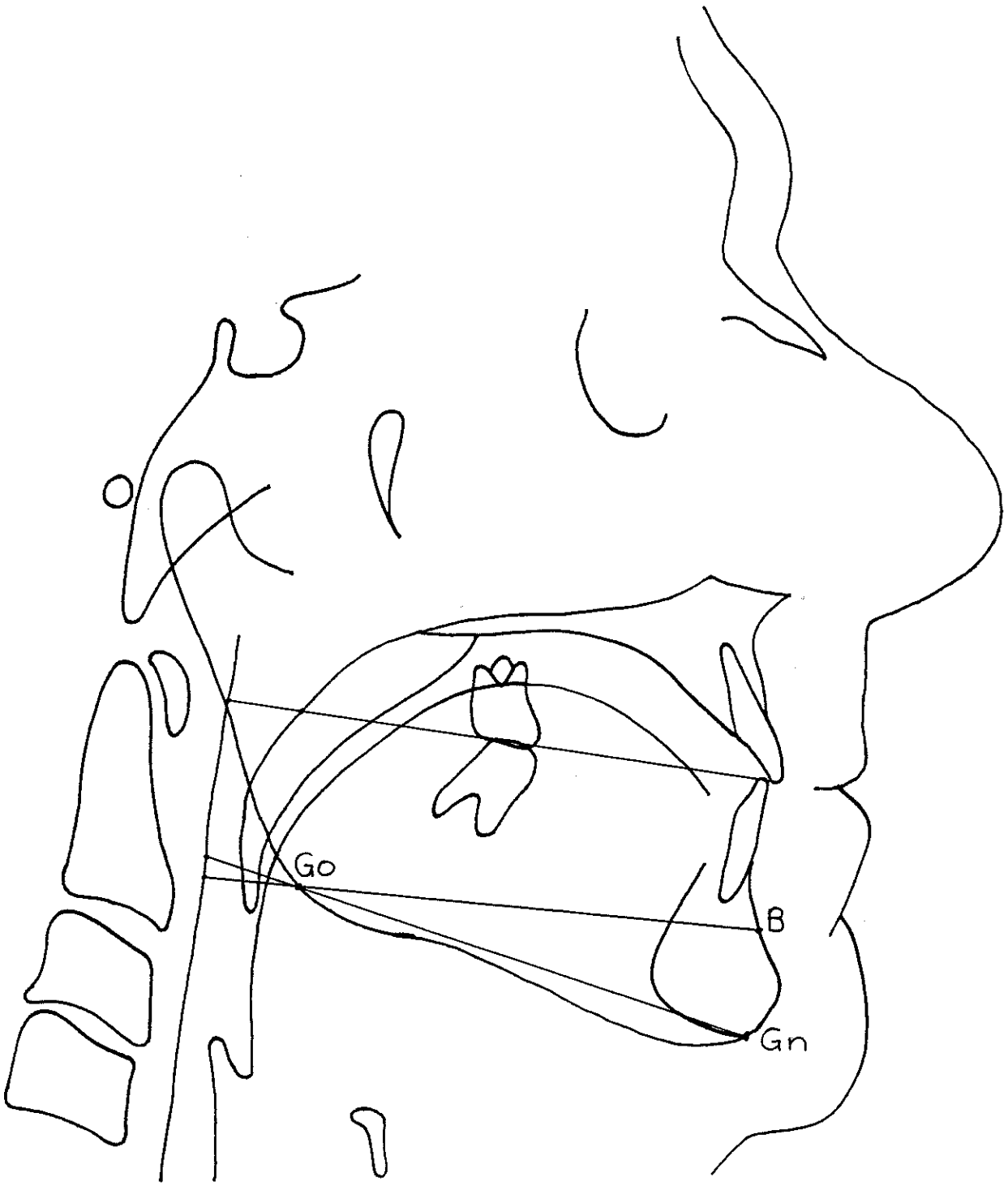


**Figure 6.6-1 Oropharyngeal Airway cephalometric measures.**

## 6.7 Hypopharyngeal Airway

The hypopharyngeal airway was defined by Lowe et al (1996) as being the area outlined by the inferior border of the oropharynx, posterior surface of epiglottis, a line parallel to palatal plane through point C4 and the posterior pharyngeal wall.

When comparing the sagittal dimension of the hypopharyngeal airway between obstructive sleep apnoeic subjects and controls, the differences are consistently less pronounced than for other segments of the upper airway. Sakakibara et al (1999) and Strelzow et al (1988) both measured a significant decrease in the hypopharyngeal airway at the level of the epiglottic tip. This was true for non-obese obstructive sleep apnoea subjects only, with no difference between obese obstructive sleep apnoea subjects and control or between obese and non-obese obstructive sleep apnoea subjects. Most other studies have found no significant difference in this measurement (deBerry-Borowiecki et al, 1988; Mochizuki et al, 1996 and Strelzow et al, 1988) or did not consider the hypopharyngeal airway at this level.

Measurements of the hypopharynx from the tongue base (vallecula) to the posterior pharyngeal wall have also yielded conflicting results. deBerry-Borowiecki et al (1988); Lowe et al (1996) and Strelzow et al (1988) all found a significant decrease in sagittal airway dimension at this level in obstructive sleep apnoea subjects. Lowe et al (1996) did not find a significant difference in obstructive sleep apnoea subjects with a Class II dental malocclusion in the supine position, or subjects with a Class III dental malocclusion in the upright position. All other groups of obstructive sleep apnoea patients had a significantly decreased hypopharyngeal airway at this level. Other authors did not measure a significant difference (Hochban and Brandenburg, 1994; Solow et al, 1996; and Tangugsorn et al, 1995b) in obstructive sleep apnoea subjects.

Many investigators have not considered the area of the hypopharynx when measuring lateral cephalometric radiographs. deBerry-Borowiecki et al (1988) did not separate the oropharyngeal and hypopharyngeal areas. They found a significant decrease in this measure of the airway in obstructive sleep apnoea subjects. Lowe et al (1996) found only obstructive sleep apnoea subjects with a Class II dental malocclusion and having a lateral cephalometric radiograph in the supine position had a significantly smaller hypopharyngeal area.

Table 6.7-1 records the authors, the linear and angular measurements of the hypopharyngeal airway. Definitions of the landmarks used may be found in the glossary. Figure 6.7-1 shows diagrammatically the cephalometric measurements of the hypopharyngeal airway.

**Table 6.7-1: Hypopharyngeal Airway Measurements**

| | | | ET-PhW | TB-PhW | V-PhW | Area |
|---|---|---|---|---|---|---|
| deBerry-Borowiecki et al (1988) | A vs C | U | NS | 0.038 | | 0.003[1] |
| Hochban et al (1994) | A vs C | U | | | NS | |
| Lowe et al (1996) I | A vs C | U | | | 0.001 | NS |
| Lowe et al (1996) I | A vs C | S | | | 0.020 | NS |
| Lowe et al (1996) II | A vs C | U | | | 0.000 | 0.03[1] |
| Lowe et al (1996) II | A vs C | S | | | NS | NS |
| Lowe et al (1996) III | A vs C | U | | | NS | NS |
| Lowe et al (1996) III | A vs C | S | | | 0.046 | NS |
| Mochizuki et al (1996) | A vs S | U | NS[2] | | | |
| Sakakibara et al (1999) | A1 vs C | U | 0.001 | | | |
| Sakakibara et al (1999) | A2 vs C | U | NS | | | |
| Sakakibara et al (1999) | A1 vs A2 | U | NS | | | |
| Solow et al (1996) | A vs C | U | | | NS | |
| Strelzow et al (1988) | A vs C | U | 0.05[3] | | 0.05 | |
| Strelzow et al (1988) | A vs C | U | NS | | | |
| Tangugsorn et al (1995b) | A vs C | U | | | NS | |

A = obstructive sleep apnoea subject; C = non-snoring, non-apnoeic subject; S = snoring, non-apnoeic subject; A1 = non-obese obstructive sleep apnoea subject; A2 = Obese obstructive sleep apnoea subject.

1 Combined area of the oropharynx and the hypopharynx; 2 Linear distance between the posterior tongue base and the pharyngeal wall passing through the tip of the epiglottis; 3 Linear distance from the epiglottis apex to the pharyngeal wall.

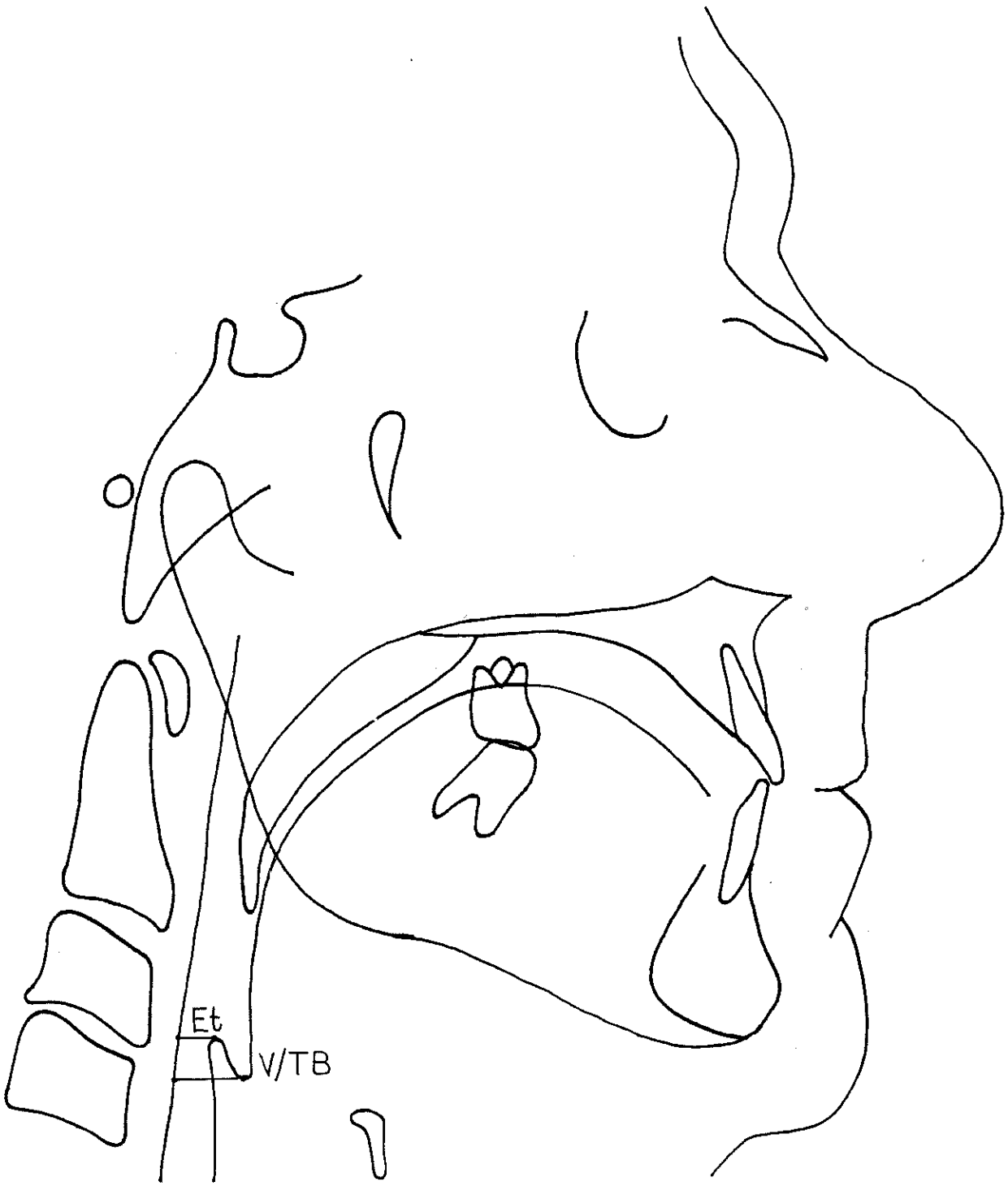


**Figure 6.7-1 Hypopharyngeal Airway cephalometric measures.**

# Chapter 7

# Non-Surgical Management of Obstructive Sleep Apnoea

## 7.1 Introduction

The management of obstructive sleep apnoea syndrome has been principally non-surgical since the initial reports of the successful use of nasal continuous positive airway pressure ventilation by Sullivan et al (1981). The decision on when to treat a patient presenting with snoring and evidence of upper airway obstruction is complicated by the differing opinions on what constitutes a diagnosis of obstructive sleep apnoea and on whether such a diagnosis is of clinical significance for the person. For example, it seems the greatest risk of complications associated with obstructive sleep apnoea are in people aged less than fifty years of age (Benaim et al, 1992). Does this mean people aged greater than fifty years who show evidence of obstructive sleep apnoea should be aggressively treated, or can more latitude be given to this group? In Chapter 2 the literature regarding the medical complications associated with obstructive sleep apnoea was reviewed, and many investigators have found an increase in mortality with an AHI>20 events per hour. This parameter would therefore seem a reasonable line of demarcation for treatment.

The literature also shows that people who experience less frequent episodes of airway obstructive sleep apnoea may still be afflicted with excessive daytime sleepiness or snoring that is troublesome to sleeping partners. Should these factors also be considered when determining who should be treated? There is no clear answer from the literature, however there are several suggested protocols for determining who should be treated for upper airway obstruction during sleep.

Powell and Riley have published extensively on the diagnosis and management of obstructive sleep apnoea. They have suggested a protocol to identify those people with obstructive sleep apnoea who may benefit from treatment, whether surgical or non-surgical Powell and Riley (1993). They include one subjective measure and four objective measures derived from polysomnographic study in their criteria, which are:

1. Excessive daytime sleepiness;
2. AHI > 20 or pathologic excessive daytime sleepiness with an AHI < 20;
3. Oxygen desaturation < 90%;
4. Arrhythmia's associated with obstructive events; or
5. Negative oesophageal pressures associated with sleep fragmentation.

Patients with obstructive sleep apnoea may present with one or more of these criteria and the authors recommend treatment if one of the five criteria is met. Treatment of obstructive sleep apnoea may be non-surgical or surgical. In this chapter the non-surgical management

of obstructive sleep apnoea will be considered, whilst Chapter 8 will review the surgical treatment of this condition.

Non-surgical management of obstructive sleep apnoea may involve weight loss (Browman et al, 1984; Harman et al, 1982; and Smith et al, 1985), pharmacotherapy, physical repositioning of the skeletal or soft tissues of the UAW without surgery, head and neck extension collars and sleep position modification (Cartwright, 1984; and Cartwright et al, 1985).

The "gold standard" of treatment, surgical or non-surgical, of obstructive sleep apnoea has been nCPAP since its description by Sullivan et al (1981).

Movement of the tissues of the UAW without surgery has been achieved by a number of intraoral appliances worn by the patient whilst sleeping. These appliances either advance the mandible, thus advancing the soft tissues attached to the mandible and increasing posterior airway space, or by advancement of the tongue with similar effect on the oropharyngeal airway.

## 7.2 Weight Loss

Obesity is known to be a physical characteristic of many patients suffering from obstructive sleep apnoea, and there is an increased incidence of obstructive sleep apnoea in patients who are obese compared with the normal population (Young et al, 1993). Weight loss is effective in reducing the AHI, the extent of arterial oxygen desaturation and the amount of sleep disruption seen in patients with obstructive sleep apnoea.

Smith et al (1985) reported on 6 obese patients who lost an average 20 kg of weight. All had a lower AHI, less oxygen desaturations and less hypersomnolence than control patients whose weight remained unchanged for the period of the study.

Harman et al (1982) reported on four patients who underwent jejeuno-ileal bypass. The patients had weight loss from an average 231 kg preoperatively to 123 kg postoperatively. All patients suffered obstructive sleep apnoea with preoperative AHI ranging from 15 to 196 events/hour. Postoperatively the AHI fell to 0.20 to 0.98 events/hour. Browman et al (1984) reported upon one patient with multiple sleep studies over a three year period and considerable variation in weight. The patient showed a disproportionate decrease in his AHI compared with weight loss. At a weight of 111kg the patient had an AHI of 59.6 events/hour, with a decrease to 85 kg the AHI fell to 3.1 events/hour.

Partinen et al (1988) conducted a retrospective study of 198 patients diagnosed with obstructive sleep apnoea syndrome who were treated by tracheostomy (71 patients) or weight loss (127 patients). At a minimum five year follow up 14 patients had died. All these

patients were treated conservatively by weight loss. This group of patients had a lower mean apnoea index (43 events/hour versus 69 events/hour) and lower mean BMI (31 $kg/m^2$ versus 34 $kg/m^2$) than the group treated by tracheostomy.

Weight loss has been suggested for patients suffering a variety of medical conditions. Foreyt and Goodrick (1993) have demonstrated that patients who lose weight are unlikely to maintain their new weight and most return to their previous condition despite the best intentions. Patients studied over a three to five year period showed an average duration of behavioural change of 18 weeks, with an average weight loss of 0.5 kg per week. 33% of patients had returned to their baseline weight at 52 weeks and almost all patients showed a gradual return to baseline weight over the 3 – 5 year follow up.

Alteration of sleep position has also been shown to influence the level of AHI experienced by some patients with obstructive sleep apnoea. Both snoring and obstructive sleep apnoea appear to be more severe with patients in the supine position. Cartwright (1984) reported that 24 unselected obstructive sleep apnoea patients had a 100% increase in their AHI when sleeping supine as compared to the lateral cubitus position. He found sleep position modification to be adequate treatment in some of this group. This treatment is found to be most effective for patients who are close to their ideal weight and is rarely sufficient treatment of obstructive sleep apnoea as a single modality.

## 7.3 Pharmacology

A number of pharmacological agents have been utilized to increase ventilatory drive or stimulate UAW muscles. Many of these medications have unwanted side effects and this results in non-compliance by patients.

Pharmacological treatment of obstructive sleep apnoea relies upon increased muscle neurological activity during sleep or decreasing the period of time spent in REM sleep. The most commonly used agent is the tricyclic antidepressant, Protriptyline (Bonora et al, 1985; and Brownell et al, 1982). These medications act to decrease the number of oxygen desaturations during nREM sleep, suppresses REM sleep and specifically increases the tone of UAW muscles. Side effects of this medication are anticholinergic in nature i.e. xerostomia, urinary retention, constipation and impotence.

The requirement for life-long medication and the relative ineffectiveness of agents used in the control of obstructive sleep apnoea has meant they are not first line choices of treatment.

## 7.4 Nasal Continuous Positive Airway Pressure

The first report of the use of nCPAP for treatment of obstructive sleep apnoea was by Sullivan et al (1981). This report of the abolition of obstructive sleep apnoea by continuous airway pressure applied through the nares on five patients with obstructive sleep apnoea revolutionized the treatment of obstructive sleep apnoea. Since this pioneering report the nCPAP has been the gold standard for management of obstructive sleep apnoea against which all other modalities must be compared. See Figure 7.4-1 for an example of a patient utilising nCPAP during a polysomnographic sleep study.

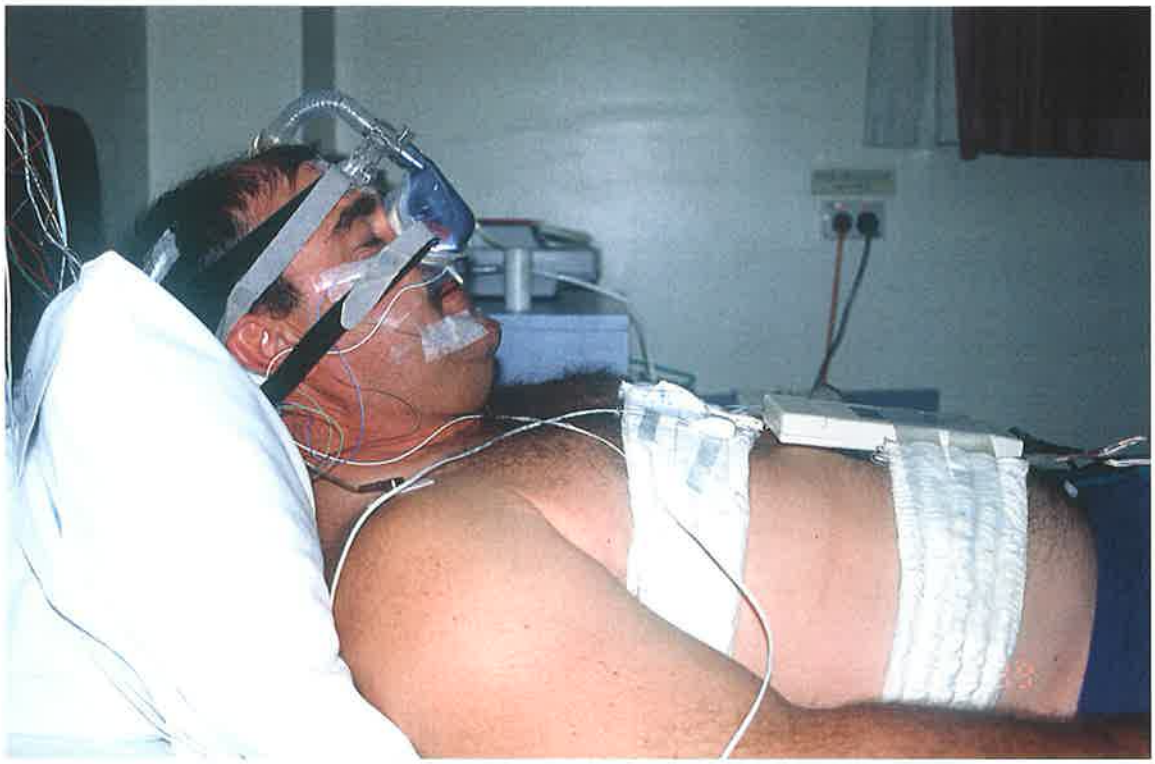

**Figure 7.4-1Titration of nCPAP in the sleep laboratory**

Sullivan et al (1981) hypothesized the effectiveness of nCPAP lies in its ability to splint the airway open. This is achieved by blowing air into the UAW through a mask covering the nares. The pressure needed to maintain patency varies between patients and must be titrated, usually during an overnight sleep study.

Not all patients require the same level of pressure applied by nCPAP to abolish apnoeic episodes. The pressure required is <10cm H2O as reduced cardiac output and renal function have been reported at pressures greater than this (Sullivan et al.,1981).

AHI and neck circumference was found to be the strongest variables in predicting the intensity of nCPAP required in a study by Miljeteig and Hoffstein (1993) involving 208 patients where BMI, neck circumference and waist circumference were measured in patients confirmed as suffering obstructive sleep apnoea by polysomnography.

nCPAP has been postulated to work via a number of different mechanisms:

1. Pneumatic splinting of the UAW (Rapaport et al, 1983; and Sullivan et al, 1981);
2. Nasal stimulation resulting in reflex UAW muscle activation (Kaufman and Wright, 1969; and Rapaport et al, 1983);
3. Increased functional residual capacity increasing pharyngeal patency (Hoffstein et al, 1984); or
4. Increased systemic oxygenation leading to decreased hypoxic cerebral depression and respiratory drive instability (Santiago et al, 1984).

Unfortunately up to 54% of patients do not comply with nCPAP therapy because of physical or psychological reasons (Kribbs et al, 1993). In these patients it is necessary to treat their obstructive sleep apnoea using a different method.

Patients may by intolerant to nCPAP for a number of reasons, usually related to the mask or airflow pressure generated. Strollo et al (1995) in their review noted that side effects of nCPAP may be either mask or airflow related.

Complaints regarding the mask may be related to poor fit and resultant leak or to a feeling of claustrophobia experienced by some patients. Ill fitting masks may cause skin abrasion or rash and conjunctivitis from an air leak. It may by possible to manufacture the mask to fit better, and a variety of different masks are available. In extreme cases a custom fit mask may need to be manufactured for the patient.

A patient who experiences claustrophobia whilst wearing the mask presents a difficult problem to overcome. Harris et al (1990) used a nasal pillow to replace the nasal mask.

They demonstrated that no significant difference in the pressure requirements between nasal masks and nasal pillows provided a good seal is achieved.

Pressure or airflow related problems might be related to a feeling of excess pressure intranasally preventing the patient from falling asleep or a smothering sensation. In this case modification of the nCPAP machine by including a gradual increase in pressure, usually over 15 – 30 minutes allow the patient to fall asleep prior to development of maximal pressure entering the nares via the mask. At this stage there are no published figures validating the assumption that this feature increases compliance with nCPAP.

Airflow related complications include rhinorrhoea, nasal congestion or dryness, chest discomfort, aerophagia or sinus discomfort. Bilevel positive airway pressure may be utilized in these patients. This system allows a differential pressure to be generated between inspiration and expiration. The result of this system is a net decrease in airway pressure and thus a decrease in resistance to respiration provided by the positive airway pressure machine.

When high flows are required to adequately treat patients obstructive episodes a complaint that the air is too cold may occur. Elevation of the machine may solve the problem, alternatively a heated humidifier may need to be incorporated in the circuit.

Machine noise is also cited as a reason for noncompliance with nCPAP. The bedpartner rather than the patient usually make this complaint. Attempts can be made to soundproof the machine, and certainly the nCPAP machines have evolved to become much less noisy and also less cumbersome. Yet other patients claim the machine is too cumbersome to transport and for some patients their compliance is thus affected.

## 7.5 Mandibular Repositioning Appliances

Pierre Robin in 1934 described a monoblock functional appliance that he used to pull the tongue and mandible forward in-patients with micrognathia. Extra-oral appliances have also been described to advance the mandible in infants with micrognathia and airway obstruction (Eley and Farber, 1930; and Longmire and Sanford, 1949). These developmental problems affecting the mandible resulted in narrowing of the upper airway and obstruction during respiration.

Many dental devices have been reported in the literature to be effective in decreasing the severity of obstructive sleep apnoea in patients. Unfortunately many of these studies do not use polysomnography to quantify the improvement in sleep apnoea. The few studies that do use polysomnography indicate dental devices that protrude the mandible and increase the

mouth opening may help some patients in the mild to moderate range of obstructive sleep apnoea.

The principle behind these devices is non-surgical advancement of the mandible and attached musculature (tongue and pharyngeal muscles). Proponents advocate these devices on the grounds they are non-invasive, have minimal side effects and patient compliance is comparable to nCPAP (Clark et al, 1996).

The removable appliances placed intraorally for the treatment of obstructive sleep apnoea may take one of three forms:

1. Tongue retaining devices;
2. Fixed mandibular advancement appliances (see Figure 7.5-1); and
3. Adjustable mandibular advancement appliances (see Figure 7.5-2).

The tongue retaining device uses suction in order to advance the tongue and prevent its relapse into the oropharynx and/or hypopharynx during sleep. Cartwright and Samelson (1982) reported these devices to be effective, however only a small number of patients were included in their study. They also found these devices often fail because of loss of suction whilst the patient is sleeping.

A large number of appliances are marketed to advance the mandible. The more basic designs involve a fixed upper and lower acrylic or polymer appliance advancing the mandible a proscribed distance that is constructed at a fixed protrusive measurement. A variation of these appliances allows adjustment of the amount of mandibular protrusion, thus allowing easy adjustment of the amount of mandibular protrusion by the physician in order to optimize the degree of advancement for individual patients. This is advantageous as there is a decreased need for appliance remake, and allows easy adjustment if the patient returns complaining of temporomandibular joint symptoms.

Thornton and Roberts (1996) reported the advantages of mandibular repositioning appliances to be an increase in posterior airway space; stabilization of the mandible in an anterior and closed position; tongue advancement; an increase in genioglossal muscle activity. They also cited the benefits that these devices are simple to fabricate, have no known permanent sequelae for the patient following discontinuation of use and are cost effective compared to other methods of treating obstructive sleep apnoea.

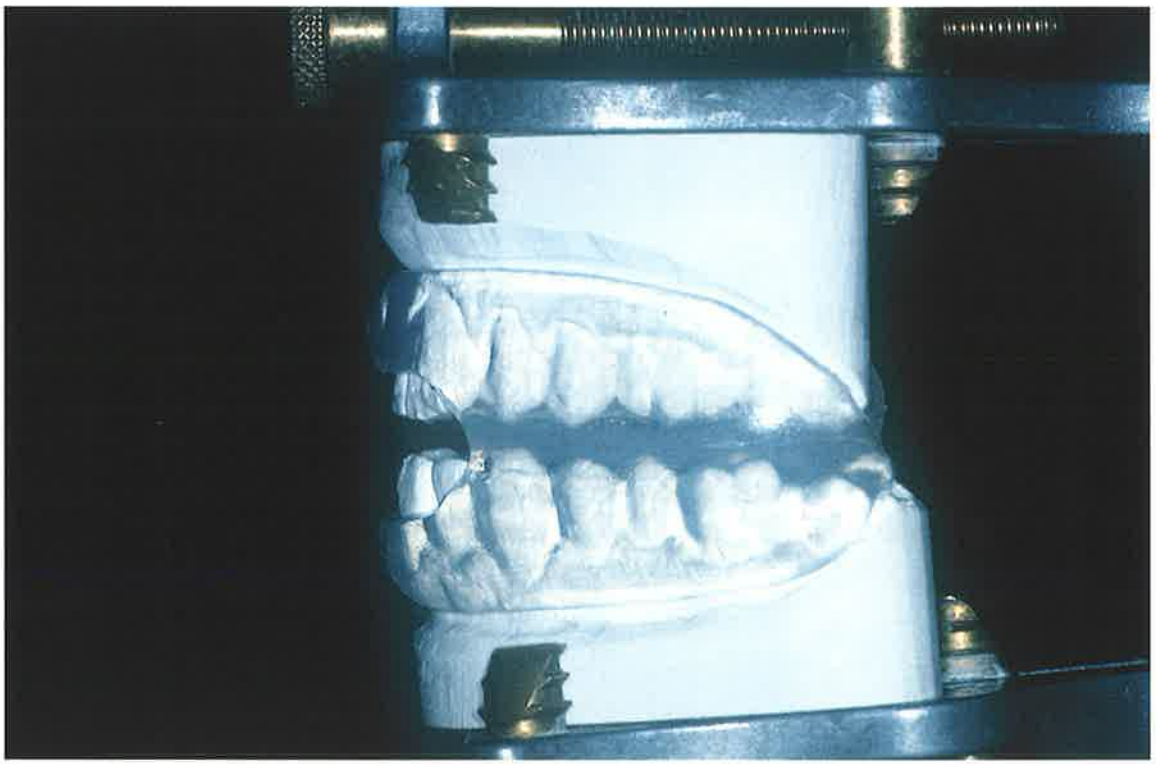

**Figure 7.5-1 Non-adjustable Mandibular Repositioning Appliance**

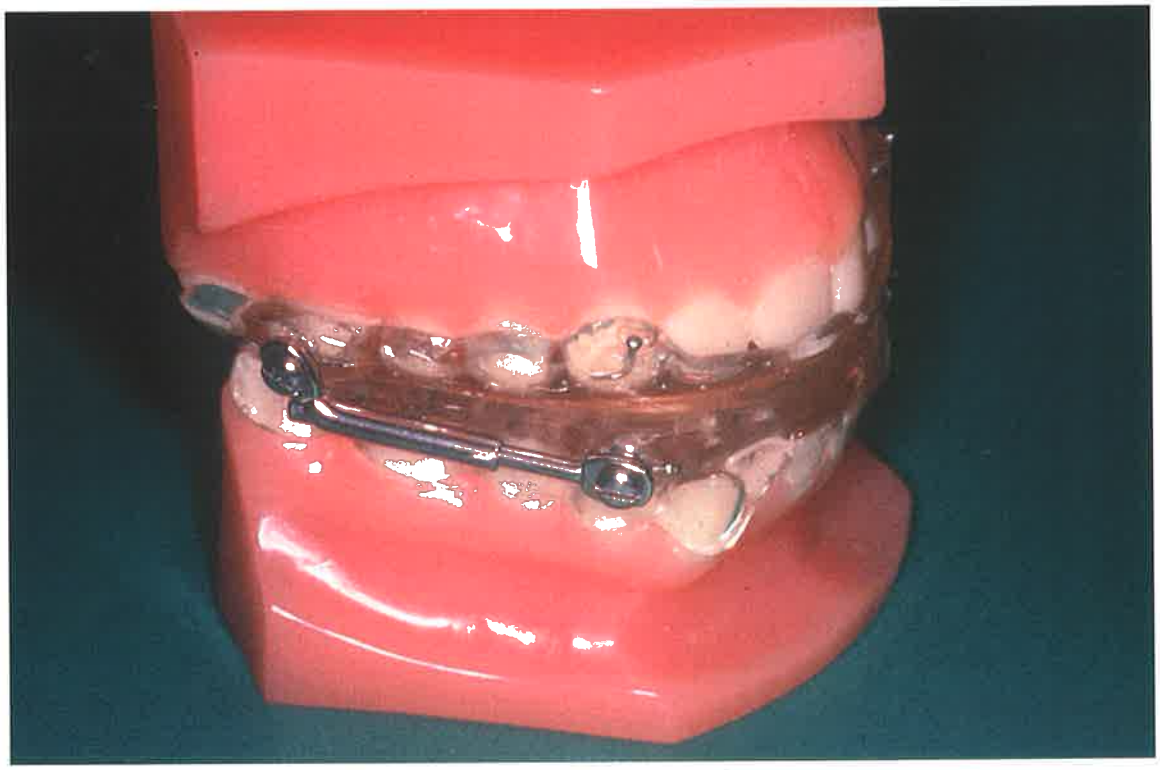

**Figure 7.5-2 Adjustable Mandibular Repositioning Appliance**

Meurice et al (1996) reported an increase in upper airway collapsibility when the mouth was held open 15mm by a plastic appliance but no decrease in posterior airway space. This study was performed on six non-obese patients who did not suffer from obstructive sleep apnoea and who consumed no medication or alcohol prior to polysomnographic study. The expected change in cephalometric parameters was only significant for a decreased MP-H ($p<0.001$) and a significant increase in ANS - Gn ($p<0.01$). The authors concluded mouth opening might move the mandible posteriorly leading to a decrease in the size of the oropharyngeal lumen and predisposing it to obstruction. They further suggested this posterior rotation of the mandible decreases the length of the suprahyoid muscles and therefore decreases their contractile efficiency.

The possible clinical significance of this finding is the knowledge that treatment of obstructive sleep apnoea by an mandibular repositioning appliance invariably involves increasing the patients interincisal distance whilst the appliance is in situ. If the mandible is not protruded adequately there may be a tendency to exacerbate the obstructive events that the appliance is designed to prevent. This study is small and a larger sample size would lend more credibility to the results however until disproved it should be taken into consideration when treating patients with a mandibular repositioning appliance for obstructive sleep apnoea or even simple snoring.

A recent trend has been for general dentists to construct mandibular advancement appliances of varying design in order to "treat" snoring and or obstructive sleep apnoea of their patients, often at a fraction of the cost and time involved in other treatment modalities. Loube and Strauss (1997) published the results of a questionnaire survey of 355 dentists, all of whom belong to the Sleep Disorders Dental Society. This association is a group of dentists, predominately North American who aims to treat obstructive sleep apnoea patients by use of mandibular repositioning appliances. Only 124 of the questionnaires were returned (35%). 95% of respondents used pretreatment polysomnography, but disturbingly only 18% of members routinely used post-treatment polysomnography. Incredibly, despite voluminous evidence in the literature to the contrary, 7% of these dentists believe subjective reports are an adequate substitute for post treatment polysomnography and 37% believe nocturnal pulse oximetry, pre or post treatment is an adequate substitute for polysomnography. Nocturnal pulse oximetry has a sensitivity of 90 – 98% but a specificity of only 48 – 75% for obstructive sleep apnoea, considerably less than that for polysomnography (Levy et al, 1996; Series et al, 1993; and Yamashiro and Kryger, 1995).

### 7.5.1 Protocol for Dental Appliance Therapy

The following is a suggested protocol for dental appliance therapy proposed by the Sleep Disorders Dental Society:

1. Medical assessment;
2. Polysomnogram;
3. Referral to dentist;
4. Dental examination;
5. Trial appliance (3 – 7/7);
6. Final appliance;
7. Adjustment and evaluation (2-3/12);
8. Repeat polysomnogram;
9. Dental appliance modification, remake as required;
10. Repeat adjustment and evaluation;
11. Physician for ongoing evaluation;

Follow-up as required.

Kloss et al (1986) first published the use of mandibular advancement appliances specifically for the management of obstructive sleep apnoea. A mandibular repositioning appliance should be used during sleep for life, be comfortable and preferably involve full occlusal coverage in order to prevent vertical changes to the dentition over time.

Studies of mandibular repositioning appliance and their effect on obstructive sleep apnoea are often marred by the failure to adequately assess the severity of the obstructive sleep apnoea by use of polysomnography. Clark et al (1993) studied 24 patients with obstructive sleep apnoea and the effect of an mandibular repositioning appliance with mandibular advancement 75% of the maximum possible for the patients. All patients had a polysomnographic sleep study preappliance however only 15 patients underwent a repeat study with the appliance in place. Reasons given for failure to obtain the second sleep study included a subjective assessment by the patient that their symptoms had not changed, inability of the patient to tolerate the mandibular repositioning appliance or inability of the patient to afford a second sleep study.

Results of this study showed a decrease in respiratory distress index (RDI, an event based on a combination of a 10 second airflow cessation and a 3% oxygen saturation level decrease)

from a mean of 48.4 +/- 33.4 events/hr pretreatment to 12.3 +/- 20.6 events/hr post-treatment. This represents 14/24 (58%) patients with a reduced RDI with the mandibular repositioning appliance in position. Importantly 13/15 patients with a reduced RDI fell below the threshold of 20 events/hr that is associated with increase risk of mortality. The authors had a reasonable length of follow-up on a large number of their patients and 12/23 patients were still regularly using the appliance at 36 months or greater post insertion.

### 7.5.2 Comparison of Dental Appliances and nCPAP

Clark et al (1996) performed a cross-over study where patients used nCPAP for 2/52 and an mandibular repositioning appliance for 2/52. Each of the 21 subjects had pre- and post-treatment polysomnographic sleep studies. The advancement provided by the mandibular repositioning appliance was 65% of maximum. The results of the study are presented in Table 7.5-1. The authors found that in severe cases of obstructive sleep apnoea the mandibular repositioning appliance was less effective at lowering the AHI to acceptable levels when compared with nCPAP.

**Table 7.5-1: Comparison of nCPAP and mandibular repositioning appliance (Clark et al, 1996)**

| | NCPAP (events/hour) | Mandibular Repositioning Appliance (events/hour) |
|---|---|---|
| Pretreatment AHI | 33.86 +/- 14.30 | 33.86 +/- 14.30 |
| Post treatment AHI | 11.15 +/- 3.93 | 19.94 +/- 12.75 |

A similar study by Ferguson et al (1996) assessed the use of nCPAP and a mandibular repositioning appliance on 27 patients who suffered obstructive sleep apnoea. The mandibular repositioning appliance advanced the mandible 3mm less than maximal protrusion and produced a maximal incisal opening of 7mm. Successful treatment of obstructive sleep apnoea in this study was defined as an AHI<10 and resolution of symptoms. This study used patients suffering mild to moderate obstructive sleep apnoea (AHI range 15 – 50 pre treatment).

The results of this study showed both treatments were successful at treating obstructive sleep apnoea (see Table 7.5-2), however nCPAP produced a lower AHI in more patients (mandibular repositioning appliance 19/25 AHI 9.7 +/- 7.3 events hr c.f. nCPAP 20/25 AHI 3.5 +/-1.6 events/hr). Both treatment modalities significantly decreased AHI at the 5% level. 6/25 patients were non-compliant with the mandibular repositioning appliance c.f. 4/25 with nCPAP. The level of oxygen desaturation (SaO2) was significantly improved with nCPAP

but not with the mandibular repositioning appliance ($p<0.005$). Interestingly side effects were more common and the patients less satisfied with nCPAP ($p<0.005$) despite the fact is was less efficacious at reducing the AHI and also at decreasing daytime sleepiness ($p<0.005$). 12 patients were successfully treated by mandibular repositioning appliance according to the criteria of this study and 11 of these patients chose to continue its use long term in preference to nCPAP.

**Table 7.5-2: Comparison of nCPAP and mandibular repositioning appliance (Ferguson et al, 1996)**

| | nCPAP (events/hour) | Mandibular Repositioning Appliance (events/hour) |
|---|---|---|
| Pretreatment AHI | 17.5 +/- 13.2 | 19.7 +/- 13.8 |
| Post treatment AHI | 3.6 +/- 1.7 | 9.7 +/- 7.3 |

### 7.5.3 Side Effects of Dental Appliances

Side effects of mandibular repositioning appliance use have been reported by a number of authors, and many of these side effects are similar to those reported for nCPAP. O'Sullivan et al (1995) reported the treatment of obstructive sleep apnoea by way of an mandibular repositioning appliance in 37 patients with obstructive sleep apnoea that advanced the mandible 75% of maximal protrusion and caused maximal incisal opening of 10mm. Unfortunately this study included both simple snorers and obstructive sleep apnoea patients and the data was not reported separately for each group. 14/26 patients with an AHI > 20 on pre treatment polysomnography had a post treatment AHI < 20 at subsequent polysomnography. This was significant in the authors' opinion, as all these subjects were unable or unwilling to tolerate nCPAP for treatment of their obstructive sleep apnoea. The adverse side effects reported in this study from the use of an MRA is shown in Table 7.5-3.

**Table 7.5-3: Side effects of mandibular repositioning appliances reported by snoring and obstructive sleep apnoeic subjects (O'Sullivan et al, 1995)**

| Side Effect | Number of subjects affected |
|---|---|
| Jaw discomfort on waking | 38/57 (67%) |
| Jaw discomfort on waking lasting longer than three weeks | 16/57 (28%) |
| Excessive salivation | 11/57 (19%) |
| Dry mouth | 12/57 (21%) |
| Gingival irritation | 4/57 (7%) |
| Bruxism | 3/57 (5%) |

Schmidt-Nowara et al (1991) provided further evidence as to the effectiveness of mandibular repositioning appliance in treating mild to moderate obstructive sleep apnoea. Their study consisted of 20 people (19 males and 1 female) with an average AHI pre treatment of 47.4 and an average AHI post treatment of 19.7. Only 7/20 patients achieved an AHI<20 i.e. effective treatment of their obstructive sleep apnoea. In this study the mandibular repositioning appliance was constructed with an average maximal incisal opening of 7.2 +/- 2 mm and an edge to edge incisal relationship.

The above study also constructed mandibular repositioning appliance for snoring patients, totaling 71 patients with an mandibular repositioning appliance for treatment of snoring or obstructive sleep apnoea. They report 75% of patients (51/71) were still using the mandibular repositioning appliance at an average 7 months post insertion (range 2 – 25 months). There were 25 % of patients (17/71) no longer wearing the mandibular repositioning appliance, and only one cited TMJ discomfort as the reason.

Compliance is a major problem in any non surgical treatment modality for obstructive sleep apnoea, whether it be nCPAP, mandibular repositioning appliance or weight loss. Long term compliance for the use of a mandibular repositioning appliance has been reported between 48% (Nadazawa et al, 1992) and 52% (Clark et al, 1993) at three years.

Eveloff et al (1994) in a small study of 19 patients challenged the widely held assumption that mandibular repositioning appliance gained some of their therapeutic effect by increasing the posterior airway space. In 19 patients studied there was no significant difference in posterior airway space on lateral cephalometric radiographs taken with or without the appliance. 10/19 patients were reported as responders, defined by the authors as an AHI<10 on polysomnographic sleep study with the appliance in place. All patients in this study were mild to moderate sufferers of obstructive sleep apnoea with the average AHI falling from 34.7 +/-5.3 to 12.9+/-2.4 across the 19 subjects. There was a significant decrease in the MP-H in all patients studied. The length of the soft palate (PNS-P) was not significantly different between responders and nonresponders on the baseline cephalometric radiograph, however this measurement was significantly shorter in patients who responded to the appliance when the lateral cephalometric radiograph was repeated. This change in soft palate length has also been reported by Bonham et al (1988) who used a modified orthodontic functional appliance to advance the mandible in patients with obstructive sleep apnoea syndrome.

This study raises the interesting possibility that advancement of the mandible may impact upon the soft palate such that the upper airway space is increased. Advancement of the mandible could be expected to result in advancement of those soft tissues directly attached to the mandible. The soft palate and uvula, consisting of tensor veli palatini and levator veli

palatini has no direct attachments to the mandible. The authors do not comment upon the mechanism for this change seen in soft palate dimensions. Further study should be done to determine if mandibular advancement by surgery results in the same change in palatal dimension, or if the measured alteration in soft palate length on lateral cephalometric radiography is dependent upon the presence of an intraoral mandibular repositioning appliance. If this change in soft palate length is seen only with a mandibular repositioning appliance what is the mechanism of its action upon the soft palate musculature. Is there increased resting tone in these muscles or is there a vector of force placed upon the attachments of these muscles such that they shorten? Only further research into this effect may provide answers.

# Chapter 8

# Surgical Management of Obstructive Sleep Apnoea

## 8.1 Introduction

A number of surgical procedures have been advocated in the literature for the treatment of obstructive sleep apnoea, either as primary therapy or as an adjunct to other treatment. Tracheostomy was the method of treatment, as reported by Kuhlo et al (1969) prior to the advent of nCPAP by Sullivan et al (1981). This obviously is successful in all patients suffering obstructive sleep apnoea, however there is significant morbidity associated with this therapy and it is little used as definitive treatment at this time unless the patients life is in immediate danger.

Surgical treatment is aimed at either reducing the soft tissue of the UAW or at altering the skeletal framework over which this soft tissue is draped. Many of the surgical therapies advocated for the treatment of obstructive sleep apnoea were originally used for other purposes, however with the increase in knowledge of obstructive sleep apnoea in the last few decades many of these procedures have taken on new clinical importance. Surgical treatment for obstructive sleep apnoea reported in the literature includes tracheostomy; nasal reconstruction; UPPP; mandibular osteotomy involving movement of the genial tubercles and hyoid; bimaxillary advancement; or base of tongue resection.

In assessing the obstructive sleep apnoea patient for possible surgical treatment it obviously obligates the surgeon to determine as accurately as possible the site of obstruction for each patient and hence tailor the surgery accordingly.

The surgical management of obstructive sleep apnoea is aimed at relieving the site of UAW obstruction by removing soft tissue or by altering the skeletal base upon which soft tissue is draped.

Surgical correction of aberrant UAW anatomy in this group of patients should be performed as there is a reasonable success reported in the literature of effective treatment of obstructive sleep apnoea when surgery of the affected area of the UAW is undertaken. This is not to say all patients suffering obstructive sleep apnoea are surgical candidates, and it might be argued surgery should be reserved only for those patients who fail to tolerate other treatments for their sleep disorder.

Consideration prior to surgery must be given to comorbidities and the potential difficulties associated with the perioperative management of these patients. Prior to surgery there must be optimization of these comorbidities in order to decrease potential complications. This may include weight loss, strict control of hypertension and the use of a CPAP machine in the days leading up to surgery.

The correction of underlying anatomic anomalies should be undertaken because this may prevent the patient from a life reliant on CPAP, or worse still non-compliance to CPAP and ineffective management of this potentially life-threatening condition.

## 8.2 Tracheostomy

Tracheostomy obviously required no information regarding the site of obstruction of the UAW as it simply bypassed the UAW completely. The first treatment for obstructive sleep apnoea syndrome was tracheostomy, and was described by Coccagna et al (1972). This was effective in 100% of cases of obstructive sleep apnoea however the morbidity associated with this procedure is high and alternative treatments were sought.

Conway et al (1981) performed tracheostomy on 11 patients for treatment of obstructive sleep apnoea found 9/11 had complete reversal of their symptoms within two days of surgery. One patient developed a post operative wound infection. Other post operative infectious problems included pneumonia and recurrent, purulent bronchitis. Haemoptysis and tracheal obstruction developed four to twelve months post tracheostomy in several patients. The most frequent morbidity associated tracheostomy was psychosocial. Preoperatively 8/11 patients reported psychosocial problems, with 10/11 patients reporting these problems postoperatively. Their symptoms included disability, depression, adjustment reactions, marital discord and alcohol or drug abuse or dependency.

A second study published in the same year reviewed fifty patients who had undergone tracheostomy for management of obstructive sleep apnoea (Guilleminault et al, 1981). They reported a number of complications, acute, subacute, local and general. They noted these complications were resolved such that there were no ongoing clinical symptoms and patients were able to return to normal social and family activity. As expected, temporary occlusion of the stoma caused recurrence of the obstructive sleep apnoea, thereby confirming tracheostomy to provide symptomatic relief only.

## 8.3 Nasal Surgery

Decreased nasal air entry has been postulated to contribute to the development of obstructive sleep apnoea (Olsen, 1991) and surgery can be directed at any aberrant anatomy in this area. Surgery to decrease the resistance to airflow through the nasal cavity has been undertaken in an effort to decrease the incidence and severity of more distal upper airway narrowing and occlusion during sleep. Dayal and Phillipson (1985) and Hester et al (1995) both reported small numbers of patients who underwent nasal surgery to increase airflow as management

for obstructive sleep apnoea. Neither study used polysomnographic measures in follow-up to assess the effectiveness of surgery, although both claim subjective improvement in patients and Hester et al (1995) report an improvement in arterial oxygen saturation measures in 12/15 (80%) of subjects.

As mentioned in Chapter 1 (Howard, 1971) likened the upper airway to a Starling resistor. Blakley and Mahowald (1987) postulated that if the nose acts as a Starling resistor then - increased nasal resistance will cause a greater degree of negative pharyngeal pressure. They tested this hypothesis by measuring the upper airway resistance on 37 subjects with no evidence of upper airway obstruction during sleep and 53 patients with polysomnographically proven obstructive sleep apnoea. This resistance was measured by determining the amount of negative pressure (i.e. pressure below normal atmospheric pressure) generated in the posterior nasopharynx. They found that the subjects with obstructive sleep apnoea did have increased nasal resistance compared to the control subjects, however there was no correlation between the severity of nasal obstruction and the severity of obstructive sleep apnoea as measured by AHI or oxygen desaturation. They concluded that nasal resistance may play a role in the pathogenesis of obstructive sleep apnoea but it was not the major contributing factor in upper airway narrowing or occlusion.

A similar study measuring combined nasal resistance and highest unilateral nasal resistance by Atkins et al (1994) found no significant difference in nasal airway resistance between a population of snoring subjects and those with obstructive sleep apnoea. There was also no correlation between the degree of nasal airway resistance and the AHI. Miljeteig et al (1992) also measured bilateral and unilateral nasal airway resistance on 683 subjects with obstructive sleep apnoea or simple snoring to determine its possible relationship with obstructive sleep apnoea. They too found no correlation between the degree of nasal resistance and the development of upper airway narrowing or occlusion during sleep.

Maxillary constriction in the lateral dimensions is known to increase nasal resistance to airflow and increase the incidence of mouth breathing. Expansion of the maxilla, either surgically or with orthodontic assistance is known to improve nasal airflow. Ten subjects diagnosed with mild obstructive sleep apnoea (AHI 19.4 +/- 4 and minimum $SaO_2$ 89 +/- 1%) and a constricted maxillary arch underwent rapid maxillary expansion. Four patients had surgical expansion of their maxilla whilst the other six patients underwent surgically assisted rapid maxillary expansion in conjunction with orthodontic treatment (Cistulli et al, 1998). One subject failed to improve, seven had their AHI < 5, and the remaining two had an AHI < 10. This study indicates that if there is evidence of reduced nasal airflow then surgery directed at the maxilla, rather than the nose per se may be of benefit if their is constriction of the maxillary arch.

Inflammation of the nasal mucosa has been postulated to contribute to increased nasal resistance to airflow. This pathology is not amenable to surgical correction but nasal decongestants may be of some benefit. Rubinstein (1995) examined the nasal lavage fluid for markers of inflammation in eight obstructive sleep apnoeic subjects and six control subjects. All subjects were non-smokers and had fluid removed in the evening and early the following morning. There was a significantly increased concentration of markers of inflammation (polymorphonuclear leucocytes, bradykinin and vasopressin) in the subjects with obstructive sleep apnoea. The author suggested nasal inflammation may play a role in nasal airflow limitation in obstructive sleep apnoeic subjects. This also suggests there may be a group of patients who do not have aberrant nasal anatomy contributing to restriction of nasal airflow. Surgery in all patients exhibiting signs of nasal airflow limitation contributing to their obstructive sleep apnoea is therefore not necessarily indicated.

## 8.4 Uvulopalatopharyngoplasty

Uvulopalatopharyngoplasty (UPPP) is commonly used in the treatment of simple snoring, the removal of redundant palatal mucosa and associated tonsillar pillars acts to decrease, and in some cases abolish snoring in some patients.

Ikematsu (1964) first described the surgical procedure, uvulopalatopharyngoplasty (UPPP), as a surgical treatment for snoring.

Fujita et al (1981) reported UPPP as a surgical procedure to enlarge the potential airway space in the oropharynx, thus alleviating the obstructive episodes in obstructive sleep apnoea patients. This report of a small series of 12 patients reported an average preoperative AHI of 54 events/hr and postoperative AHI 27.9 events per hour. 8/12 (66%) had an AHI < 20 post op and therefore were considered successfully treated, 2/12 (16%) had a decrease in AHI but the level remained above 20 events per hour and were therefor considered failures. Significantly 2/12 (16%) of patients had an increased AHI postoperatively, a most unfortunate outcome for the patients involved.

Larsson et al.,(1994) showed evidence that the effectiveness of UPPP in the management of obstructive sleep apnoea may decrease with time.

Katsantonis and Walsh (1986) performed a study on 26 patients with obstructive sleep apnoea using somnofluoroscopy as the imaging modality before and after UPPP. They found 15/26 (58%) of patients obstructed at a level above a horizontal line drawn through C2 (corresponding to the tip of the soft palate in most patients).

All 26 patients underwent UPPP irrespective of the site of obstruction, only 11/26 (42%) were deemed as responding successfully to the surgery with an AHI < 50% the initial level and SaO2<85% less than 15% per hour of sleep.

These criteria are somewhat questionable as it is generally recognized that AHI<20 should be the criteria for success of any treatment regime for obstructive sleep apnoea. Patients with an AHI above this level are at greater risk of developing systemic comorbidities related to their obstructive sleep apnoea (see Chapter 2). Furthermore this study failed to quantify pre and post surgery AHI for any individual patient, or even an average for the group. The important finding was 3/15 (20%) patients who obstructed in the nasopharyngeal segment failed to respond to UPPP. This study again demonstrated that there would appear to be multiple factors contributing to UAW obstruction in some patients with obstructive sleep apnoea and these patients cannot be classified according to anatomical factors alone.

Shepard and Thawley (1990) reported on six patients before and after UPPP with all obstructing in the nasopharynx and three with extension into the oropharynx. All patients continued to obstruct post operatively with only one patient deemed a success by the authors as measured by AHI (decrease from 52 events/h to 23 events/h). The average AHI preoperatively for this group was 71 +/-16 events/h and decreased to average 69 +/- 17 events/h postoperatively. The three patients obstructing in the nasopharynx preoperatively continued to do so postoperatively. The fourth patient continued to collapse in the oropharynx, another had extension into the hypopharynx whilst the final patient had collapse confined to the nasopharyngeal segment.

Selection of procedure according to the anatomic problem is a seemingly obvious statement. When UPPP was first advocated as a treatment modality for obstructive sleep apnoea, Fujita et al (1981) utilized direct visualization to determine the site of obstruction. These authors examined and operated on 12 patients and found all patients examined had a shallow oropharyngeal space, a relatively large uvula and wide posterior pillar mucosa. The posterior wall of the oropharynx contained redundant mucosa and was wrinkled. In only 50% of the patients was the tongue base enlarged. The patients were examined in the supine position as it was felt that the palatopharyngeal arch might be lowered in some patients in this position, although appearing patent when the patient was upright.

One of the problems of UPPP has been a failure rate of up to 50% in some studies. Riley et al (1985) reported a small study of 14 patients with obstructive sleep apnoea treated by UPPP and found nine patients failed in the goal of decreasing AHI to 20 events/hr or less.

They examined lateral cephalometric radiographs of all patients and found that in eight of these patients had an inferiorly positioned hyoid (MP – H) and all had significantly shorter palatal lengths (30.3 mm) compared with the five treatment successes (36 mm).

Woodson et al (1997) utilized lateral cephalometric radiographs in the assessment of patients undergoing UPPP. They found responders (defined as a postoperative AHI < 20) had shorter total and lower airway lengths and a longer posterior mandibular height ($p<0.05$) than nonresponders.

Complications of UPPP have been reported in the literature and range from nasopharyngeal stenosis to death. Fairbanks (1990) conducted a retrospective study by questionnaire of 72 locations in the United States that performed UPPP for treatment of obstructive sleep apnoea. The study was aimed at determining the complications associated with this procedure. There was no estimate made of the total number of UPPP procedures performed across the 72 locations. There were 16 fatalities and seven near fatalities. Airway loss postoperatively occurred in 17 cases. Nasopharyngeal stenosis occurred in 46 patients and there was permanent palatal incompetence resulting in fluid regurgitation in 42 patients and speech impediment in seven patients. Haemorrhage was estimated by respondents to occur at the same rate as for tonsillectomy (reported at 1% to 7% of patients) and wound dehiscence and infection was "common".

Riley et al (1993) reported nasal reflux in 12% of patients postoperatively with resolution of all cases.

A review of the literature by Sher (1995) cited the following as the most common complications encountered following UPPP:

1. Loss of airway resulting in patient death;
2. Haemorrhage, with a similar incidence to tonsillectomy;
3. Palatal incompetence leading to nasal reflux, although this rarely persists;
4. Altered voice quality;
5. Nasopharyngeal stenosis; and
6. Changes in swallowing and throat sensation.

He et al (1988) noted that treatment by tracheostomy or nCPAP significantly increased the survival of patients suffering obstructive sleep apnoea, however there was no significant increase in survival between patients treated by UPPP and those patients who declined all intervention.

## 8.5 Tongue Reduction Surgery

Some authors, to increase the oropharyngeal/hypopharyngeal airway space, have also advocated tongue reduction surgery. There are no series reported in the literature indicating success for this treatment as the sole treatment modality.

Shukowsky first published the concept of tongue advancement as a treatment for UAW obstruction in 1911. He reportedly sutured the tongue around the lower incisors on an infant with micrognathia.

In 1946 Douglas described a surgical procedure whereby the ventral surface of the tongue was denuded and sutured to the lower lip, inducing a sublingual scar that held the tongue forward.

Macroglossia, either relative or absolute has been implicated in the development of obstructive sleep apnoea in some studies. Two main approaches have been used to treat this condition dependant upon the diagnosis. For patients with relative macroglossia surgery to increase the volume of the oral cavity, or at least advance the tongue within the oral cavity to avoid obstruction in the posterior oropharynx has been attempted. The procedure varies however the net effect is advancement of the genial tubercles and hence advancement of the main body of the tongue (see Section 8.7).

Patients diagnosed with absolute macroglossia may be treated by tongue reduction surgery, principally reduction in muscle bulk of the posterior one third of the tongue. This procedure causes significant morbidity immediately post operatively, and usually requires intensive airway management until the oedema subsides.

## 8.6 Hyoid Suspension

Hyoid suspension has been advocated to increase the hypopharyngeal cross-sectional area. This procedure may be done alone or in combination with genial tubercle advancement. The combination procedure also advances the tongue, opening the airway at the base of the oropharynx.

For patients whose site of obstruction lies in the oropharynx or hypopharynx there is limited or no benefit from UPPP. Riley et al (1989) advocates surgical advancement of the tongue base if there is no obvious skeletal deficiency on a lateral cephalometric radiograph. To avoid altering the profile of the patient the surgery involves genial advancement and removal of the buccal cortex of the bony window containing these structures, hyoid myotomy and suspension of the hyoid to the mandible. This suspension may be performed by way of a fascia lata graft or thick non resorbable suture (Riley et al, 1994).

## 8.7 Genial Advancement

Riley et al (1989) studied a group of 55 patients, assessing them pre operatively for site of obstruction by way of nasendoscopy and lateral cephalometric radiography. Polysomnographic sleep studies were performed on all subjects pre and post operatively. 42/55 patients had obstruction at the oropharyngeal and hypopharyngeal levels (Fujita type II), whilst 6 had obstruction confined to the hypopharynx (Fujita type III), and 7 had previously endured a failed UPPP. All patients underwent genioglossus advancement, the range of advancement reported was 8 – 18 mm, with an average of 13 mm.

This study found 37 patients responded to the surgery, with the AHI falling below 20 and the $SaO_2$ normal or with only minimal falls below 90%. 26/37 patients had a UPPP combined with the inferior mandibular osteotomy and hyoid suspension procedure. 11/17 patients who pre operatively experienced hypertension for which they took medication became normotensive post operatively. Other studies report similar success 12/15 (80%).

Patients who failed to respond satisfactorily post operatively consisted of 16/18 patients who had UPPP and the inferior mandibular osteotomy and hyoid suspension procedure, 1/18 had UPPP only and 1/18 had an inferior mandibular osteotomy and hyoid suspension procedure only.

Johnson and Chinn (1994) reported on seven patients treated with concurrent UPPP and genial tubercle advancement and two patients treated sequentially with these surgeries. Seven of the patients had a postoperative RDI < 10 which was deemed successful treatment.

## 8.8 Mandibular Advancement

Mandibular advancement alone has not been widely reported in the literature for the surgical management of obstructive sleep apnoea. This procedure has been combined most commonly with hyoid suspension (Riley et al, 1986) and genial tubercle advancement (Riley et al, 1989). Kuo et al (1979) first reported mandibular advancement for the treatment of obstructive sleep apnoea. Powell et al (1983) reported a single case where an obstructive sleep apnoeic patient who had a retrognathic mandible underwent a mandibular advancement osteotomy. This procedure was performed after a number of other interventions, both surgical and non-surgical, had failed.

Isono (1995) investigated the effect mandibular advancement has on the pressure generated in the upper airway. They studied 13 patients under general anaesthetic and total muscle paralysis with obstructive sleep apnoea, inducing apnoeic events by manipulating nasal airflow. They then maximally advanced the mandible (manually) and in 12/13 patients the

upper airway remained patent at nasal airflow pressures that had previously resulted in apnoea. They concluded from this study that the upper airway is increased in width at the base of the tongue and the nasopharynx as a result of mandibular advancement. This confirms the principle behind the use of mandibular repositioning appliances and indicates surgical advancement in patients with a retrognathic mandible should be considered.

Mandibular advancement alone is not a widespread surgical treatment for obstructive sleep apnoea. The incidence of a skeletal Class II malocclusion in the Australian Caucasian population is reported to be 11.1% (Clinch, 1951). Surgery to advance the mandible would produce an unacceptable malocclusion in approximately 90% of cases if subjects with obstructive sleep apnoea had the same proportion of skeleltal malocclusions as the general population. Reversible, non-surgical advancement by means of mandibular repositioning appliances is common. The use of these devices was reviewed in Chapter 7.

## 8.9 Bimaxillary Advancement

A final procedure to move the skeletal base involves a bimaxillary osteotomy. Treatment of obstructive sleep apnoea by maxillomandibular advancement has been advocated over the last decade or so in selected patients who generally do not tolerate nCPAP and for whom other treatment may have failed.

Orthognathic surgery has been used for many years for the treatment of skeletal abnormalities of the maxillofacial region for both functional and aesthetic reasons. More recently this type of surgery has been advocated as a possible treatment for patients with obstructive sleep apnoea.

The literature describing orthognathic surgical treatment of obstructive sleep apnoea reports maxillary osteotomies principally being at the Le Fort 1 level and mandibular advancement via bilateral sagittal split osteotomy. Riley et al (1987) recommend maxillary advancement of 4 – 8 mm (average 6 mm) and mandibular advancement of 12 – 24 mm (average 16 mm).

Riley et al (1990) compared the results of treating obstructive sleep apnoea with bimaxillary advancement osteotomy or nCPAP. They found in a group of 30 patients no significant difference between either treatment modality when comparing any of the respiratory variables measured an average of 12.6 months postoperatively (range 6 – 24 months). Furthermore when comparing the baseline polysomnographic results with nCPAP and surgery the results were highly significant ($p<0.00001$).

Bimaxillary advancement was successful in 89/91 patients and produced comparable polysomnographic results to nCPAP (Riley et al, 1993). The mean follow up for these patients was nine months and the results are reproduced as Table 8.9-1.

**Table 8.9-1: Results of bimaxillary surgery on 91 patients from Riley et al (1990)**

| | Preoperative | nCPAP | Postoperative |
|---|---|---|---|
| **RDI (events/hr)** | 68.3 +/- 23.3 | 7.6 +/- 5.9 | 8.4 +/- 5.9 |
| **$SaO_2$ min (%)** | 63.2 +/- 17.5 | 87.0 +/- 3.9 | 86.6 +/- 3.4 |
| **BMI ($kg/m^2$)** | 31.1 +/- 6.3 | | 30.5 +/-5.9 |

These authors had proposed a surgical protocol for the management of obstructive sleep apnoea and 24/91 had undergone previous genioglossus advancement with hyoid myotomy and UPPP. This surgery had been unsuccessful with the RDI and $SaO_2$ improving only marginally (75.1 to 56.3 events/hour and 64.0 to 71.9% minimum $SaO_2$). Seven of the ninety one patients had a skeletal deformity as determined from lateral cephalometric radiographs and the remaining 58 patients were from outside referral centres who had undergone unsuccessful UPPP.

A finding of some studies using lateral cephalometric radiographs is a deficiency antero – posterior of the maxilla and mandible with resultant airway narrowing. Hochban et al (1994) reported on a group of 21 patients suffering obstructive sleep apnoea and fitting these criteria whom underwent bimaxillary advancement. Patients were excluded from this study if they were suffering multi-organ disease, chronic alcoholism, drug abuse or if they had a BMI>30. Additionally only those patients with an AHI>20 were considered for surgery.

The aim of the surgery was to advance the patients maxilla and mandible 10mm, this goal was achieved in 19/21 patients. 1/21 patient had their maxilla advanced 7mm and the mandible 14mm, whilst the last patient had the maxilla advanced 4mm and the mandible 14mm. The surgery involved a Le Fort I osteotomy and a bilateral sagittal split ostcotomy advancement. Preoperatively the mean AHI was 44.9 +/- 17.5 events/hr and post operatively the AHI was 3.6 +/- 4.7 events/hr ($p<0.001$). Additionally the percentage of time spent with SaO2 < 90% compared with total sleep time fell from a mean of 11.5+/-12.5 to 1.0 +/- 1.1 ($p<0.001$). The respective results for these parameters for the same patients on nCPAP were AHI 2.5 +/- 2.3 events/hr and SaO2 0.7 +/- 0.8, which were not significantly different from the results produced by surgery.

Cephalometric analysis of this group of 21 patients postoperatively showed, as expected a significant alteration in the antero – posterior position of the maxilla and mandible. They also

showed a significant increase in the posterior airway space at all levels of the UAW, ranging from an average 3.9 mm at the level of the occlusal plane to 6.9 mm measured at the mandibular plane. Measurements were done according to the parameters described by Hochban et al (1993).

Comenero et al (1991) reported on four patients, all with significant maxillofacial abnormalities underwent corrective surgery. One of the patients underwent surgery specifically to treat his obstructive sleep apnoea, whereas the other three patients had significant abnormalities for which they sought surgical correction. Two cases involved ankylosis, one in a 14 year old female secondary to a birth injury. Her preoperative AHI was 53.6, postoperatively AHI was 0. The second sixty two year old patient had congenital ankylosis of the right TMJ and a preoperative AHI of 103. His postoperative AHI was 5.4. The third twenty year old patient suffered Treacher-Collins syndrome and had a preoperative AHI of 45.2. Postoperatively her AHI was 0. The final seventeen year old patient was diagnosed with Long Face Syndrome and had a preoperative AHI of 31.4. Postoperatively her AHI was 0.

Hochban et al (1996) assume the effectiveness of maxillomandibular advancement is due to the straightening of the suprahyoid and velopharyngeal musculature and tendons. The concern is the lack of long term follow-up reports on these patients beyond five years – how much relapse will occur? The first measure of success in the long term is obviously maintenance of an AHI at a therapeutic level. The second measure is indirect and involves determining the degree of relapse occurring postsurgery. If there is soft tissue relapse then skeletal stability may be maintained but relaxation or stretching of the soft tissue may be responsible for the increase in AHI and failure of surgery in the longer term. Alternatively there may be skeletal relapse and concomitant soft tissue movement with its skeletal framework such that pathologic upper airway narrowing and obstruction occurs.

Conradt et al (1997) reported a two year follow up of 15 patients with obstructive sleep apnoea who had an average preoperative AHI of 51.4 events/hour. All patients underwent bimaxillary osteotomy surgery for obstructive sleep apnoea. The magnitude of advancement for the maxilla and mandible averaged 10 mm in each patient. The group had a mean AHI of 5.0 events 6/52 postoperatively and 8.5 events/hour at two years postoperative. In 12/15 patients the AHI was <10. One of the patients who failed treatment was found to have central sleep apnoea, which is not treated surgically because it has a different pathophysiology to obstructive sleep apnoea. Importantly there was no significant change in BMI during the study period, which was 28.3 +/- $kg/m^2$. This study shows an increase in AHI over the two year period but makes no mention of whether skeletal relapse occurred or if soft tissue "stretching" occurred. Will this increase in AHI with the passage of time be maintained such

that at five, ten or twenty years these patients are once again experiencing moderate to severe obstructive sleep apnoea?

Riley et al (2000) published a long-term review of forty patients who had undergone bimaxillary advancement for obstructive sleep apnoea. The mean follow up for this group was 61.0 +/-24.7 months. Thirty-six of the patients remained satisfactorily treated over this period when measured by polysomnographic data, whilst four patients had experienced recurrence of their obstructive sleep apnoea. The results of this study are summarized in Table 8.9-2 and Table 8.9-3.

The RDI for those patients successfully treated is interesting as the number of events per hour continued to decrease in the long term, falling from a mean 9.3 events/hour to 7.6 events/hour. This is in contrast to those results published by Conradt et al (1997). There was a small increase in BMI for both groups, although the greatest increase was in the four subjects who ultimately failed surgical treatment. The four patients who failed treatment in the long term had more severe obstructive sleep apnoea as measured by both $SaO_2$ min and RDI. They responded less convincingly to surgery at six months although there is no significant difference in RDI or $SaO_2$ min measured at this time.

**Table 8.9-2: Long-term results of 36/40 patients successfully treated for obstructive sleep apnoea by bimaxillary surgery adapted from Riley et al (2000).**

| | Preoperative | 6/12 Postoperative | Long-term |
|---|---|---|---|
| **RDI (events/hour)** | 69.9 +/- 25.9 | 9.3 +/- 5.3 | 7.6 +/- 5.1 |
| **$SaO_2$ min (%)** | 69.8 +/- 15.5 | 85.6 +/- 4.6 | 86.3 +/- 3.9 |
| **BMI ($kg/m^2$)** | 31.7 +/- 6.6 | 31.4 +/- 6.3 | 32.2 +/- 6.4 |

**Table 8.9-3: Long-term results of 4/40 patients unsuccessfully treated for obstructive sleep apnoea by bimaxillary surgery adapted from Riley et al (2000).**

| | Preoperative | 6/12 Postoperative | Long-term |
|---|---|---|---|
| **RDI (events/hour)** | 83.2 +/- 37.9 | 10.5 +/- 6.7 | 43.0 +/- 28.6 |
| **$SaO_2$ min (%)** | 66.5+/- 13.5 | 87.5 +/- 1.7 | 81.7 +/- 3.8 |
| **BMI ($kg/m^2$)** | 28.7+/- 7.1 | 28.0 +/- 6.7 | 30.6 +/- 9.2 |

Ongoing research and debate in the literature has surrounded the stability, both short term and long term of these surgical procedures. Correction of skeletal abnormalities of the maxillofacial region in obstructive sleep apnoea patients generally involves advancement of

one or both jaws. Surgical treatment of patients with no underlying skeletal abnormality involves the advancement of both the maxilla and mandible in order to maintain proper dental relationship and a functional occlusion. The difficulty encountered when comparing the literature on maxillomandibular advancements in non – obstructive sleep apnoea patients and extrapolation to obstructive sleep apnoea patients is the magnitude of the advancement. Generally the advancement in surgery for obstructive sleep apnoea is at least 10mm in each jaw, often near the upper limit in surgery for correction of skeletal discrepancies.

Numerous studies in the literature have been directed at determining the stability of the maxilla and or mandible following advancement surgery (Ching, 1995; Hing 1989; Luyk and Ward-Booth, 1985; Rubens et al, 1988; and Van Sickels et al, 1986). The possible differences when considering the surgical advancement of obstructive sleep apnoea patients jaws are the magnitude of the advancement. Most authors advocate a minimum desirable advancement of 10mm for surgical management of obstructive sleep apnoea (Hochban et al, 1994; Riley et al, 1987; and Nimkarn et al, 1995). There are few studies of relapse in the non obstructive sleep apnoeic patient who have undergone advancement of this size in one jaw, let alone both.

The method of fixation has a bearing on the stability of orthognathic surgery. The most common method of fixation is miniplates in the maxilla and miniplates and or bicortical screws in the mandible.

Relapse following orthognathic surgery is considered to occur in the short-term or the long-term and it is hypothesized the mechanisms are different.

Short-term relapse following BSSO mandibular advancement occurs within the first 6-8/52 postoperatively and is due to movement at the osteotomy site. Gassamann et al (1990) used three bicortical screws for fixation following BSSO mandibular advancement. They suggested relapse is due to stretching of the soft tissue envelope and a small area of bony contact between the two bone segments. They found both these factors were more important as the magnitude of linear advancement increased. Blomqvist and Isaksson (1994) confirmed this finding.

Methods of securing the condyle into its correct position during a BSSO abound in the literature because of reports that condylar distraction and subsequent settling into its anatomic position may be a factor in early relapse. Hing (1989) found no correlation between early relapse and condylar displacement at the time of surgery in a study of 47 patients undergoing BSSO advancement +/- Le Fort I osteotomy.

A number of authors have reported the use of skeletal wires and maxillomandibular fixation in patients undergoing large (>10mm) BSSO advancements (Mayo and Ellis, 1987; and Van

Sickels, 1991) and found decreased short term relapse when compared to patients with bicortical screw fixation only.

Long-term relapse is postulated to occur because of condylar remodeling. Merkx and Van Damme (1994) and Scheerlinck et al (1994) have reported progressive condylar resorption in patients undergoing large mandibular advancements. These authors report a change in the shape of the condyle from normal to finger shape and an associated loss of height and a later decrease in posterior facial height. This type of resorption has been seen where the BSSO fixation was wire osteosynthesis (Schendel and Epker, 1980), miniplates (Scheerlink et al, 1994) or bicortical screws (Van Sickels, 1991).

Maxillary advancement at the Le Fort I level less than 10mm has been found to be a relatively stable procedure provided rigid fixation is used (Egbert et al, 1995; Louis et al, 1993; and Luyk and Ward-Booth, 1985). Small surgical advancements of the maxilla less than 5 mm have been found to be the most stable (Louis et al, 1993; and Luyk and Ward-Booth, 1985).

In contrast, Louis et al (1993) found no significant difference in the amount of relapse between separate groups undergoing advancement of the maxilla 12.3 +/- 2.8 mm or 4.7 +/- 0.8 mm. They did note a tendency for greater relapse in the group with the larger advancement but concluded that the use of rigid fixation minimized the relapse. This surgery was performed on patients having maxillomandibular advancement for treatment of obstructive sleep apnoea and confirms that many patients with this condition undergo large advancements in an attempt to cure their condition.

Nimkarn et al (1995) studied 19 patients with an average maxillary advancement of 7.5 mm, mandibular advancement of 10mm and chin advancement of 4 mm and considered two questions. The first was to examine the long term stability of the maxillary and mandibular position on these patients. The second question was to find any association between the magnitude of the advancement and the amount of post operative movement.

The cephalometric landmarks used by the authors in the horizontal plane were A point and B point and in the vertical plane ANS, PNS and gonion. The cephalometric radiographs were taken pre operatively, one month post operatively and greater than 12 months post operative. Unfortunately there is no information given as to the average or mean follow up period of the patients, and 12 months post operative should arguably be considered medium term follow up. The authors counter this argument by quoting a MS Thesis (Nimkarn, 1994) that showed no significant difference in the stability of the landmarks measured for this surgical population between medium – (12 to 48 months) and long – term (>48 months) follow up. All patients had a Le Fort I advancement and BSSO advancement with fixation by rigid plate and screw fixation in the maxilla and three bicortical screws on each side of the mandible.

The results of this study showed no significant difference in position of the landmarks measured on the immediate post operative radiograph and that taken at a period greater than 12 months post operative.

Relapse at 61.0 +/- 24.7 months was reported by Riley et al (2000). The mean advancement for forty subjects was 7.25 +/- 1.2 mm (mandible), 10.9 +/- 2.5 mm (maxilla) and 13.3 +/- 1.8 mm (genial tubercles). They reported relapse only for the mandible as being 0.76 mm, without a standard deviation across the forty patients. Thirty-six of these patients were deemed to have been successfully treated at long term follow up according to polysomnographic criteria.

Whilst considering the possibilities for treatment of obstructive sleep apnoea by skeletal advancement it is prudent also to consider whether skeletal setback surgery has any effect on the PAS and indeed if it may provoke obstructive sleep apnoea or other sleep disorders in previously normal patients. Hochban et al (1996) conducted a prospective study to consider these questions. Sixteen patients undergoing a bilateral sagittal split osteotomy for mandibular prognathism were included. This study involved patients undergoing polysomnographic sleep studies prior to surgery and one year post operatively. The authors found no evidence of sleep disorders in any of the patients, either pre or post operatively. On a lateral cephalometric radiograph they did find that pre operatively all patients had a larger PAS than control subjects did, and post operatively the PAS was reduced to that of the control subjects. Greco et al (1990) had earlier published a paper reporting a decrease in posterior airway space following mandibular setback for treatment of mandibular prognathism when comparing preoperative and postoperative lateral cephalometric radiographs.

Complications of bimaxillary surgery for the treatment of obstructive sleep apnoea are the same as those encountered during this surgery to correct skeletal problems in other populations. Riley et al (1990) found transient paraesthesia of the mental nerve is common, although 87 % resolved at 6 – 12/12 was reported amongst this population of predominately older people.

The complicating factor with this population lies not with the procedure, but with the additional risk posed by the morbidly obese patient undergoing any surgical procedure. Risk management strategies for patients undergoing surgery who have obstructive sleep apnoea have been recommended by Riley et al (1997) and in a review of the literature by Ronderos and Boyd, (1995). They recommend nCPAP use preoperatively and all patients who routinely used nCPAP preoperatively should continue its use in the immediate postoperative period. Awake fibre-optic intubation is recommended if there is increased neck circumference or mandibular skeletal deficiency or if the anaesthetist or surgeon expects a

difficult intubation. Aggressive management of hypertension is necessary, even in patients with no previous history of hypertension. Narcotic analgesics may be used if the patient is monitored closely. They recommend ICU monitoring in the first night post operatively and continuous monitoring by pulse oximetry throughout the duration of hospitalization.

Surgery for obstructive sleep apnoea offers the potential for long term amelioration of the signs and symptoms of obstructive sleep apnoea. There is a group of patients who don't respond to surgery. It remains to be determined whether these patients have failed treatment because surgery was directed at the wrong area of the upper airway or if some other factor is involved. There are a large number of surgical treatment options available and most are not reversible, or reversible with great difficulty. Surgery for treatment of obstructive sleep apnoea should not be offered lightly and patients must be fully consented with respect to the surgical procedure and the possibility that it will not result in effective treatment of their condition.

# Chapter 9

# Materials and Methods

## 9.1 Selected Patients

Patients were recruited for this study from referrals to the Thoracic Medicine Department, Royal Adelaide Hospital. All patients had been referred by general medical practitioners for investigation of symptoms suggestive of obstructive sleep apnoea syndrome. These symptoms include snoring, hypersomnolence, irritability, fatigue, morning headaches and frequent nocturnal gasping and choking as noted by a partner.

All patients attending the sleep Medicine Clinic Outpatients were assessed by a Consultant or Registrar and questioned on their sleep habits and symptoms. Those patients suspected of sleep disordered breathing severe enough to warrant investigation by overnight polysomnography were asked to participate in this study. There was no compulsion for patients to enroll and not all Thoracic Medicine Consultants in the clinic did refer patient for the study. Parameters measured during the sleep study include:

1. Electro-encephalogram;
2. Electro-oculogram;
3. Nasal and oral airflow with thermistors;
4. Thoracic and abdominal muscle movement;
5. Oxygen saturation by way of a pulse oximeter;
6. Electro-cardiogram by three lead recording;
7. Body position;
8. Snoring loudness; and
9. Video monitoring of the patients throughout the study period.

A standard protocol is followed for all patients undergoing a polysomnographic study in the sleep laboratory at the Royal Adelaide Hospital.

A copy of the information provided to the patient and the consent form are included in the Appendix. Ethics approval was sought and obtained from the ethics committee of the Royal Adelaide Hospital to record lateral cephalometric radiographs on patients agreeing to participate in the study who were being investigated for obstructive sleep apnoea.

Those patients willing to participate in the study were asked to attend the Adelaide Dental Hospital for recording of a lateral cephalometric radiograph and measurement of neck circumference after this initial consult appointment. Those patients who could not attend at

that time were asked to present to the Adelaide Dental Hospital on the morning after their overnight sleep study.

Those patients requiring a sleep study had a delay of no more than three weeks between recording of the lateral cephalometric radiograph and recording of the sleep study.

This method of patient selection and recording of the lateral head radiograph was chosen so the investigator did not know which patients were OSA or simple snorers as defined by polysomnographic study. It also ensured we received a population sample that was not biased toward those with OSA syndrome.

## 9.2 Radiographic Technique

Radiographs were obtained at the Radiology unit at the Adelaide Dental Hospital using Fuji HR-S film (24 cm x 30 cm). The film was inserted into a Kodak Lanex cassette with regular screens. The cassettes were inserted into the film holder. A film to midsagittal distance of 16cm was used for all cases. Lateral cephalometric radiographs were obtained for all patients enrolled in the study on a single radiographic machine (Philips Super 50 CP lateral cephalometer). All subjects were aligned in a reproducible manner within the cephalostat.

Those patients wearing dental prostheses had a cephalometric radiograph taken with the prostheses in position and a separate exposure with the prostheses removed.

## 9.3 Tracing and Digitizing Procedure

All radiographs were traced onto acetate paper in a darkened room on a lightbox by the same investigator. A cardboard frame was placed around the radiograph to exclude extraneous light. The cephalometric landmarks (hard and soft tissue) were identified and marked onto the acetate paper with a 0.05mm diameter B lead pencil. The SN-7 line was constructed on each tracing, originating at sella and at seven degrees to the sella – nasion line. The location of each cephalometric point was recorded with the film orientated to the SN-7 line.

The radiographic tracings were digitized to enable accurate recording of linear and angular measures from the tracings. Each tracing was placed on the digitizer tablet and orientated to the line FH. The digitizer was a *Hewlett Packard 9874A* configured to an *Apple IIe* computer. The software program *Cephs*, developed by Brown (1986, personal communication) was used to record and manipulate the data obtained from the radiographic tracings. Patient details Unit Record Number, age and gender were entered. The magnification factor of 8.8% was corrected. This program accepts the digitized record and "transforms" the cartesian coordinates relative to line FH. Each traced landmark point was

aligned centrally in a crosshair and notated by depressing a perimeter button on the circular cursor. The data for the points were transformed by the software program and saved to a 3.5" computer disc. The one investigator recorded all data.

The investigator remained unaware of the status of the patient with respect to their sleep study during this phase of the study.

## 9.4 Reference Points and Lines

The angle formed between the cranial base and Frankfort horizontal has been measured to determine the angulation of the cranial base. The sella – nasion line was used extensively in this study to allow angular measures of anteroposterior position of the hard and soft tissue structures of interest. Frankfort horizontal was also used in a similar manner. Frankfort horizontal closely approximates the natural head position (Bjerin, 1957) and the influence of growth on the angulation of this line is limited. The average angle between Frankfort horizontal and sella – nasion has been studied extensively and found to be approximately $7^0$. Koski and Virolainen (1956) measured 100 cephalometric radiographs and found a mean difference of $6.8^0$ =/- 0.26 for this angle. Fifty male subjects were examined by Wei (1968) and they reported a mean difference of $7.2^0$ +/- 0.42.

### 9.4.1 Hard Tissue Points Identified on a Lateral Cephalometric Radiograph

Figure 9.4.1-1 shows these hard tissue landmarks.

**Sella (S):** The centre of the pituitary fossa of the sphenoid bone determined by inspection (van der Linden, 1971; Vincent and West, 1987).

**Nasion (N):** The most anterior point of the frontonasal suture (Brown, 1973)

**Orbitale (Or):** The lowest point on the average of the right and left borders of the bony orbit (Riolo et al, 1974).

**Porion (Po):** The most superior point on the external auditory meatus (Vincent and West, 1987). The external auditory meatus has three radiolucent areas which distinguish it from the internal auditory meatus: the fenestrum vestibulae superiorly; the fenestrum cochlea posteriorly; and the promontory anteriorly (Yen, 1960).

**Anterior nasal spine or acanthion (ANS):** The tip of the median sharp bony process of the maxilla at the lower margin of the anterior nasal opening (Riolo et al, 1974).

**Posterior nasal spine (PNS):** The most posterior point at the sagittal plane on the bony hard palate (Riolo et al, 1974).

**Articulare (Ar):** The point at the junction of the contour of the external cranial base and the dorsal contour of the condylar processes projected in the midsagittal plane (Brown, 1973).

**Down's Point A or subspinale (A):** The deepest point in the midsagittal plane between the anterior nasal spine and supradentale, usually around the level of and anterior to the apex of the maxillary central incisors (Burstone, 1978).

**Down's Point B or supramentale (B):** The deepest point in the midsagittal plane between infradentale and pogonion, usually anterior to and slightly below the apices of the mandibular incisors (Burstone, 1978). According to Moyers (1987) B point cannot be determined of the chin profile is flat.

**Menton (Me):** The most inferior point on the symphseal outline (Riolo et al, 1974).

**Gonion (Go):** The point of intersection of the line tangent to the lower border and the tangent to the posterior border of the ramus.

**Hyoid (H):** The most superoanterior point on the body of the hyoid bone (Athanasiou et al, 1991)

**Cervical vertebra 4 (C4):** The most anterior inferior point of the fourth vertebral corpus (Pae et al, 1994).

**Cervical vertebra 3 (C3):** The most anterior inferior point of the third vertebral corpus (Pae et al, 1994).

**Upper incisal apex (AS):** The root tip of the maxillary central incisor (Riolo et al, 1974).

**Upper incisal edge (U1):** The incisal edge of the maxillary central incisor (Riolo et al, 1974).

**Lower incisal apex (AI):** The root tip of the mandibular central incisor (Riolo et al, 1974).

**Lower incisal edge (L1):** The incisal edge of the mandibular central incisor (Riolo et al, 1974).

**Mandibular Mid-point (H1):** The point of perpendicular intersection between a line from H to the plane C3-RGn (from Pae et al, 1994).

**Retrognathion (RGn):** The most posterior point of the mandibular symphysis along a line perpendicular to the FH (Frankfort Horizontal) plane (Pae et al, 1994).

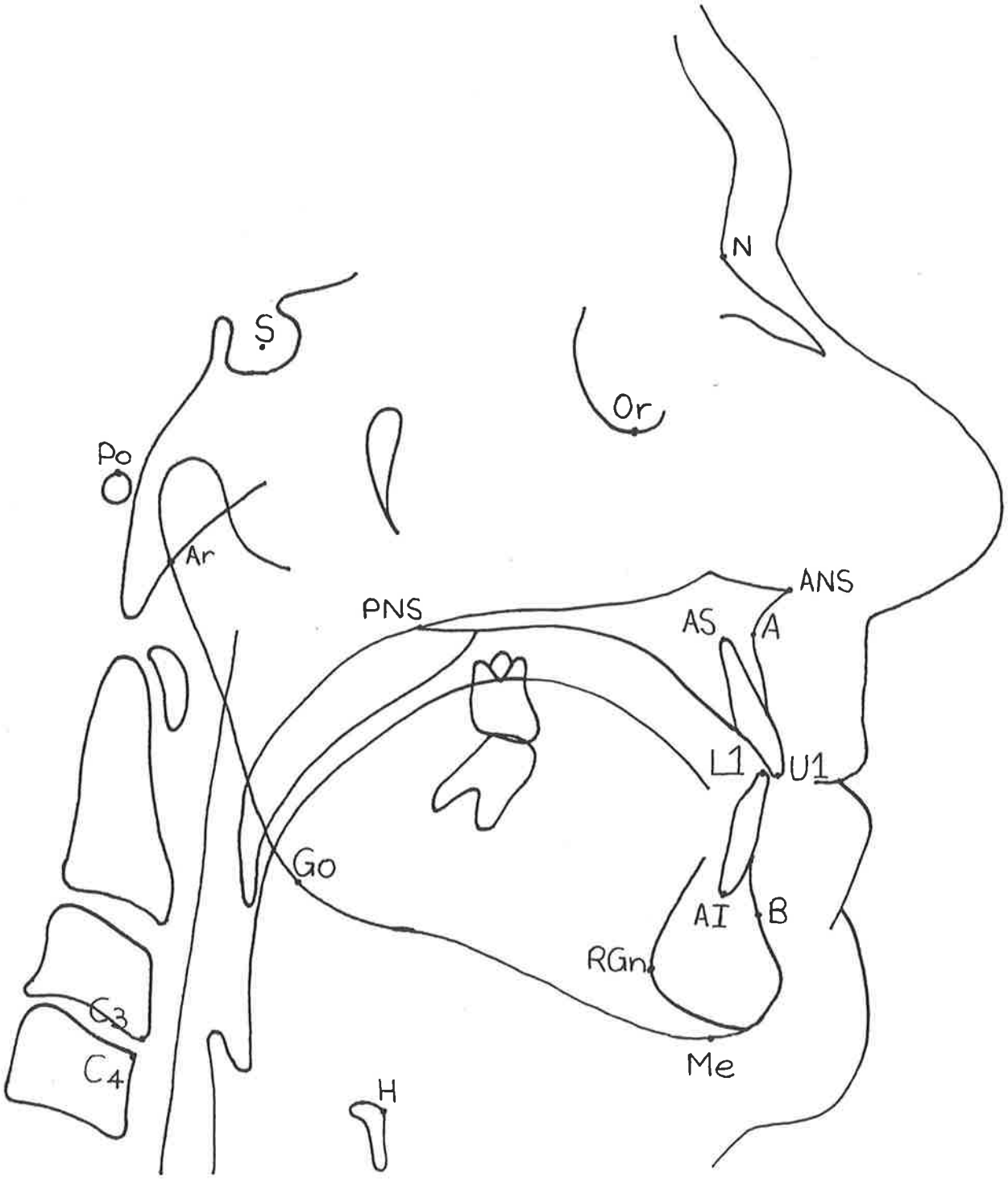


**Figure 9.4.1-1 Hard tissue cephalometric landmarks**

### 9.4.2 Soft Tissue Points Identified on a Lateral Cephalometric Radiograph

Figure 9.4.2-2 shows the soft tissue landmarks used in this study.

**Dorsal tongue protrusion (TP):** The point on the dorsal surface of the tongue where line Go-B intersects the dorsal surface (from Pae et al, 1994).

**Epiglottis base (Eb):** Base of the epiglottis. The deepest point of the epiglottis (Pae et al, 1994).

**Epiglottis tip (Et):** Apex of the epiglottis (deBerry-Borowiecki et al, 1988)

**Tongue tip (TT):** The border between the ventral and dorsal surfaces of the tongue tip (Pae et al, 1994).

**Uvula thickness ventral (UW1):** The point of intersection on the oral surface of the soft palate of a line perpendicular to PNS-P through the thickest portion of the soft palate (from Pae et al, 1994).

**Uvula thickness dorsal (UW2):** The point of intersection on the pharyngeal side of the soft palate of a line perpendicular to PNS-P through the thickest portion of the soft palate (from Pae et al, 1994).

**Soft palate tip (UT):** Apex of the soft palate: the lowest point on the soft palate (deBerry-Borowiecki et al, 1988).

**Soft palate protrusion (UP):** The greatest posterior convexity of soft palate (deBerry-Borowiecki et al, 1988).

**Pharyngeal wall 1 (PhW1):** Point of intersection on the caudal pharyngeal wall on a line parallel to Frankfort Horizontal passing through the tip of the soft palate (UT) (deBerry-Borowiecki et al, 1988).

**Pharyngeal wall 2 (PhW2):** Point of intersection on the caudal pharyngeal wall on a line parallel to Frankfort Horizontal passing through (deBerry-Borowiecki et al, 1988).

**Pharyngeal wall 3 (PhW3):** Point of intersection on the caudal pharyngeal wall on a line parallel to Frankfort Horizontal passing through the posterior nasal spine (PNS) (deBerry-Borowiecki et al, 1988).

**Pharyngeal wall 4 (PhW4):** Point of intersection on the caudal pharyngeal wall on a line parallel to Go-B passing through the point of greatest posterior convexity of the soft palate (UP) (Lowe et al, 1996).

**Pharyngeal wall 5 (PhW5):** Point of intersection on the caudal pharyngeal wall on a line parallel to Go-B passing through the tip of the soft palate (UT) (Lowe et al, 1996).

**Pharyngeal wall 6 (PhW6):** Point of intersection on the caudal pharyngeal wall along an extension of the line Go-B (Lowe et al, 1996).

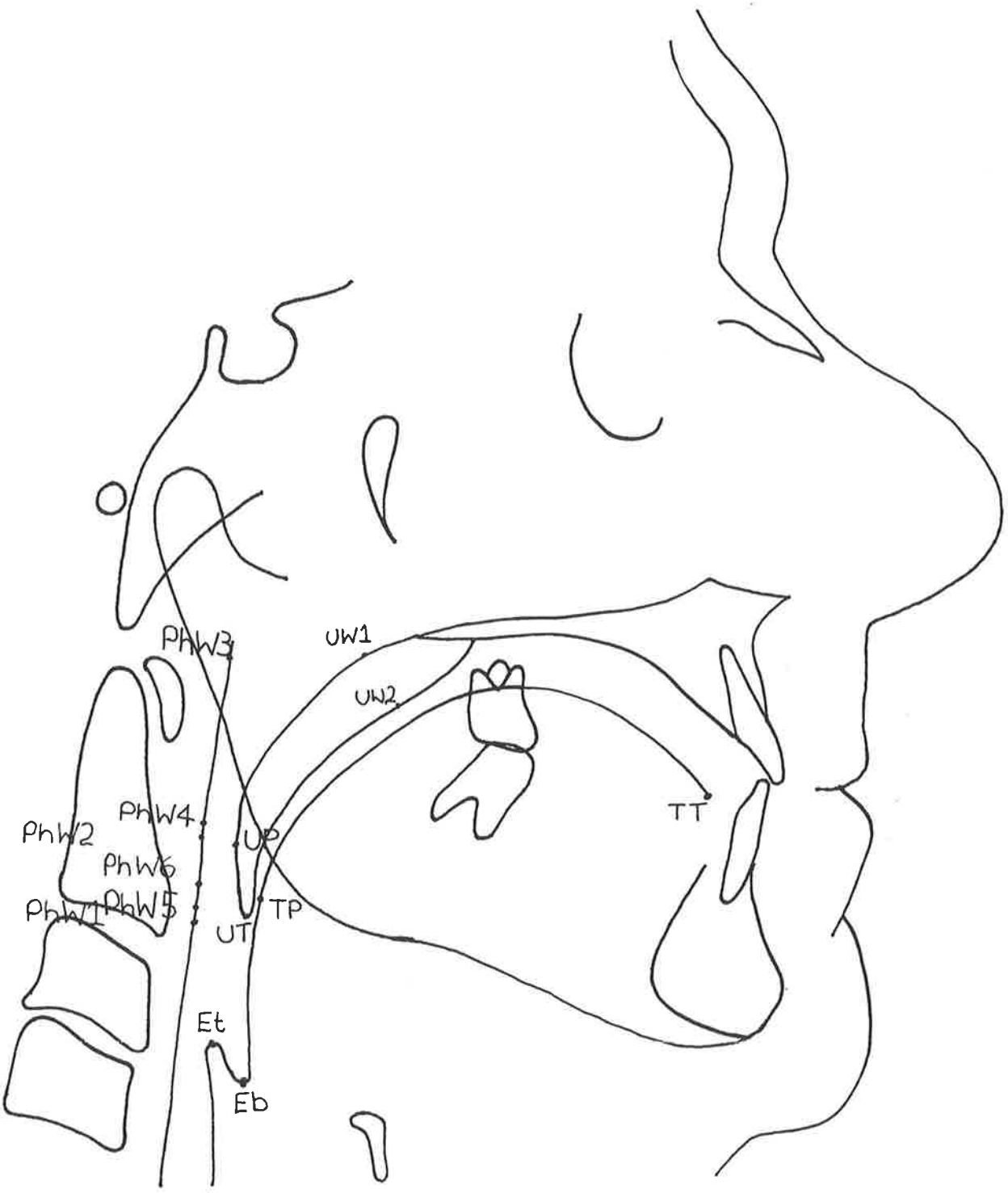


**Figure 9.4.2-2 Soft tissue cephalometric landmarks**

## 9.5 Calculation of Linear and Angular Variables

The variables were selected from those reported by Bacon W et al (1989), Battagel and L'Estrange (1996), deBerry-Borowiecki et al (1988), Lowe et al (1996), Pae et al (1994) and Tsuchiya et al (1992). A second program by Brown (1996, personal communication), New Scorer, was used to compute all measurements. A menu within the program allows a variety of combinations between any of the digitized points. Twenty linear (Figure 9.5.1-1) and nine angular variables (Figure 9.5.2-2) were calculated from the digitized points and stored as disk files. The results were tabulated on Microsoft Excel 97 spreadsheets for final editing and statistical evaluation.

### 9.5.1 Constructed Linear Variables

**MxUL:** Maxillary unit length: The linear distance from the anterior nasal spine (ANS) to the posterior nasal spine (PNS) (Lowe et al, 1996).

**MdUL:** Mandibular unit length: The linear distance from menton (Me) to gonion (Go) (Lowe et al, 1996).

**TGL:** Tongue length: The linear distance between TT and Eb (Pae et al, 1994).

**PNS-UT:** Soft palate length: The linear distance between PNS and UT (deBerry-Borowiecki et al, 1988).

**UW1 – UW2:** Maximum palate thickness: The maximum thickness of the soft palate measured on a line perpendicular to the line PNS-P (Pae et al, 1994).

**UT – PhW1:** Linear distance from the tip of the uvula to the posterior pharyngeal wall measured along a line parallel to the Frankfurt horizontal (deBerry-Borowiecki et al, 1988).

**UP – PhW2:** Linear distance from the point of greatest posterior convexity of the soft palate to the posterior pharyngeal wall measured along a line parallel to the Frankfurt horizontal (deBerry-Borowiecki et al, 1988).

**PNS – PhW3:** Linear distance from the posterior nasal spine to the posterior pharyngeal wall measured along a line parallel to the Frankfurt horizontal (deBerry-Borowiecki et al, 1988).

**UP – PhW4:** Superior posterior airway space: The thickness of the airway behind the soft palate along a line parallel to the Go-B point plane intersecting with the point of greatest convexity on the posterior soft palate (Pae et al, 1994).

**UT – PhW5:** Middle airway space: The thickness of the airway along a line parallel to the Go-B point plane through UT (Pae et al, 1994).

**TP – PhW6:** Inferior airway space: The thickness of the airway along a line extended through the Go-B point plane (Pae et al, 1994).

**H – C4:** Linear distance between H and C4 (Lowe et al, 1996).

**H – C3:** Linear distance between H and C3 (Tsuchiya et al, 1992).

**MP – H:** Linear distance along a perpendicular from H to the mandibular plane (Pae et al.,1994)

**Me – H:** Linear distance from Me to H (Battagel and L'Estrange, 1996).

**Ar – H:** Linear distance from Ar to H (deBerry-Borowiecki et al, 1988).

**S – H:** Distance between sella and the most anterior superior point on the body of the hyoid (deBerry-Borowiecki et al, 1988).

**TT – ET:** Distance from the apex of the tongue to the tip of the epiglottis (deBerry-Borowiecki et al, 1988).

**VAL:** Linear distance between PNS and Eb (Lowe et al, 1996).

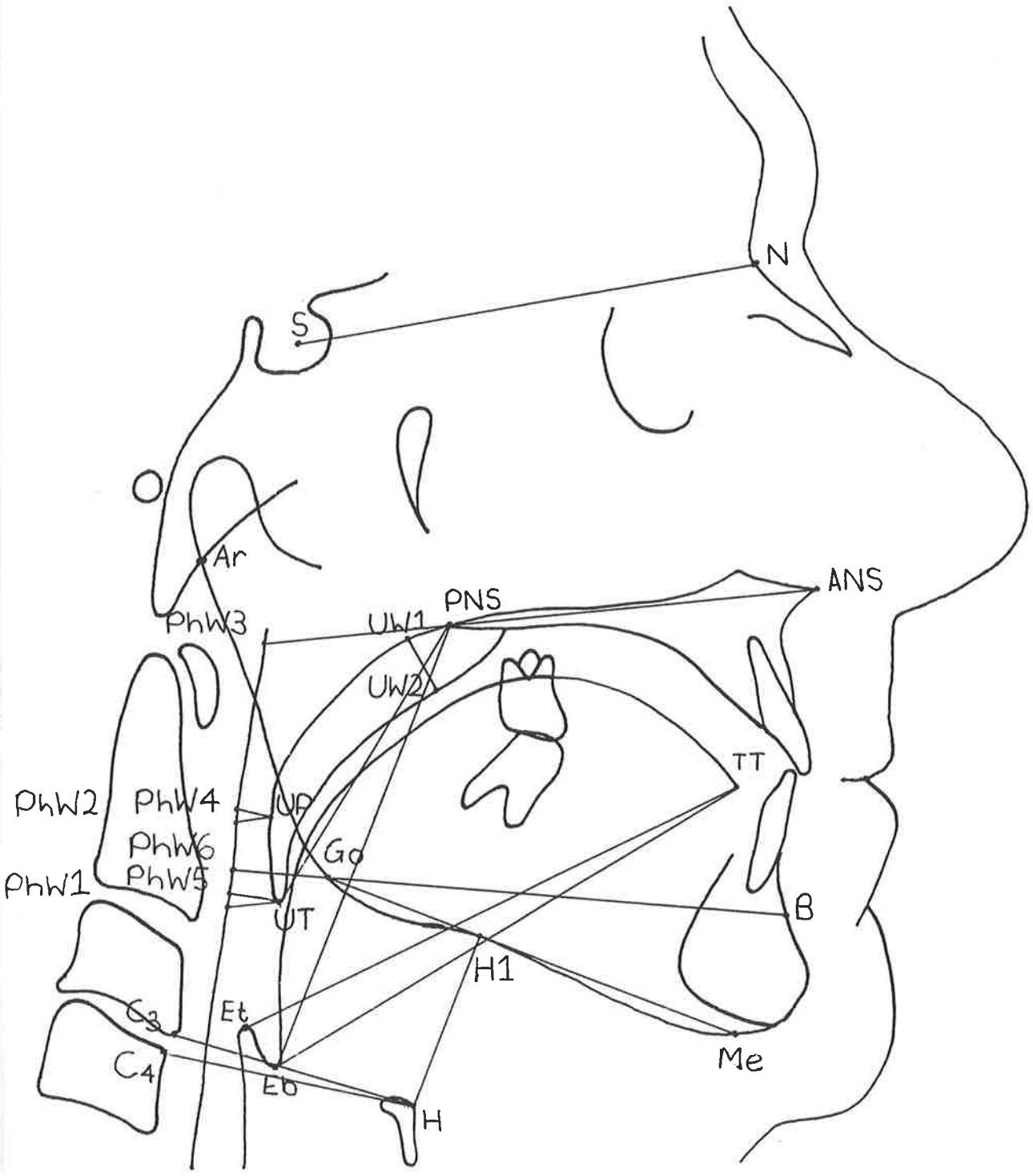


**Figure 9.5.1-1 Linear cephalometric measures**

### 9.5.2 Constructed Angular Variables

**<SNA:** Anteroposterior maxillary position: Angle between the cranial base (S-N) and subspinale (A) (deBerry-Borowiecki et al, 1988).

**<SNB:** Anteroposterior mandibular position: Angle between the cranial base (S-N) and supramentale (B) (deBerry-Borowiecki et al, 1988).

**<SN – MP:** Facial divergence: Angle between the cranial base (S-N) and the mandibular plane (Go-Me) (deBerry-Borowiecki et al, 1988).

**<U1 – SN:** Upper incisor inclination: Angulation of the upper incisor (AS-U1) relative to the cranial base (S-N) (Lowe et al, 1995).

**< L1 – MP:** Lower incisor inclination: Angulation of the lower incisor (AI-L1) relative to the mandibular plane (Go-Me) (Lowe et al, 1995).

**<SN – MxUL:** Palatal plane angulation: Angle between the cranial base (S-N) and the palatal plane (ANS-PNS) (Bacon et al, 1989).

**<N – S – H:** Angular measure of vertical hyoid position: Angle between the cranial base (N – S) and the hyoid bone (H) (deBerry-Borowiecki et al, 1988).

**<Go – Me – H:** Angular measure of vertical hyoid position: Angle between the body of the mandible (Go – Me) and the hyoid bone (H) (deBerry-Borowiecki et al, 1988).

**<NS – ArH:** Angular measure of vertical hyoid position: Angle between the cranial base (N – S) and the line Ar – H (deBerry-Borowiecki et al, 1988).

**<H – H1:** (H – RGn – $C_3$) (Pracharktam et al, 1994)

**< A – N – B:** Angular measurement of the anteroposterior position of the maxilla and mandible (Lowe et al, 1995)

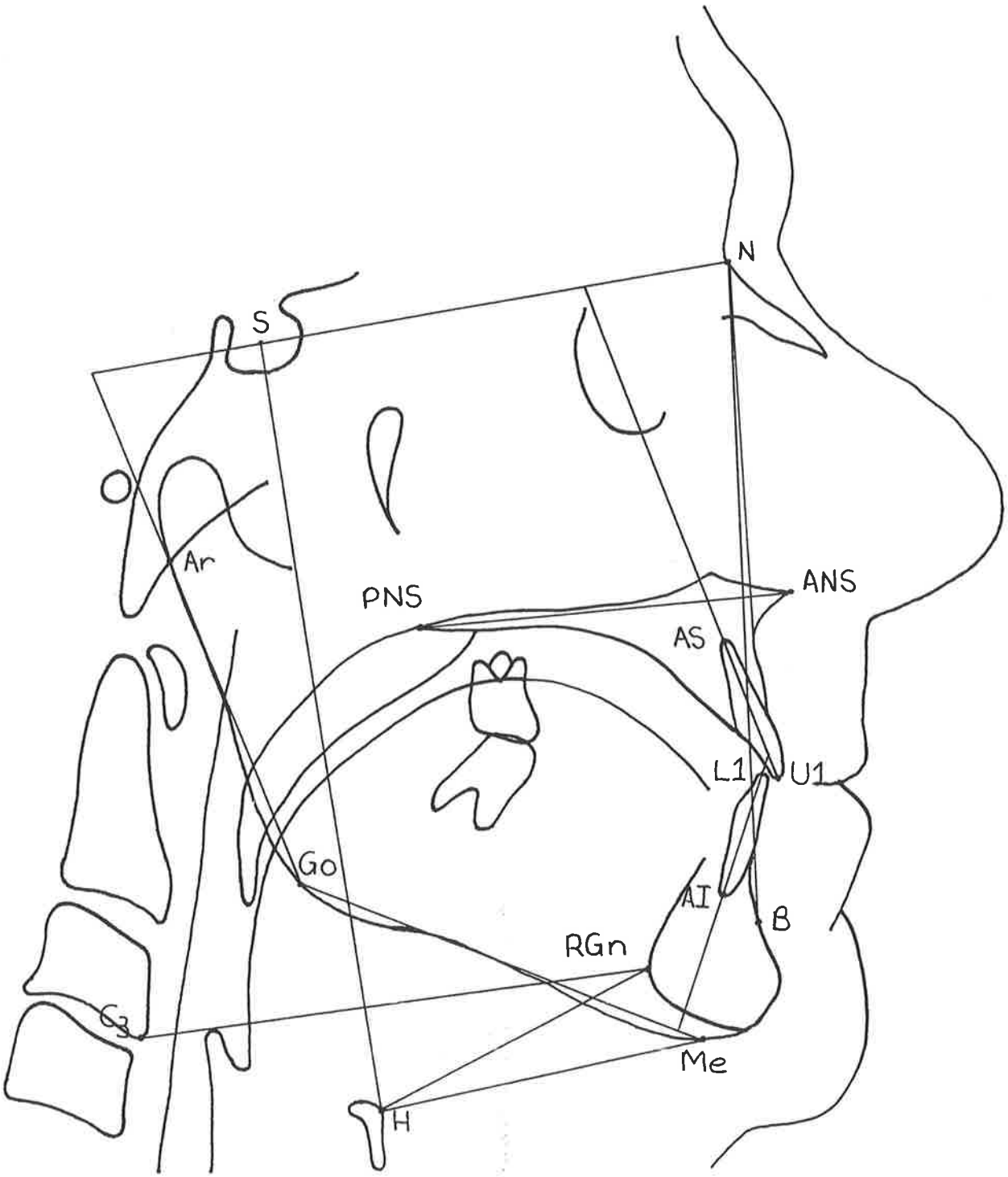


**Figure 9.5.2-2 Angular cephalometric measures**

## 9.6 Statistical Analysis

The linear and angular variables were transcribed from a hard copy of the data produced by the computer program *Cephs* and entered into a Microsoft Excel 97 software program on a PC. The data was analysed using the statistical component of this software program. Charts were produced from the tables generated in the Microsoft Excel 97 software program. Statistical analyses performed were a Pearson correlation, Student's paired t-test and a Pearson's chi-square test with Yates' continuity correction where appropriate. Confirmation of these results was obtained by submitting the data for analysis by the Department of Statistics, University of Adelaide who performed the above statistical analyses on *Apple Mac* computers using *S-plus* code software.

Statistical analysis of the subjects was undertaken by examining the sample as a whole, and following subdivision into groups based upon RDI. Division based upon RDI arbitrarily separated the population into "snoring" patients and those with obstructive sleep apnoea. An RDI of 10, 15 and 20 events per hour was used for these analyses.

For the whole group comparisons the independent variable was RDI for the first analyses and BMI for the second. The dependant variables for these analyses were those cephalometric measures defined at Chapter 10.5.1 Constructed Linear Variables and at Chapter 10.5.2 Constructed Angular Variables. Additional dependant variables used were minimum arterial oxygen saturation ($SaO_2$), neck circumference, sex, age and BMI or RDI (depending upon which was the independent variable for the analysis).

A limited statistical analysis was undertaken comparing the position of the maxilla, mandible and cranial base relative to the Caucasian cephalometric norms utilised by our unit. Orthognathic surgery undertaken within our unit requires presurgical and postsurgical cephalometric analysis of all patients by Oral and Maxillofacial surgeons and Orthodontists. Known cephalometric norms for a Caucasian population are used during these planning procedures. For patients from a different ethnic background alternative values are used where they have been reported in the literature.

Additional examination of the data was undertaken to determine the maxillary and mandibular position based upon constructed cephalometric angles and consideration of BMI and RDI. The length of the cranial base (S – N) and the angle of the cranial base (Ba – S – N) was also compared with RDI and skeletal pattern. This was undertaken after the distribution of skeletal pattern was revealed. Distance S – N and angle Ba – S – N were manually measured from the original cephalometric tracings by the one investigator.

# Chapter 10

# Errors of the Method

## 10.1 Materials and Methods

The validity of the results of a study involving the tracing and digitizing of cephalometric radiographs requires knowledge of the magnitude of error involved in these processes. The magnitude of error associated with these processes was determined by a series of double determinations for thirteen lateral cephalometric radiographs from ten subjects. These radiographs were randomly selected by the author from the ninety four subjects with complete records who were assessed in this study.

Repeat tracing and digitizing was performed two weeks following the completion of the initial analysis of the full set of ninety four lateral cephalometric radiographs by the one investigator. The same method was used in this process. The repeat tracing of the ten radiographs was performed in a darkened room with a light box. Tracings were orientated on the digitizer table and secured with cellulose tape. The thirty four cephalometric landmarks and two fiducial points (x and y) were recorded on a Hewlett Packard 987A digitizer configured to an Apple IIe computer (Figure 10.1-1). The cephalometric points were identified and placed in the central cross-hair cursor and registered by depressing a perimeter button. The position of the cursor when the button was depressed is transformed into an x and y co-ordinate by the computer. Magnification of 8.8% was corrected, as for the initial series.

The software program transforms the x and y cartesian co-ordinates relative to a nominated reference line, in this instance x – y served as a line of reference. Professor Tasman Brown, The University of Adelaide, developed the software. The error associated with the digitizing equipment has been assessed by Farrer (1984) and reported to be +/- 0.01 mm under normal operating conditions.

The transformed numerical data was transferred to a PC with Microsoft Excel 97 software for further analysis of each of the thirty four points to assess the magnitude of error. Scattergrams were produced to illustrate the reproducibility of each point in accordance with the method described by Broch et al (1981). The origin for each point was designated as the first reading for each point. The points on each scattergram represent the difference between the first and second recording of each landmark.

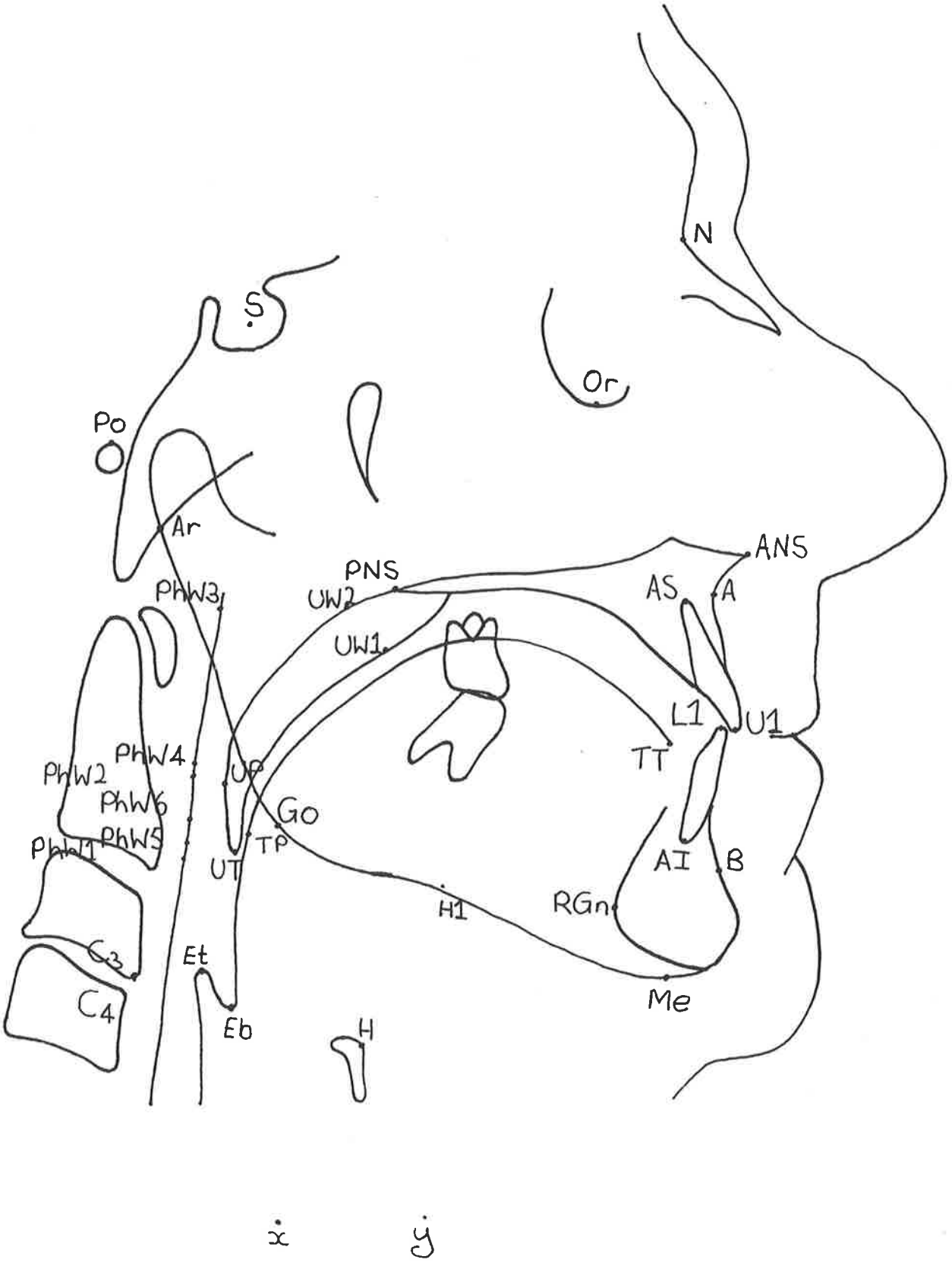


**Figure 10.1-1: Digitized Cephalometric Landmarks**

The differences between the first and second determinations for each cephalometric landmark were recorded as the mean difference ($M_{diff}$), the standard error of the mean difference $E(M_{diff})$ and the standard deviation of a single determination (S error). The Student's t-test for paired values was used to assess whether differences between the first and second determinations differed significantly from zero at the 5% (t=2.262) and 1% (t=3.250) levels for 12 degrees of freedom. Table 10.1-1 lists the respective formulae.

**Table 10.1-1: Statistical Analysis of the Error of the Method**

| | | |
|---|---|---|
| $M_{diff}$ | Mean difference between two determinations | $\sum_{diff}/N$ |
| $E(M_{diff})$ | Standard error of the mean difference | $S_{diff}/\sqrt{N}$ |
| S (error) | Standard deviation of a single deviation | $\sqrt{\sum_{diff}^2/2N}$ |
| t value | Student's paired t-test | $M_{diff}/E(M_{diff})$ |

# Chapter 11

# Results

## 11.1 Introduction

One hundred and four subjects were enrolled in the study between December 1997 and May 1998. There were records for of 74 males and 30 females. The age and sex distribution of the subjects is included at Table 11.1-1. The mean age of this population is 50.5 years which is significantly greater than the mean age of patients undergoing orthognathic surgery in our unit. Ching (1995) reported the mean age of these patients to be 23 years. This difference in age precluded the use of these lateral cephalometric radiographs as a control population as Lewis and Roche (1988) reported growth of the craniofacial skeleton, as measured on lateral cephalometric radiographs, continuing into the fifth decade in some subjects. They reported the total increments in growth of the cranial base and the mandible ranged from 1.01 to 5.53 mm after age 18 until the cessation of growth between ages 29 and 39 years. They concluded this growth is important because it is markedly greater than the errors of measurement.

**Table 11.1-1: Age and Sex Distribution of Enrolled Subjects**

| Sex | Number | Mean Age (yrs) | Std. Dev. | Min. | Max. | Range |
|---|---|---|---|---|---|---|
| **Male** | 74 | 51.6 | 13.0 | 21 | 79 | 58 |
| **Female** | 30 | 47.9 | 9.6 | 27 | 68 | 41 |
| **Total** | 104 | 50.5 | 12.2 | 21 | 79 | 58 |

One hundred and two patients underwent overnight polysomnography of whom ninety-four had results able to be included in the study. Twenty-two subjects did not have a record of their neck circumference and are therefore excluded from analysis of the group when this variable is compared. Five patients had the results of the sleep study and their cephalometric radiograph but did not have a record of their BMI with the sleep study or in the hospital casenotes. These patients were excluded from analyses that used BMI as one of the variables in the statistical analysis. Eleven patients were missing upper central incisor teeth and six patients were missing lower central incisor teeth thus excluding them from analyses when these variables are required.

Nine patients who enrolled in the study and had lateral cephalometric radiographs were excluded. One patient had a sleep study however no record of the results could be found in the patients casenotes. One patient had an inconclusive sleep study, managing just 11 minutes of sleep during the period of the sleep study. This patient who was judged by the thoracic medicine physicians to be suffering OSA on clinical grounds but this could not be further defined. Two patients agreed to participate and had a lateral cephalometric

radiograph but then declined a sleep study. Five other patients did not have results of their sleep study available.

There were 25 patients who wore a dental prosthesis (15 with OSA, 7 simple snorers and 3 patients for whom polysomnographic results were not available). The radiographs utilised for statistical purposes in this study were those taken with the dental prosthesis removed.

One patient had a lateral cephalometric radiograph taken without the hyoid bone in the radiographic field. For this patient the parameters involving the hyoid bone could not be determined. All other subjects had complete lateral cephalometric radiographs for the purposes of this study.

## 11.2 Linear Variable Results

The linear measurements for the subjects included in the statistical analysis and comparison of results is shown in Table 11.2-1. This table also reports the median, standard deviation and the range of each linear variable. There has been no separation of the subjects on the basis of BMI or RDI.

**Table 11.2-1: Linear Measurements of Cephalometric Parameters**

| | Mean | Median | Standard Deviation | Minimum | Maximum |
|---|---|---|---|---|---|
| **MxUL** | 52.02 | 51.57 | 3.79 | 43.06 | 63.59 |
| **Go-H** | 41.07 | 41.58 | 8.38 | 25.23 | 95.91 |
| **MdUL** | 40.54 | 41.56 | 6.41 | 58.96 | 88.05 |
| **TGL** | 76.94 | 76.44 | 7.25 | 62.43 | 98.11 |
| **PNS - UT** | 42.22 | 41.85 | 5.31 | 31.06 | 57.27 |
| **UW1 - UW2** | 10.42 | 10.30 | 1.84 | 6.5 | 15.93 |
| **UT - PhW1** | 20.00 | 19.42 | 6.62 | 4.72 | 41.2 |
| **UP - PhW2** | 35.13 | 35.34 | 6.13 | 14.1 | 53.44 |
| **PNS - PhW3** | 32.87 | 32.24 | 6.30 | 19.88 | 49.11 |
| **UP - PhW4** | 2.06 | 1.62 | 3.46 | 0.05 | 34.67 |
| **UT - PhW5** | 11.15 | 10.77 | 3.85 | 3.98 | 23.1 |
| **TP -PhW6** | 20.70 | 20.31 | 7.92 | 6.62 | 40.59 |
| **H - C4** | 40.68 | 40.19 | 8.41 | 24.31 | 82.88 |
| **H - C3** | 39.66 | 39.72 | 7.47 | 23.01 | 81.80 |
| **MP - H** | 23.09 | 22.10 | 8.21 | 2.06 | 74.1 |
| **Me - H** | 44.08 | 44.19 | 7.74 | 4.8 | 64.59 |
| **Go - H** | 41.17 | 41.62 | 8.44 | 25.23 | 95.91 |
| **Ar - H** | 87.56 | 88.49 | 10.28 | 58.37 | 138.71 |
| **S - H** | 113.83 | 115.38 | 10.45 | 92.82 | 162.80 |
| **FH** | 75.14 | 76.15 | 8.33 | 7.71 | 86.6 |
| **TT - ET** | 74.39 | 74.85 | 7.24 | 59.93 | 95.14 |
| **NS- FH** | 2.43 | 2.61 | 4.39 | -8.70 | 11.80 |
| **VAL** | 71.29 | 71.46 | 7.35 | 53.80 | 86.91 |
| **S-B** | 47.33 | 47.50 | 3.96 | 36.24 | 55.04 |
| **S-N** | 77.27 | 77.75 | 4.27 | 64.5 | 84.5 |

## 11.3 Angular Variable Results

The mean angular measurements for the subjects included in the statistical analysis and comparison of results is shown in Table 11.3-1. This table also reports the median, standard deviation and range of the measurement for each angular variable. There has been no separation of the subjects on the basis of BMI or RDI.

**Table 11.3-1: Angular Measurements of Cephalometric Parameters**

| | Mean | Median | Standard Deviation | Minimum | Maximum |
|---|---|---|---|---|---|
| <SNB | 84.04 | 84.41 | 5.18 | 67.17 | 93.50 |
| <SNA | 83.26 | 84.27 | 4.98 | 70.70 | 94.98 |
| <ANB | 0.78 | 0.36 | 3.62 | -9.50 | 7.72 |
| <SN - MP | 29.46 | 29.81 | 6.81 | 13.44 | 48.48 |
| <U1 - SN | 106.29 | 108.00 | 8.68 | 64.00 | 117.14 |
| <L1 - MP | 89.23 | 90.00 | 1.87 | 64 | 115 |
| <SN - MxUL | 2.57 | 2.15 | 4.12 | -7.86 | 15.74 |
| <NSH | 85.01 | 84.90 | 5.19 | 70.1 | 96.24 |
| <Go - Gn - H | 32.23 | 30.90 | 10.87 | 8.85 | 94.79 |
| <NS - ArH | 72.88 | 73.24 | 5.80 | 59.74 | 88.36 |
| <H - H1 | 19.52 | 18.09 | 10.80 | -10.96 | 72.81 |
| <N-S-B | 124.15 | 124.00 | 6.39 | 106.47 | 139.58 |
| <Ba-S-N | 123.83 | 123.00 | 6.20 | 108 | 140 |

## 11.4 Significant Correlations With RDI

A Pearson correlation statistical test was performed on ninety four patients with results from an overnight polysomnographic sleep study and a cephalometric radiograph. The independent variable in the first analysis was the respiratory disturbance index (RDI). All cephalometric constructed linear and angular variables were correlated with RDI. Additional non-cephalometric variables considered were age, sex, BMI and neck circumference. The data is reported as significant at the $p<0.01$ or $p<0.05$ level. Results where there is no significant correlation are not reported.

### 11.4.1 Body Mass Index (BMI)

The mean body mass index for the total subject population was 32.28 +/- 0.72 kg/m$^2$ and ranged from a maximum value of 51.5 kg/m$^2$ to a minimum of 18.9 kg/m$^2$. The correlation co-efficient r = 0.53 was significant at the 0.01 level of significance with 92 degrees of freedom. Figure 11.4-1 shows diagrammatically the relationship of BMI to RDI with a positive correlation between the two variables.

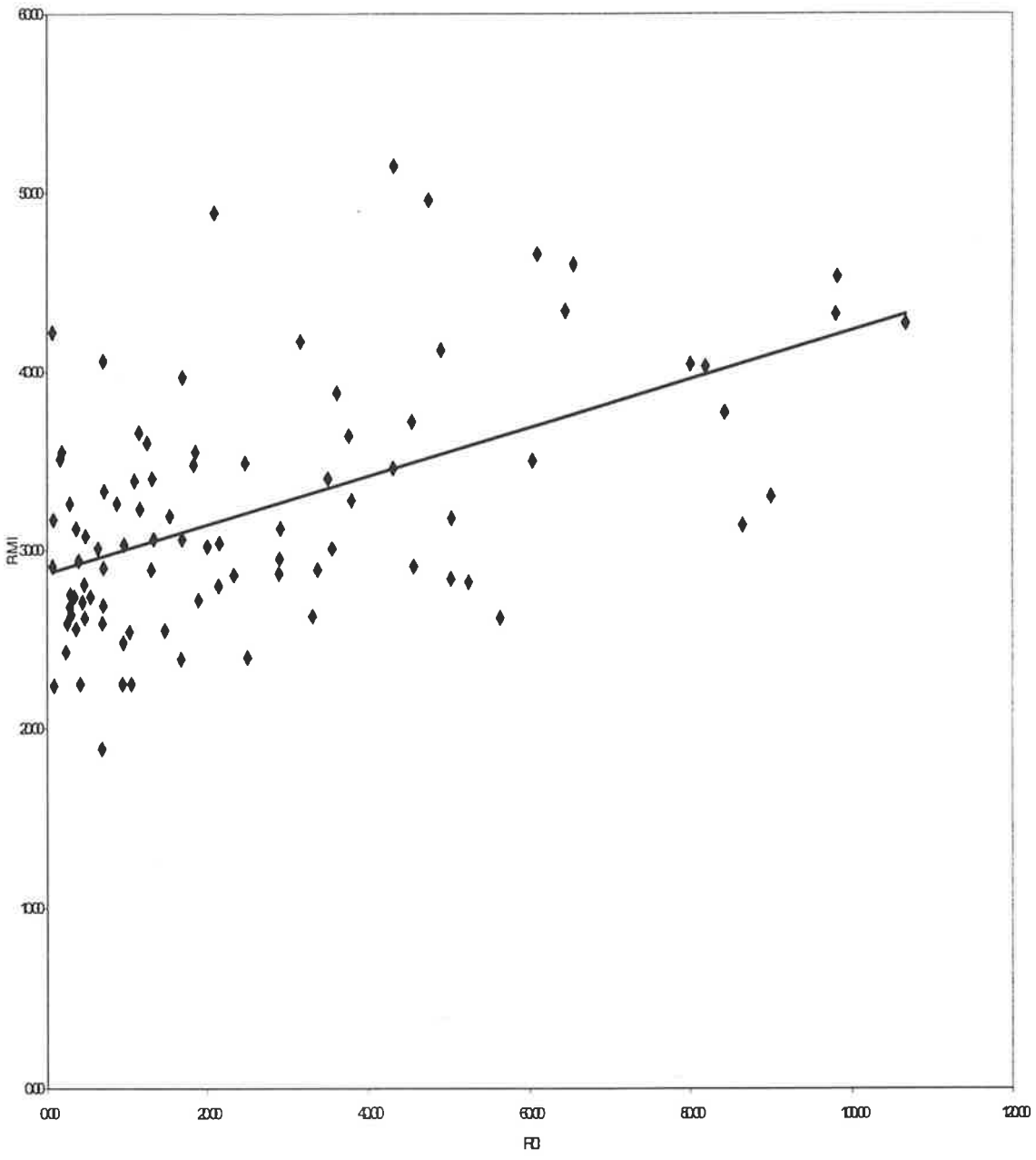


**Figure 11.4-1 BMI vs RDI**

### 11.4.2 Minimum Arterial Oxygen Saturation ($SaO_2$)

The minimum arterial oxygen saturation ($SaO_2$) ranged from a minimum of 49% to a maximum of 95% with a mean value of 80.44 +/- 1.42%. This value represents the minimum measured $SaO_2$ at any time during the polysomnographic study. The correlation co-efficient r = -0.53 was significant at the 0.01 level with 92 degrees of freedom. Figure 11.4-2 shows the relationship of $SaO_2$ to RDI with a negative correlation between the two variables indicating arterial oxygen saturation decreases as RDI increases.

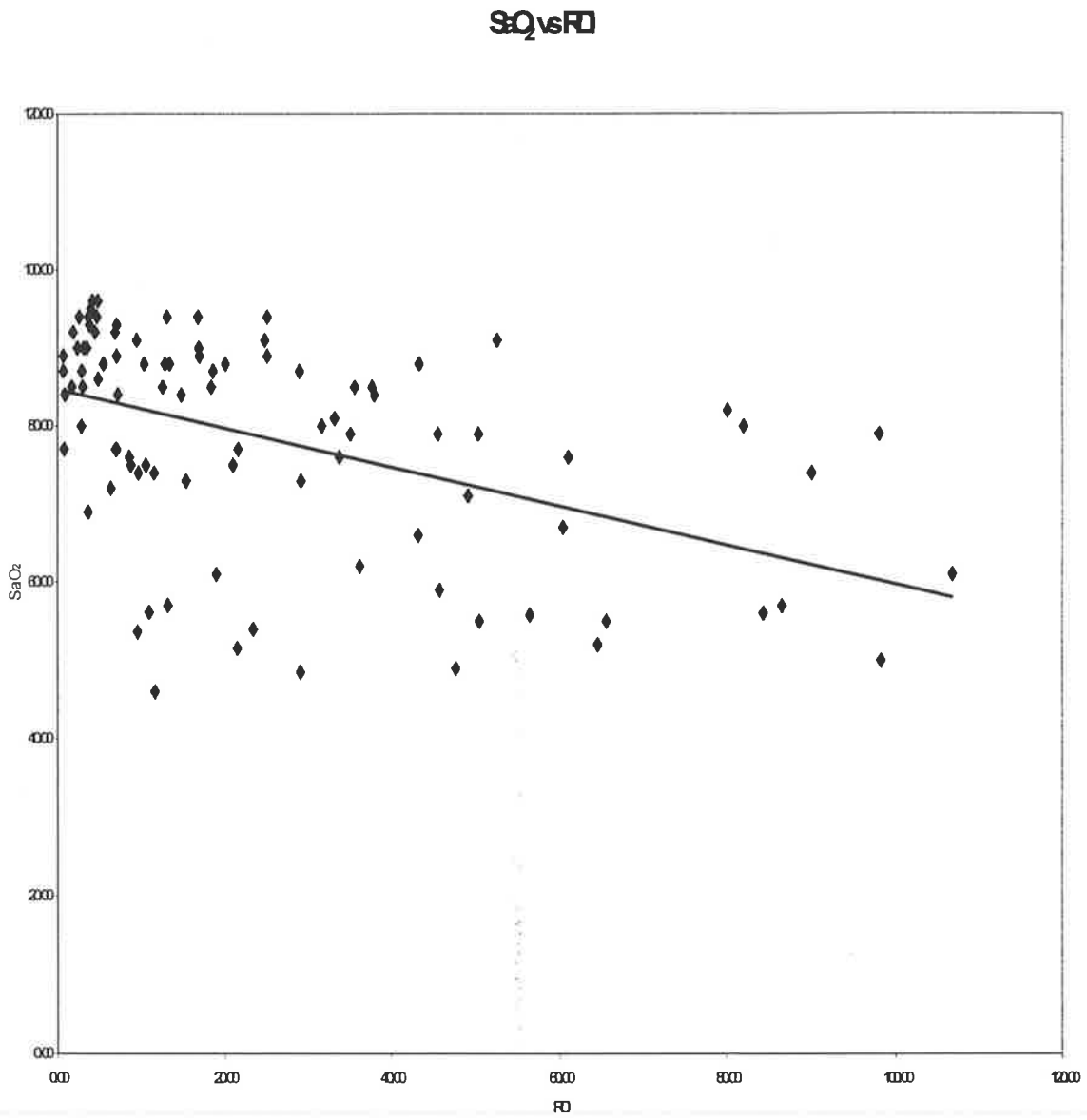


**Figure 11.4-2 $SaO_2$ vs RDI**

### 11.4.3 Neck Circumference (NC)

The neck circumference was known for only seventy two subjects for whom BMI was also known. There was a positive relationship between RDI and neck circumference. The mean neck circumference was 41.7 +/- 0.51 cm with a range from 33 cm to 51 cm. The correlation co-efficient r = 0.47 which is significant at the 0.01 level with 70 degrees of freedom. Figure 11.4-3 shows the positive relationship between neck circumference and RDI.

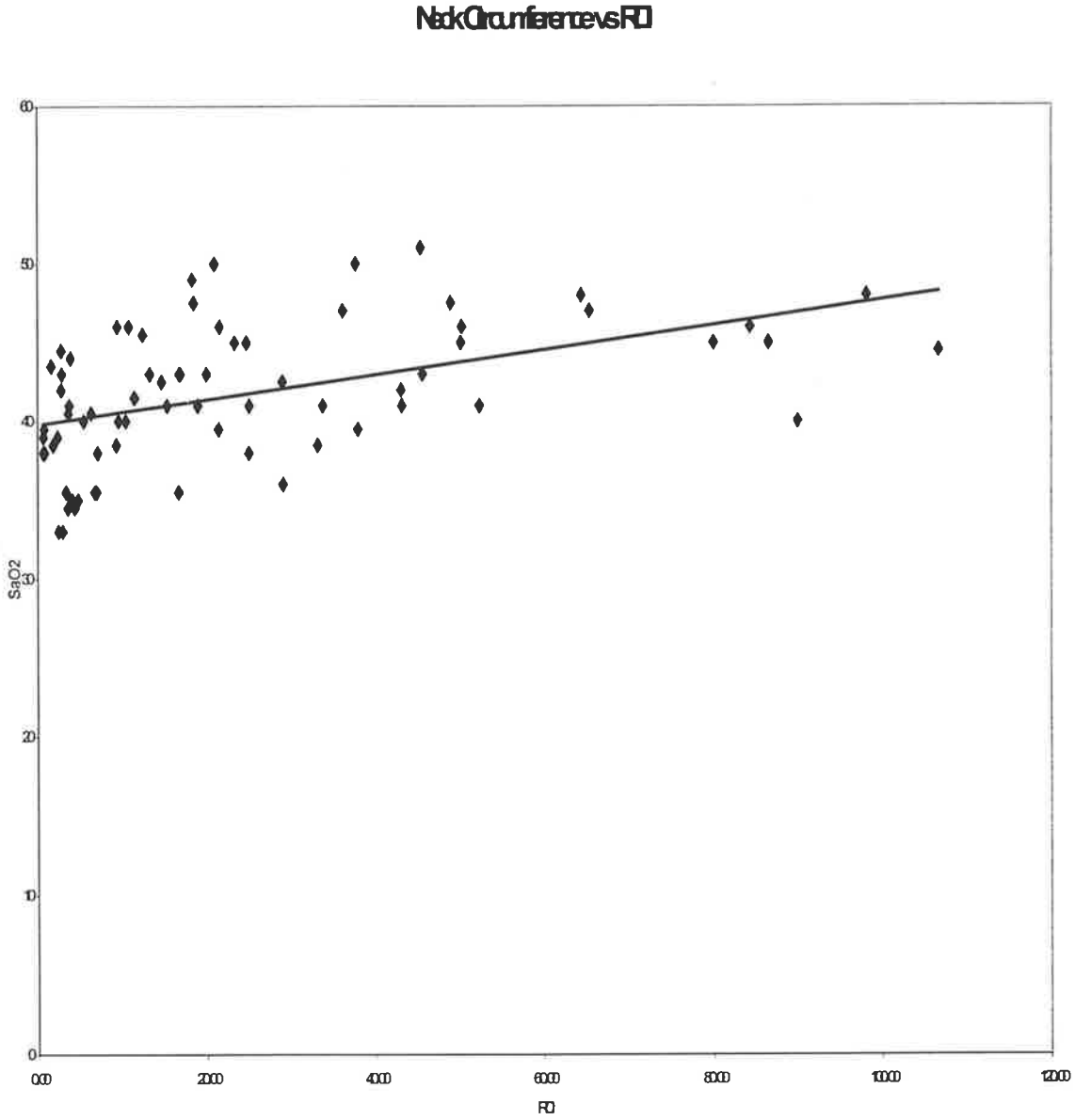


**Figure 11.4-3 NC vs RDI**

### 11.4.4 Distance from Hyoid to the Fourth Cervical Vertebra (H – C4)

The average distance from the most anterosuperior point on the hyoid bone to the most anterosuperior point on the fourth cervical vertebra (H – $C_4$) was 40.43 +/- 0.76 mm with a maximum value of 82.88 mm and a minimum value of 27.71 mm. This was significant at the 0.05 level with a correlation co-efficient of r = 0.21 and 93 degrees of freedom. The distance H-$C_4$ increases as RDI increases and this relationship is diagrammatically shown in Figure 11.4-4.

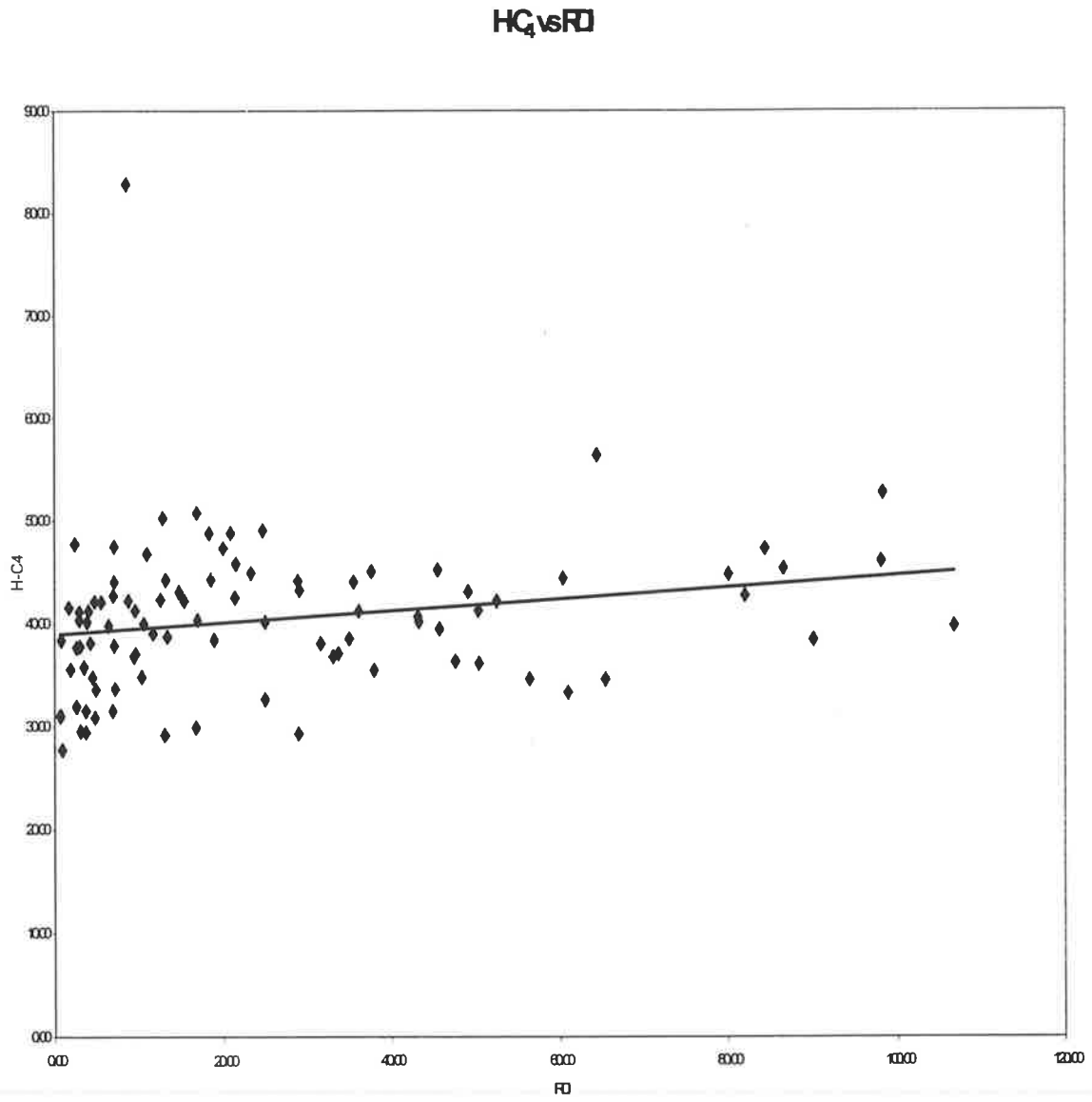


**Figure 11.4-4 H – C4 vs RDI**

### 11.4.5 Distance from Hyoid to the Third Cervical Vertebra (H – C3)

The distance from the most anterosuperior point on the hyoid bone to the most anteroinferior point on the third cervical vertebra (H – $C_3$) ranged from a minimum of 26.69 mm to a maximum of 96.24 mm. The average distance between these two points for all subjects was 43.04 +/- 1.48 mm. The correlation co-efficient r = 0.21 was significant at the 0.05 level with 93 degrees of freedom. Figure 11.4-5 shows the relationship of H-$C_3$ to RDI and indicates this distance increases as RDI increases.

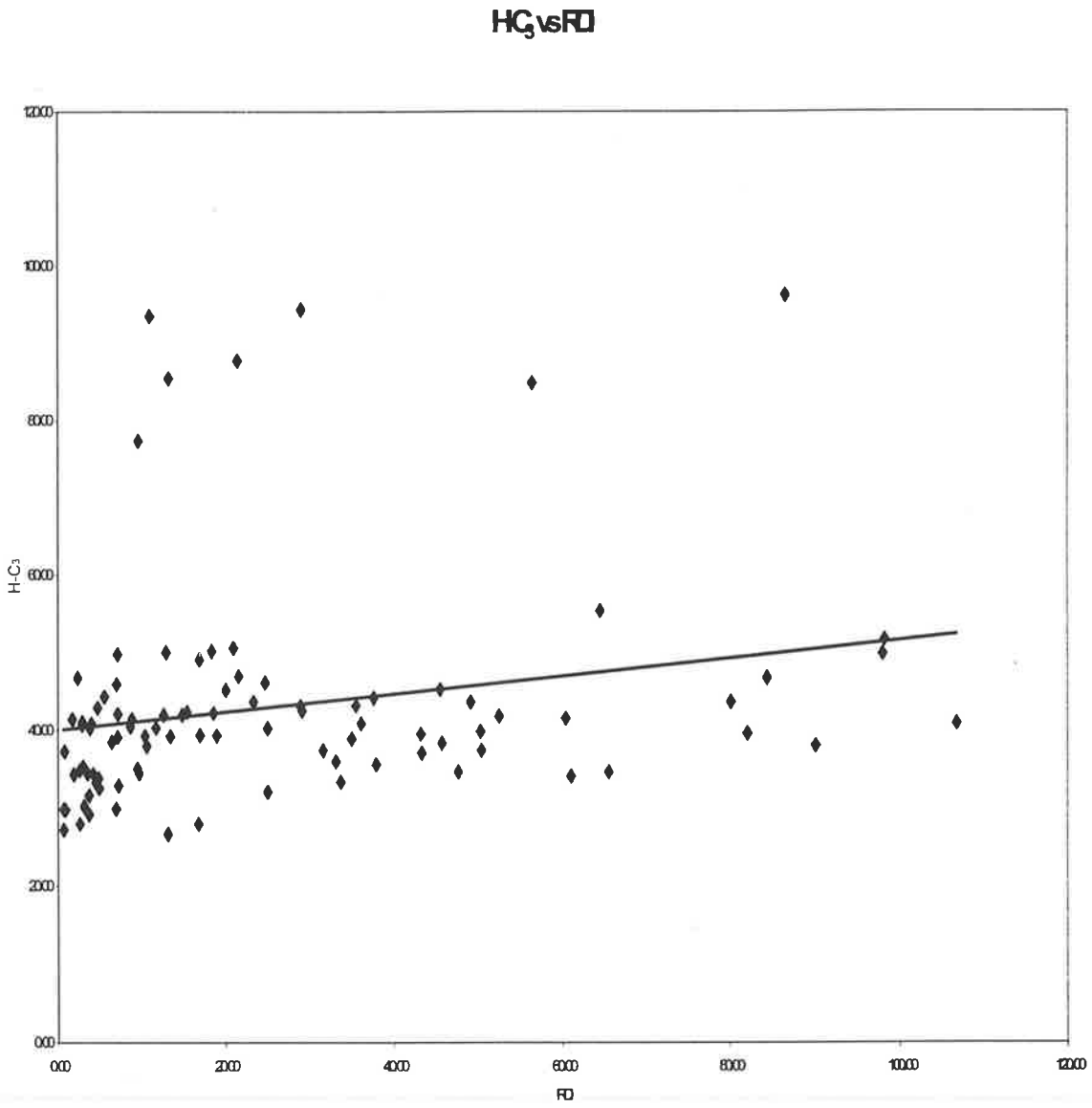


**Figure 11.4-5 H – C3 vs RDI**

### 11.4.6 Distance from the Mandibular Plane to Hyoid (MP – H)

The perpendicular distance from the most anterosuperior point on the hyoid bone to the constructed mandibular plane (MP – H) was found to be significantly correlated to RDI at the 0.05 level of significance with a correlation co-efficient r = 0.24. The mean value for this distance across all 94 subjects was 22.99 +/- 0.67 mm with a range from 2.06 mm to 36.74 mm. The distance MP-H increases as RDI increases, this relationship is shown in Figure 11.4-6.

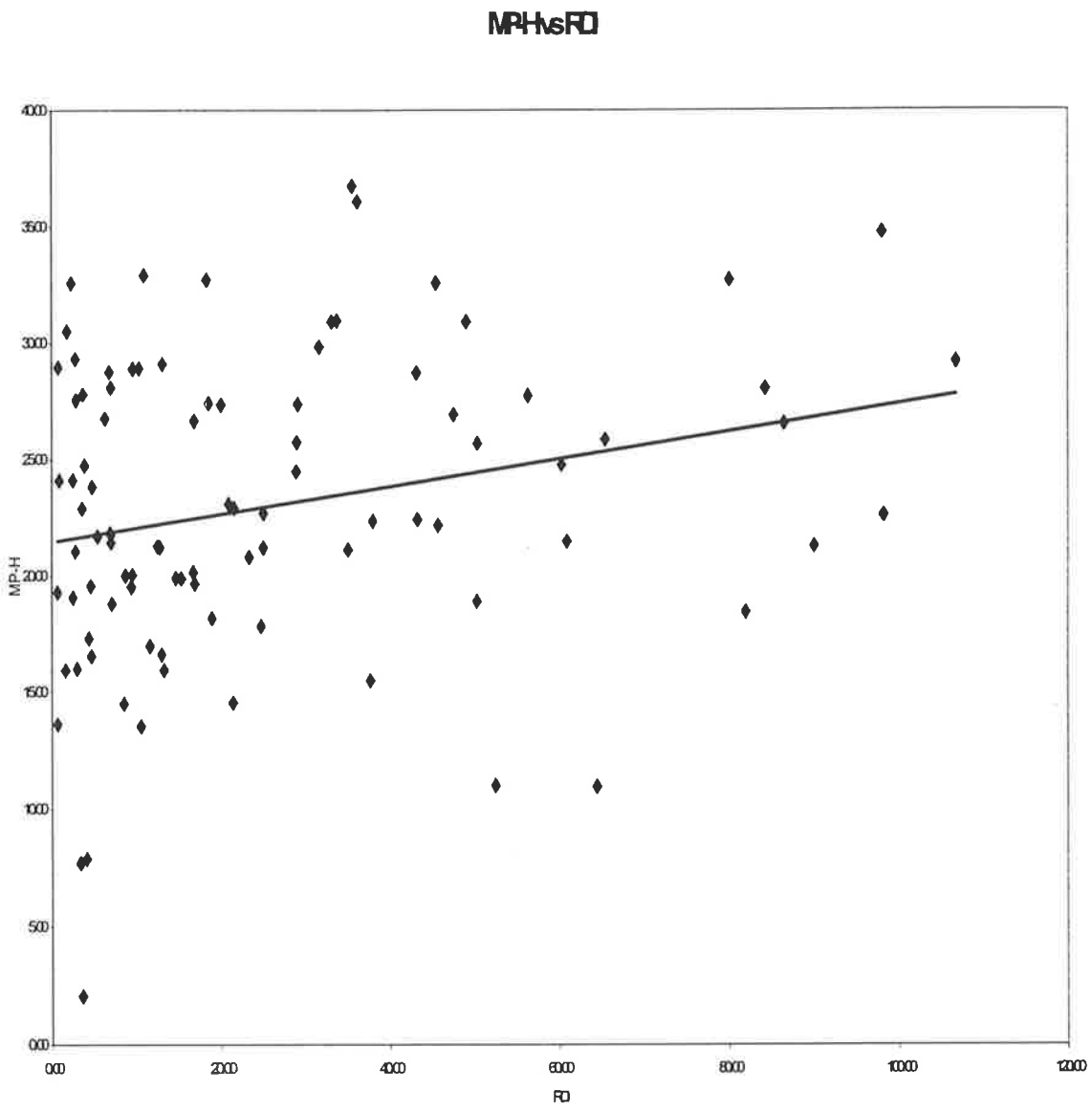


**Figure 11.4-6 MP – H vs RDI**

### 11.4.7 Pharyngeal Length (PNS – Eb)

The height of the pharynx measured from the posterior nasal spine to the base of the epiglottis (PNS – Eb) was found to correlate significantly with RDI. The mean distance between these two cephalometric landmarks was 76.15 +/- 1.66 mm. The maximum distance was 138.00 mm and the minimum 53.84 mm. The correlation co-efficient r = 0.22 was significant at the 0.05 level with 93 degrees of freedom. Figure 11.4-7 shows the relationship of pharyngeal length to RDI and indicates as pharyngeal length increases so too does RDI.

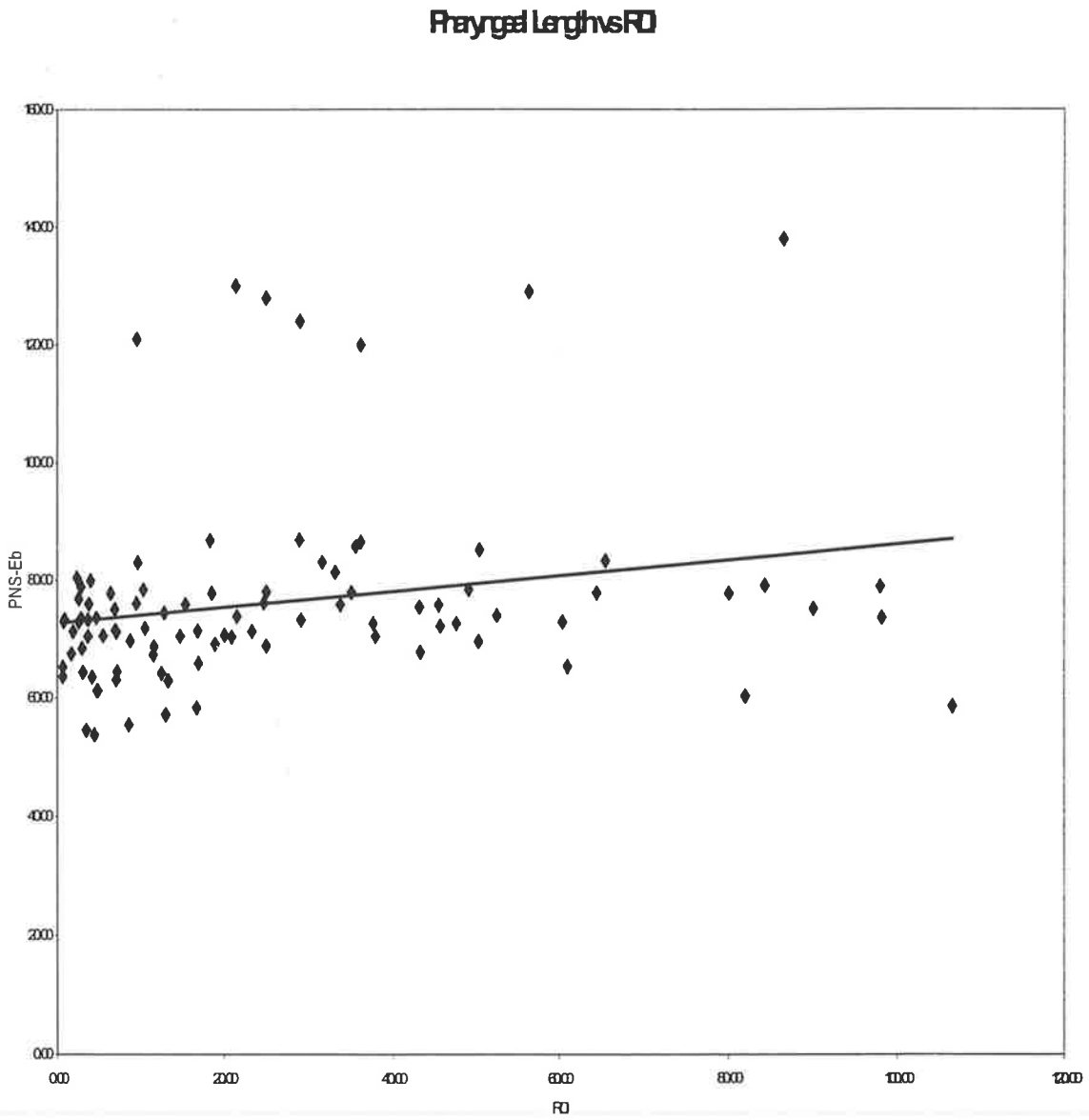


**Figure 11.4-7 PNS – Eb vs RDI**

A summary of the landmarks and measures that were significantly correlated with RDI is presented in Table 11.4-1. Hyoid position was most likely to correlate with RDI, as measured by the horizontal hyoid distance to the cervical spine and the vertical distance to the lower border of the mandible.

**Table 11.4-1: Significant Correlations Between BMI and Measured Variables**

| | Mean | SD | Min | Max | p Value |
|---|---|---|---|---|---|
| **BMI** | 32.28 kg/m$^2$ | 0.72 | 18.9 | 51.5 | 0.01 |
| **$SaO_2$** | 80.44 % | 1.42 | 49 | 95 | 0.01 |
| **NC** | 41.7 mm | 0.51 | 33 | 51 | 0.01 |
| **H – C4** | 40.43 mm | 0.76 | 27.71 | 82.88 | 0.05 |
| **H – C3** | 43.04 mm | 1.48 | 26.69 | 96.24 | 0.05 |
| **MP – H** | 22.99 mm | 0.67 | 2.06 | 36.74 | 0.05 |

## 11.5 Significant Correlations with BMI

A Pearson correlation statistical test was performed on eighty nine patients with results from an overnight polysomnographic sleep study, a cephalometric radiograph and a record of their BMI. The independent variable in the analysis was the body mass index (BMI). All cephalometric constructed linear and angular variables were correlated with BMI. Additional non-cephalometric variables considered were age, sex, RDI and neck circumference. The data is reported as significant at the $p<0.01$ or $p<0.05$ level. Results where there is no significant correlation are not reported.

### 11.5.1 Minimum Arterial Oxygen Saturation ($SaO_2$)

There was a significant negative correlation between the minimum arterial oxygen saturation ($SaO_2$) and BMI. The correlation co-efficient r = -0.43 was significant at the 0.01 level with 88 degrees of freedom. The average minimum $SaO_2$ was 79.41 +/- 1.31 % with a maximum value of 96% and a minimum value of 46%. The minimum arterial oxygen saturation decreased as RDI increased and this is illustrated diagrammatically in Figure 11.5-1.

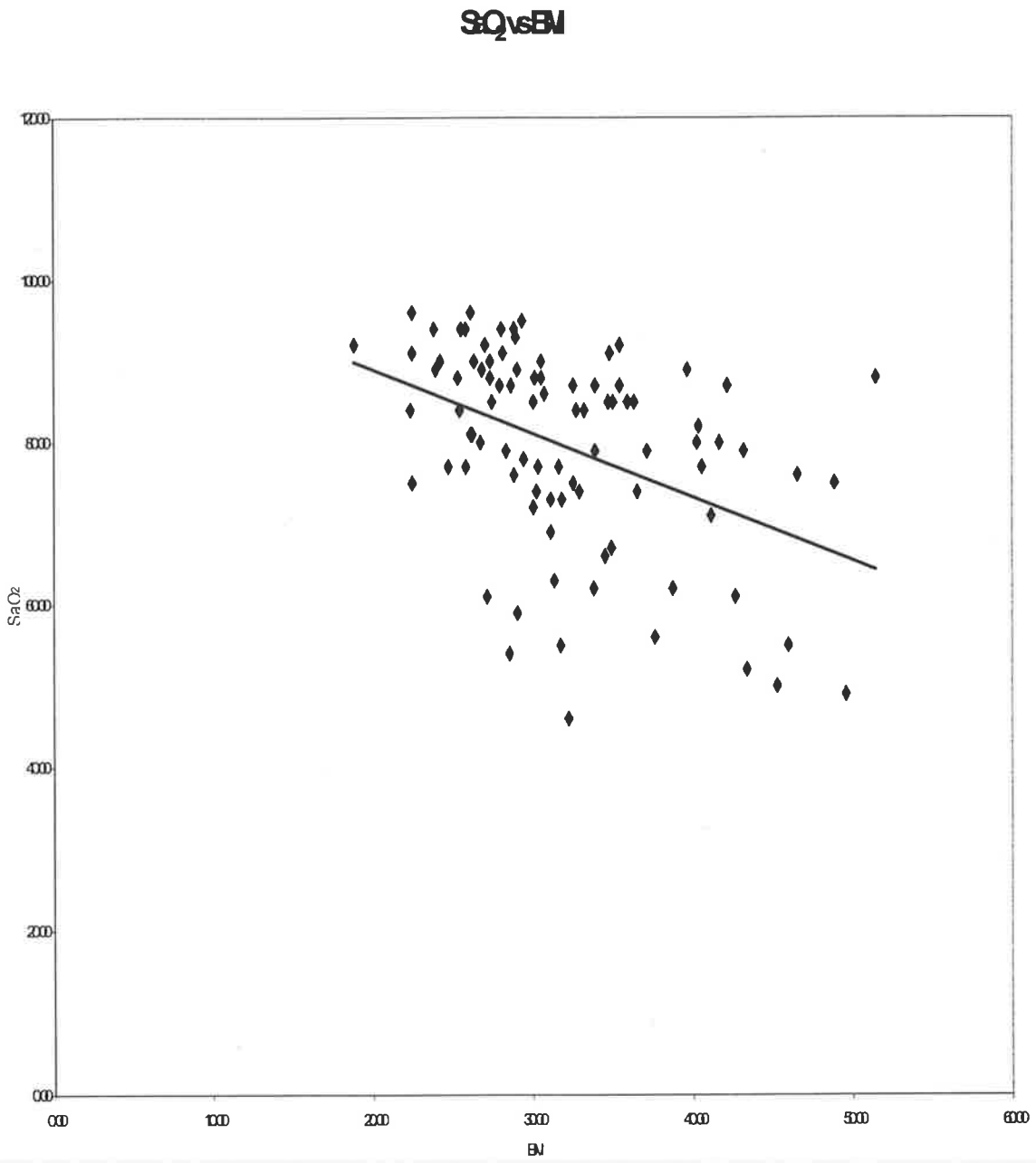


**Figure 11.5-1 $SaO_2$vs BMI**

## 11.5.2 Neck Circumference (NC)

The mean neck circumference of the total population for whom this parameter and BMI was known was 41.76 +/- 0.53 cm with a maximum value of 51 cm and a minimum value of 33cm. Seventy subjects were assessed for these two parameters. A significant correlation at the 0.01 level was found with the correlation co-efficient r = 0.59. The relationship between neck circumference and BMI was positive indicating an increasing neck circumference as BMI increased. This relationship is illustrated in Figure 11.5-2.

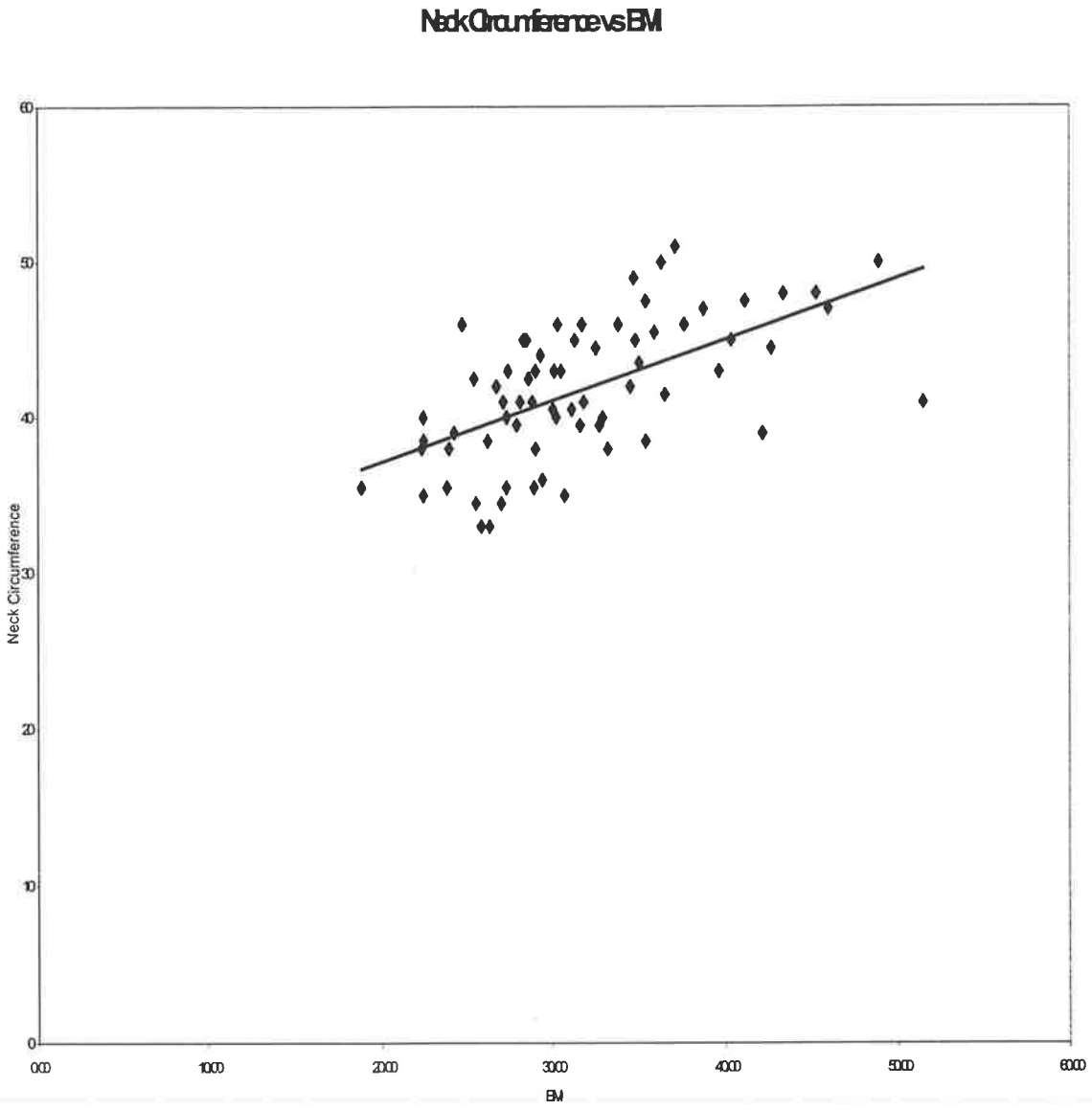


**Figure 11.5-2 NC vs BMI**

### 11.5.3 Angle Sella – Nasion – Subnasale (SNA)

The angle formed between the points sella, nasion and subspinale (S – N – A) is a measure of the anteroposterior position of the maxilla with respect to the cranial base. The mean measure of this angle was 83.83 +/- 0.55 degrees with a range of values from 70.70 degrees to 94.98 degrees. The correlation co-efficient r = 0.21 was significant at the 0.05 level with 88 degrees of freedom. This angle increased as BMI increased and is illustrated diagrammatically in Figure 11.5-3.

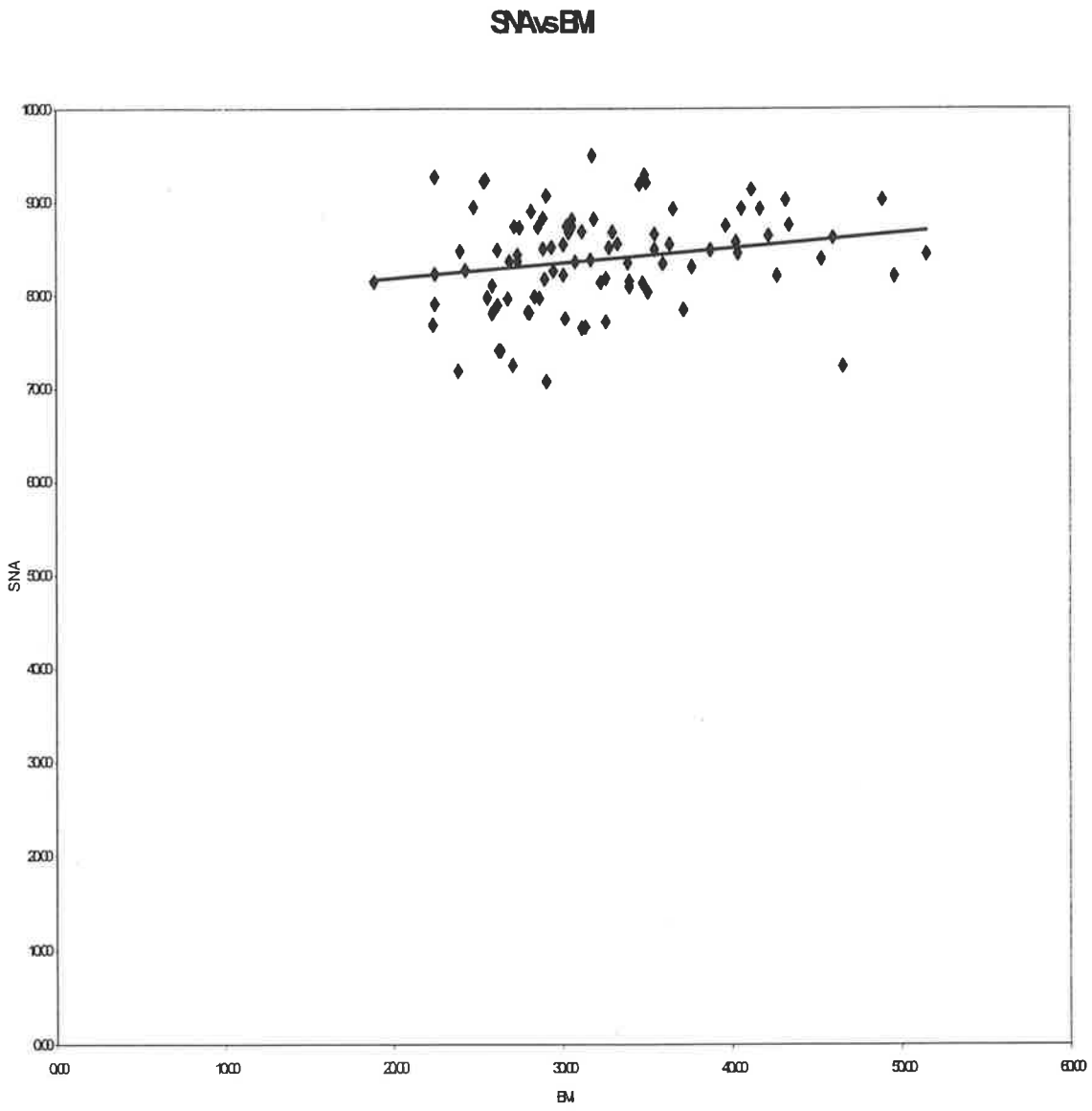


**Figure 11.5-3 SNA vs BMI**

### 11.5.4 Angle Sella – Nasion – Supramentale (SNB)

The angle formed between the points sella, nasion and supramentale (S – N – A) was significantly correlated with BMI. This angle is a measure of the anteroposterior position of the mandible with relation to the cranial base. The mean angle formed between these three points was 83.12 +/- 0.53 degrees with a maximum value of 93.5 degrees and a minimum value of 67.17 degrees. The correlation co-efficient r = 0.23 is significant at the 0.05 level at 88 degrees of freedom and indicates an increasing angle as BMI increases. Figure 11.5-4 illustrates this significant correlation.

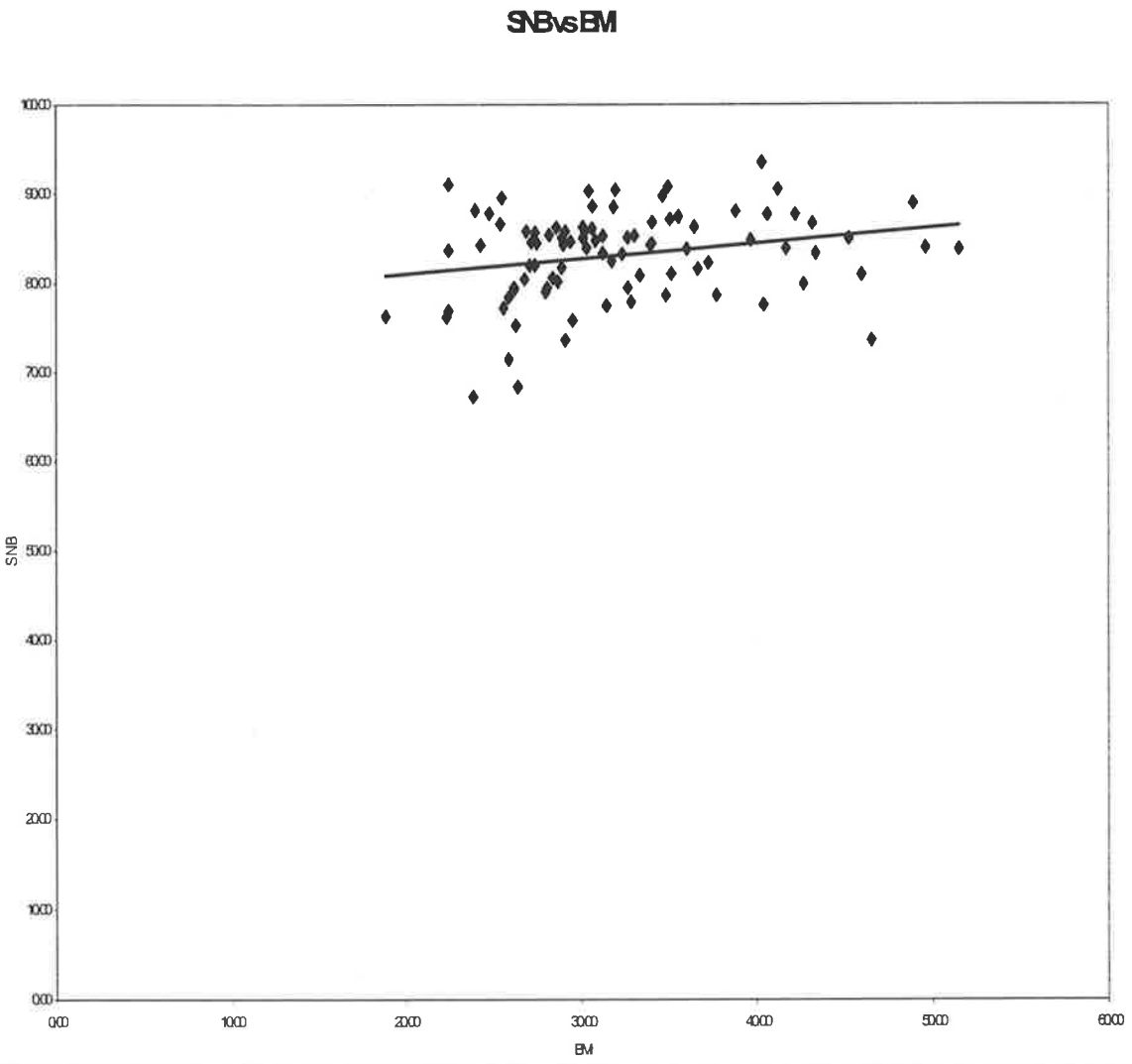


**Figure 11.5-4 SNB vs BMI**

### 11.5.5 Upper Incisal Angulation (U1 – SN)

The angle formed between the upper central incisor and the cranial base (U1 – SN) was significantly correlated with BMI. The correlation co-efficient r = 0.23 is significant at the 0.05 level with 66 degrees of freedom. A number of subjects did not have their upper central incisor teeth which explains the smaller number of subjects in this population grouping. The average upper central incisor angulation was 106.78 +/- 1.01 degrees with a minimum angulation of 64.25 degrees and a maximum of 123.02 degrees. Figure 11.5-5 shows the positive correlation between upper central incisor angulation and BMI.

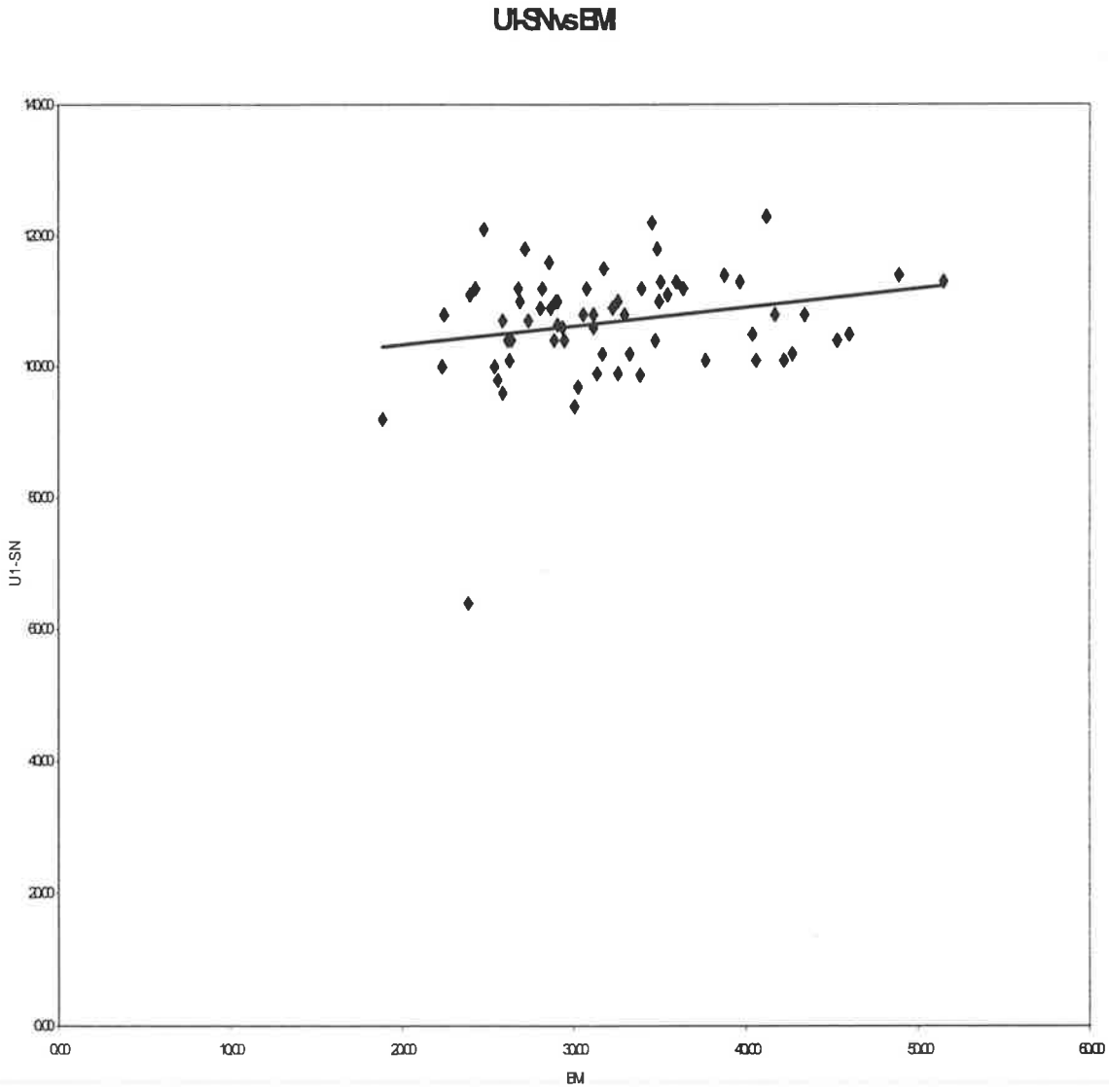


**Figure 11.5-5 U1 – SN vs BMI**

### 11.5.6 Distance from Hyoid to the Fourth Cervical Vertebra (H – C4)

The average distance from the most anterosuperior point on the hyoid bone to the most anterosuperior point on the fourth cervical vertebra (H – $C_4$) was 40.41 +/- 0.78 mm with a maximum value of 81.07 mm and a minimum value of 27.71 mm. This was significant at the 0.01 level with a correlation co-efficient of r = 0.32 and 88 degrees of freedom. The distance H-$C_4$ increases as RDI increases and this relationship is diagrammatically shown in Figure 11.5-6.

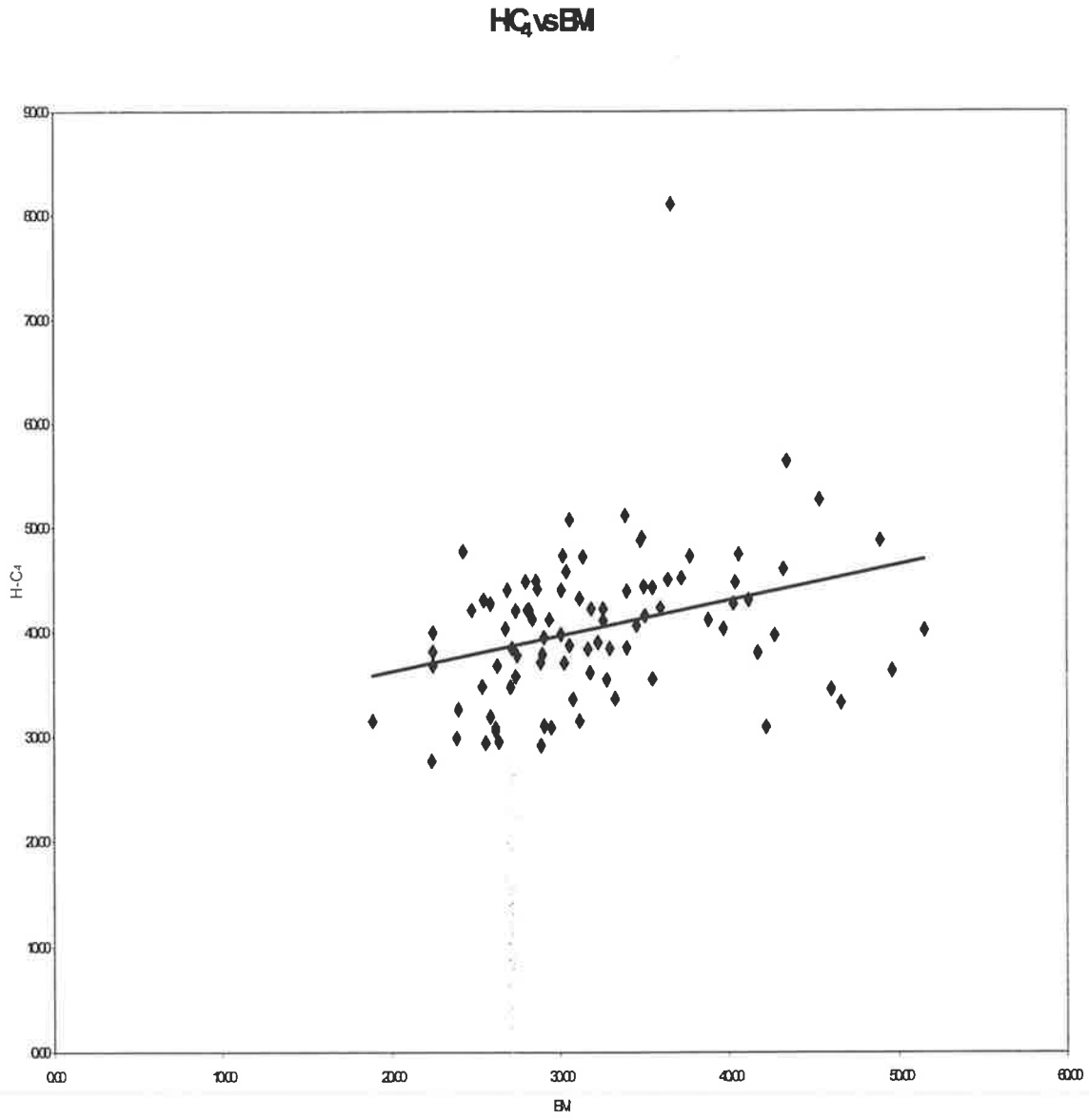


**Figure 11.5-6 H – C4 vs BMI**

### 11.5.7 Distance from Hyoid to the Third Cervical Vertebra (H – C3)

The distance from the most anterosuperior point on the hyoid bone to the most anteroinferior point on the third cervical vertebra (H – $C_3$) ranged from a minimum of 26.69 mm to a maximum of 81.80 mm. The average distance between these two points for all subjects was 39.85 +/- 0.80 mm. The correlation co-efficient r = 0.32 was significant at the 0.01 level with 88 degrees of freedom. Figure 11.5-7 shows the relationship of H-$C_3$ to RDI and indicates this distance increases as RDI increases.

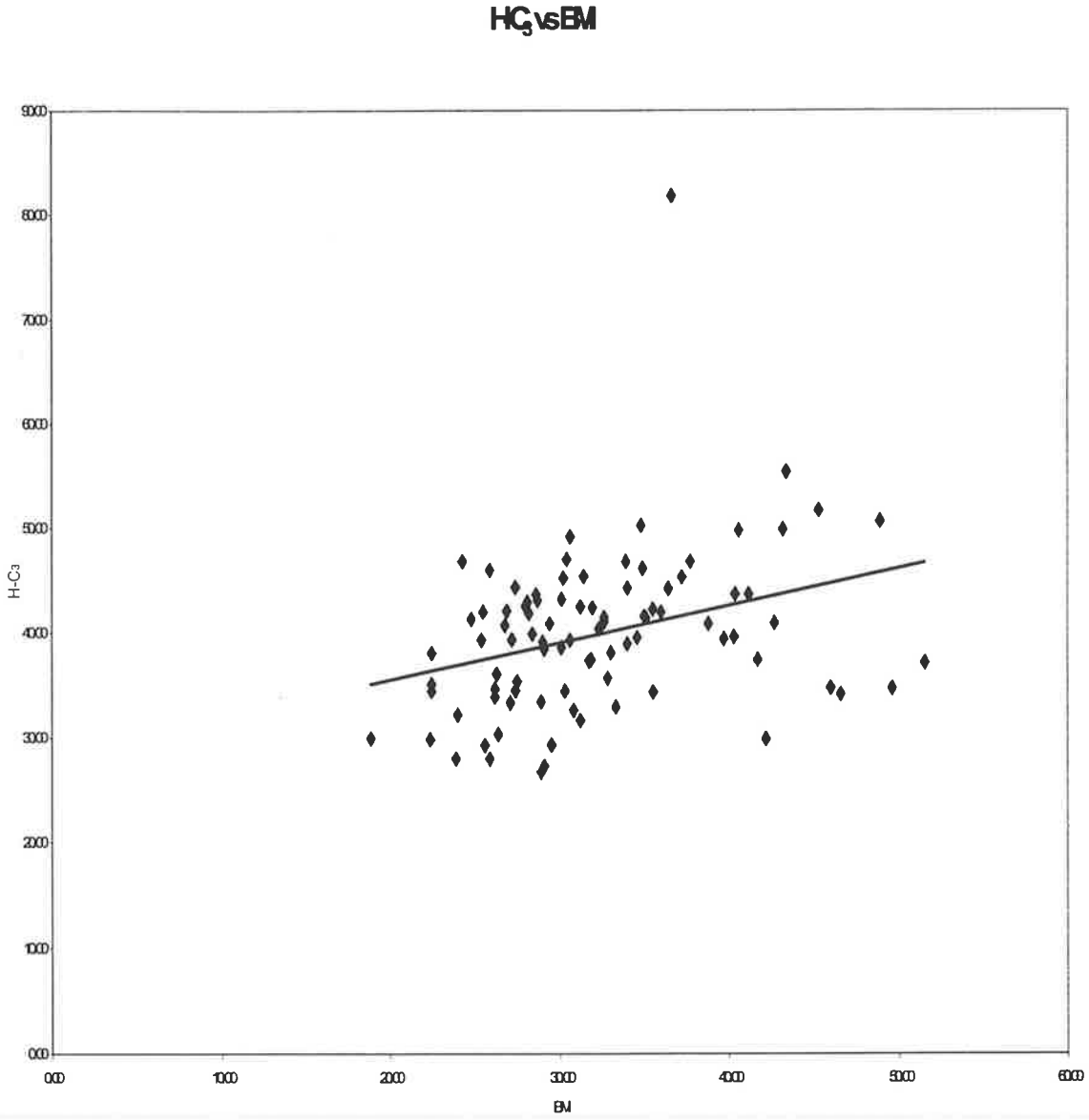


**Figure 11.5-7 H – C3 vs BMI**

## 11.5.8 Distance from Hyoid to Menton (H – Me)

The perpendicular distance from the most anterosuperior point on the hyoid bone to the menton (Me – H) was found to be significantly correlated to RDI at the 0.05 level of significance with a correlation co-efficient r = 0.23. The mean value for this distance across all 90 subjects was 44.80 +/- 0.74 mm with a range from 30.42 mm to 64.59 mm. The distance Me-H increases as RDI increases, this relationship is shown in Figure 11.5-8.

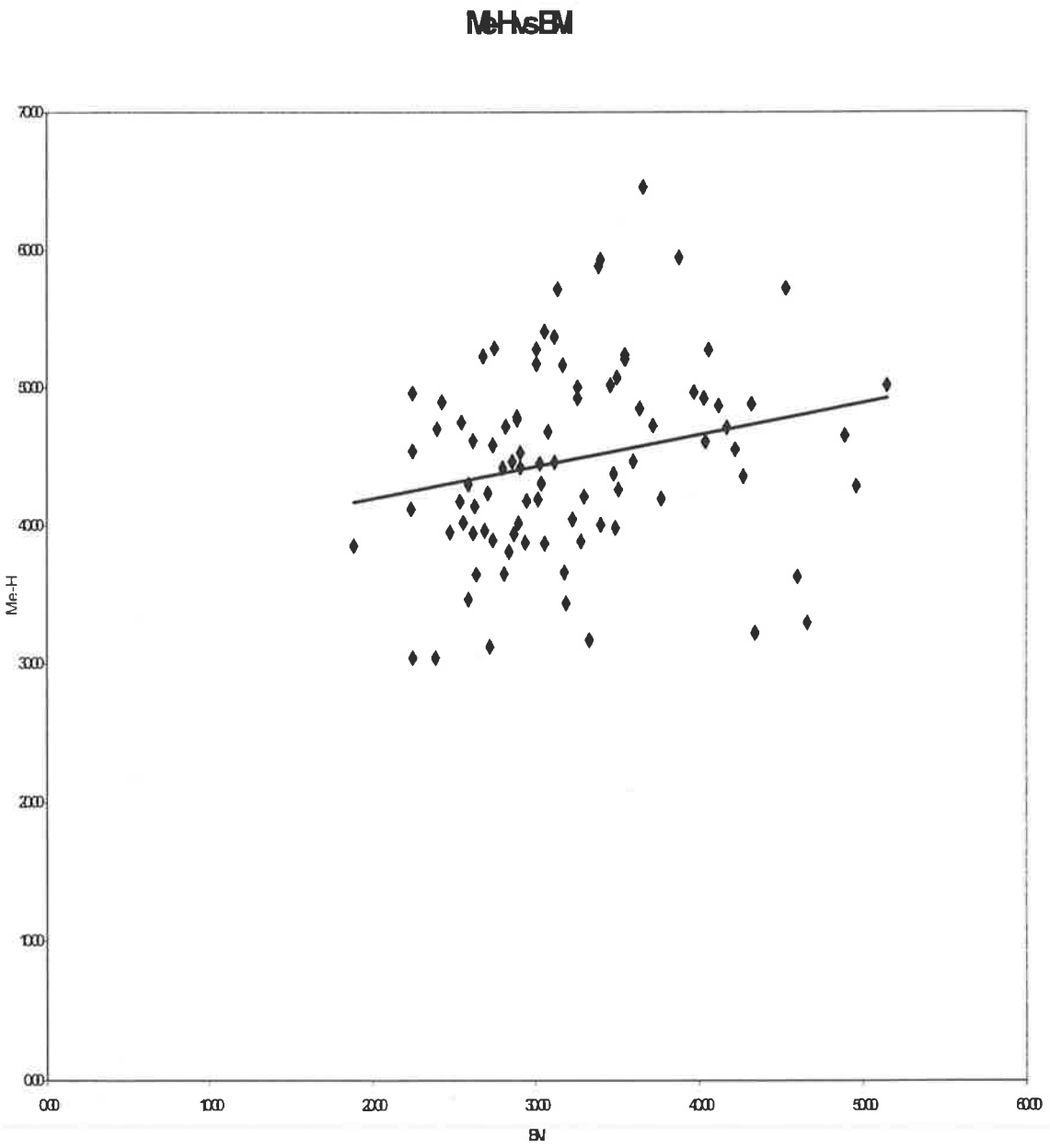


**Figure 11.5-8 Me – H vs BMI**

Table 11.5-1 summarises the variables that were found to significantly correlate with BMI across the whole sample.

**Table 11.5-1: Significant Correlations Between BMI and Measured Variables**

| | Mean | SD | Min | Max | p Value |
|---|---|---|---|---|---|
| $SaO_2$ | 79.41 % | 1.31 | 46 | 96 | 0.01 |
| NC | 41.76 mm | 0.53 | 33 | 51 | 0.01 |
| SNA | $83.83^0$ | 0.55 | 70.70 | 94.98 | 0.05 |
| SNB | $83.12^0$ | 0.53 | 67.17 | 93.5 | 0.05 |
| U1 – SN | $106.78^0$ | 1.01 | 64.25 | 123.02 | 0.05 |
| H – C4 | 40.41 mm | 0.78 | 27.71 | 81.07 | 0.01 |
| H – C3 | 39.85 mm | 0.80 | 26.69 | 81.80 | 0.01 |
| H – Me | 44.8 mm | 0.74 | 30.42 | 64.59 | 0.05 |

## 11.6 Group Differences With Cut-off RDI 10

The following analyses were performed following the division of the population into two groups. These groups were determined by the RDI recorded for an individual subject in their polysomnographic study. Those subjects with an RDI < 10 will be referred to as Type I Snorers and those with an RDI > 10 as Type I OSA subjects.

The two groups did not contain 50% of the sample each, with Type I Snorers comprising 35 subjects and Type I OSA subjects numbering 59. This difference was not enough to preclude the use of a two tail t – test assuming equal variance. Statistical analysis was performed on the same variables as for the Pearson correlation (Section 11.2 and Section 11.3) and additionally sex and age distribution was considered for the two groups.

Only those measures that were statistically significant at the $p<0.01$ or $p<0.05$ level of significance will be discussed. The variables that are significantly different between the two groups of subjects are reported in Table 11.6-2.

### 11.6.1 Sex

Division of the Type I Snorers and Type I OSA subjects on the basis of sex is presented in Table 11.6-1. A Pearson's chi-square test with Yates' continuity correction gives a

significance of p=0.0255. There is a strong correlation between group and sex with only 15% of the OSA group being female but 40% of the snoring group being female.

**Table 11.6-1: Sex of study patients separated by RDI = 10**

| | Sex | |
|---|---|---|
| | Female | Male |
| OSA | 10 | 49 |
| Snorer | 14 | 21 |

### 11.6.2 Age

Analysis of the age of the subjects in the Type I Snorer and Type I OSA groups shows a significant difference between the two. A standard two sample t – test gives p – value of 0.0074 for the difference in the mean age of the two samples. The subjects in the Type I snoring group on average 6.55 years younger than the subjects comprising the Type I OSA group. The mean age of the subjects with an RDI < 10 is 45.74 years, whilst the mean age of patients with an RDI > 10 is 52.29 years.

### 11.6.3 Body Mass Index (BMI)

The difference in BMI between the two groups was significant at the 1% level with a p-value < 0.000. Thirty two subjects were in the Type I snoring group and had a mean BMI of 28.87kg/m$^2$, whilst the Type I OSA group had a mean BMI of 34.27 kg/m$^2$.

### 11.6.4 Minimum Arterial Oxygen Saturation ($SaO_2$)

The average minimum $SaO_2$ for the Type I snoring group was 85.94% whilst the average minimum $SaO_2$ for the Type I OSA group was 76.37%. This was significantly different with a p-value < 0.000.

### 11.6.5 Neck Circumference (NC)

The neck circumference was significantly smaller in the Type I snoring group compared with the Type I OSA group. The average neck circumference in the Type I snoring group was 38.75 cm whilst in the Type I OSA group it was 43.74 cm. This difference was highly significant with p<0.000.

### 11.6.6 Distance Hyoid to Fourth Cervical Vertebra (H – C4)

The distance from the most anterosuperior point on the hyoid bone to the most anterosuperior point on the fourth cervical vertebra was significantly different between Type I snoring subjects and those with Type I OSA (p=0.001). The average distance from hyoid to the fourth cervical vertebra was 37.66 mm in the Type I snoring group whilst this distance was 41.69 mm in the Type I OSA group.

### 11.6.7 Distance Hyoid to Third Cervical Vertebra (H – C3)

The distance from the most anteroinferior point on the body of the third cervical vertebra to the most anterosuperior point on the body of the hyoid bone was significantly less in the Type I snoring group compared with the Type I OSA group (p=0.001). The average distance from the hyoid to the third cervical vertebra was 36.91 mm in the Type I snoring group, whilst the Type I OSA group had an average distance of 41.12 mm.

### 11.6.8 Distance Sella to Hyoid (S- H)

The distance from sella to hyoid, which is a measure of the height of the pharynx, was less in the Type I snoring group than the Type I OSA group (p=0.003). The average distance for this dimension in the Type I snoring group was 110.37 mm whilst in the Type I OSA group it was 116.06 mm.

### 11.6.9 Soft Palate Thickness (UW1 – UW2)

The thickness of the soft palate at its widest point was significantly smaller in the Type I snoring group compared with the subjects with Type I OSA (p=0.008). The average thickness of the soft palate's widest point was 9.81 mm in the Type I snoring group, whilst in the Type I OSA group this measurement was 10.83 mm.

### 11.6.10 Distance Articulare to Hyoid (Ar – H)

The distance from point articulare on the mandibular condyle to the most anterosuperior point on the body of the hyoid was significantly greater in subjects with Type I OSA compared to subjects in the Type I snoring group (p=0.017). This distance is a measure of the height of the bone pharynx. The average distance in the Type I OSA group was 89.20 mm whilst in the Type I snoring group this distance averaged 84.68 mm.

### 11.6.11 Maxillary Length (MxUL)

The maxilla was shorter in its anteroposterior dimension in patients who snored compared with patients who were diagnosed with Type I OSA (p=0.030). The average maxillary length in the Type I snoring group was 50.99 mm whilst the average length in Type I OSA patients was 52.74 mm.

### 11.6.12 Distance Mandibular Plane to Hyoid (MP – H)

The perpendicular distance from the constructed mandibular plane to the most anterosuperior point on the hyoid bone was significantly less in Type I snoring patients compared to those with Type I OSA (p=0.052). The average distance from the mandibular plane to hyoid was 21.37 mm in Type I snoring subjects, whilst those with Type I OSA had an average distance of 24.07 mm.

### 11.6.13 Distance Gonion to Hyoid (Go – H)

The distance from point gonion at the mandibular angle to the most anterosuperior point on the body of the hyoid bone was significantly less in patients who snored compared with patients with Type I OSA (p=0.052). The average distance from gonion to the hyoid bone was 39.44 mm in subjects in the Type I snoring group compared with those subjects in the Type I OSA group who had an average distance of 42.00 mm.

### 11.6.14 Hyoid Angle (<H – H1)

The angle between the most anterosuperior point on the body of the hyoid, point retrognathion on the mandibular symphysis and point gonion at the mandibular angle was significantly more acute in patients who snored compared with patients in the Type I OSA group (p=0.047). The average measure for this angle in the Type I snoring group was 16.90° whilst in the Type I OSA group the average for this angle was 20.86°.

### 11.6.15 Pharyngeal Length (VAL)

The length of the pharynx, as measured from the posterior nasal spine to the base of the epiglottis, was significantly shorter in patients who snore compared to those with Type I OSA (p=0.054). The average distance for subjects in the Type I snoring group was 70.13 mm compared to 73.11 mm in those subjects in the Type I OSA group.

**Table 11.6-2: Significant Variables with Group Separation RDI > 10**

| | Mean | | Standard Deviation | | Maximum | | Minimum | | p Value |
|---|---|---|---|---|---|---|---|---|---|
| | Snorer | OSA | Snorer | OSA | Snorer | OSA | Snorer | OSA | |
| BMI | 28.87 | 34.27 | 5.01 | 7.04 | 42.2 | 51.5 | 18.9 | 22.5 | 0.00 |
| SaO2 | 85.94 | 76.37 | 7.59 | 13.17 | 96 | 94 | 69 | 46 | 0.00 |
| NC | 38.75 | 43.74 | 3.57 | 3.76 | 46 | 51 | 33 | 35.5 | 0.00 |
| H - C4 | 37.66 | 41.69 | 5.17 | 5.88 | 47.74 | 56.38 | 27.71 | 29.18 | 0.00 |
| H - C3 | 36.91 | 41.12 | 5.78 | 5.82 | 49.77 | 55.41 | 27.25 | 26.69 | 0.00 |
| S - H | 110.37 | 116.06 | 8.29 | 8.79 | 124.6 | 129.53 | 94.01 | 93.06 | 0.00 |
| UW1 - UW2 | 9.81 | 10.83 | 1.63 | 1.83 | 13.67 | 15.93 | 6.5 | 6.7 | 0.01 |
| Ar - H | 84.68 | 89.20 | 7.80 | 9.17 | 98.32 | 102.82 | 69.7 | 58.37 | 0.02 |
| MxUL | 50.99 | 52.74 | 2.92 | 4.11 | 57.76 | 63.59 | 44.24 | 43.06 | 0.03 |
| MP - H | 21.37 | 24.07 | 6.81 | 6.13 | 32.57 | 36.74 | 2.06 | 10.96 | 0.05 |
| Go - H | 39.44 | 42.00 | 6.13 | 6.01 | 51.17 | 51.92 | 25.32 | 27.59 | 0.05 |
| <H - H1 | 16.90 | 20.86 | 9.41 | 9.04 | 34.62 | 39.31 | -10.96 | 0.24 | 0.05 |
| VAL | 70.13 | 73.11 | 7.12 | 7.12 | 82.97 | 86.9 | 53.84 | 57.27 | 0.05 |

## 11.7 Group differences with cut-off RDI 15

The following analyses were performed following the division of the population into two groups. These groups were determined by the RDI recorded for an individual subject in their polysomnographic study. Those subjects with an RDI < 15 will be referred to as Type II snorers and those with an RDI > 15 as Type II OSA subjects.

The two groups contained approximately 50% of the sample each, with snorers comprising 45 subjects and OSA subjects numbering 49. Statistical analysis was performed on the same variables as for the Pearson correlation (Section 10.2 and Section 10.3) and additionally sex and age distribution was considered for the two groups.

Only those measures that were statistically significant at the 0.01 or 0.05 level of significance will be discussed. The variables that are significantly different between the two groups of patients are reported in Table 11.7-2.

### 11.7.1 Sex

Division of the Type II snoring and Type II OSA subjects on the basis of sex is presented in Table 11.7-1. A Pearson's chi-square test with Yates' continuity correction gives a significance of p=0.0255. There is a strong correlation between group and sex with 15% of the Type II OSA group being female but 40% of the Type II snoring group being female.

**Table 11.7-1: Sex of patients separated by RDI = 15**

| | Sex | |
|---|---|---|
| | Female | Male |
| OSA | 9 | 40 |
| Snorer | 15 | 30 |

### 11.7.2 Age

Analysis of the age of the subjects in the Type II snoring and Type II OSA groups shows a significant difference between the two. A standard two sample t – test gives p – value of 0.051 for the difference in the mean age of the two groups. The subjects in the Type II snoring group on average 6.55 years younger than the subjects comprising the Type II OSA

group. The mean age of the patients with an RDI < 15 (snorer) is 47.42 years, whilst the mean age of patients with an RDI > 15 (Type II OSA) is 52.08 years.

### 11.7.3 Body Mass Index (BMI)

The difference in BMI between the two groups was significant at the 0.01 level with a p-value of 0.000. Forty one subjects were in the Type II snoring group and had a mean BMI of 29.10 $kg/m^2$, whilst the Type II OSA group had a mean BMI of 35.10 $kg/m^2$.

### 11.7.4 Minimum Arterial Oxygen Saturation ($SaO_2$)

The average minimum $SaO_2$ for the Type II snoring group was 84.56% whilst the average minimum $SaO_2$ for the Type II OSA group was 75.35%. This was significantly different with a p-value of 0.000.

### 11.7.5 Neck Circumference (NC)

The neck circumference was significantly smaller in the Type II snoring group compared with the Type II OSA group. The average neck circumference in the Type II snoring group was 39.45 cm whilst in the Type II OSA group it was 43.74 cm. This difference was highly significant with p=0.000.

### 11.7.6 Distance Hyoid to Fourth Cervical Vertebra (H – C4)

The distance from the most anterosuperior point on the hyoid bone to the most anterosuperior point on the fourth cervical vertebra was significantly different between Type II snoring subjects and those with Type II OSA (P=0.007). The average distance from hyoid to the fourth cervical vertebra was 38.45 mm in the Type II snoring group whilst this distance was 41.78 mm in the Type II OSA group.

### 11.7.7 Distance Hyoid to Third Cervical Vertebra (H – C3)

The distance from the most anteroinferior point on the body of the third cervical vertebra to the most anterosuperior point on the body of the hyoid bone was significantly less in the Type

II snoring group compared with the Type II OSA group (p=0.006). The average distance from the hyoid to the third cervical vertebra was 37.79 mm in the Type II snoring group, whilst the Type II OSA group had an average distance of 41.18 mm.

### 11.7.8 Pharyngeal Length (VAL)

The length of the pharynx, as measured from the posterior nasal spine to the base of the epiglottis, was significantly shorter in patients who snore compared to those with Type II OSA (p=0.007). The average distance for subjects in the Type II snoring group was 69.86 mm compared to 73.99 mm in those subjects in the Type II OSA group.

### 11.7.9 Distance Sella to Hyoid (S – H)

The distance from sella to hyoid, which is a measure of the height of the pharynx, was less in the Type II snoring group than the Type II OSA group (p=0.008). The average distance for this dimension in the Type II snoring group was 111.32 mm whilst in the Type II OSA group it was 116.35 mm.

### 11.7.10 Tongue Length (TGL)

Tongue length as measured from the tip of the tongue to the base of the epiglottis (vallecula) was significantly shorter in Type II snoring subjects than in those subjects with Type II OSA. The mean tongue length for the Type II snoring group was 75.51 cm whilst the mean length was 79.64 cm in the Type II OSA subjects. This difference was significant at the 0.01 level of significance with p=0.010.

### 11.7.11 Soft Palate Thickness (UW1 – UW2)

The thickness of the soft palate at its widest point was significantly smaller in the Type II snoring group compared with the subjects with Type II OSA (p=0.018). The average thickness of the soft palate's widest point was 10.02 mm in the Type II snoring group, whilst in the Type II OSA group this measurement was 10.85 mm.

### 11.7.12 Hyoid Angle (<H – H1)

The angle between the most anterosuperior point on the body of the hyoid, point retrognathion on the mandibular symphysis and point gonion at the mandibular angle was

significantly more acute in patients who snored compared with patients in the Type II OSA group (p=0.029). The average measure for this angle in the Type II snoring group was 16.98° whilst in the Type II OSA group the average for this angle was 21.61°.

### 11.7.13 Distance Mandibular Plane to Hyoid (MP – H)

The perpendicular distance from the constructed mandibular plane to the most anterosuperior point on the hyoid bone was significantly less in Type II snoring patients compared to those with Type II OSA (p=0.032). The average distance from the mandibular plane to hyoid was 21.43 mm in Type II snoring subjects, whilst those with Type II OSA had an average distance of 24.58 mm.

### 11.7.14 Angle Between Hyoid and Mandibular Plane (<Go – Gn – H)

The angle constructed by joining the cephalometric points on the mandible gonion and gnathion and the most anterosuperior point on the hyoid bone was significantly less in Type II snoring patients compared to those with Type II OSA (p=0.032). The average angle formed between the mandibular plane and hyoid was 30.09° in Type II snoring subjects, whilst those with Type II OSA had an average angle of 34.21°.

### 11.7.15 Tongue Length (TT – Et)

The distance from the tongue tip to the most superior point on the tip of the epiglottis was significantly greater in subjects with Type II OSA than their Type II snoring counterparts (p=0.037). The mean tongue length as measured between these landmarks was 73.23 mm in the Type II snoring group whilst the OSA group had a mean distance of 76.64 mm.

**Table 11.7-2: Significant Variables with Group Separation RDI > 15**

| | Mean | | Standard Deviation | | Maximum | | Minimum | | p Value |
|---|---|---|---|---|---|---|---|---|---|
| | Snorer | OSA | Snorer | OSA | Snorer | OSA | Snorer | OSA | |
| BMI | 29.10 | 35.10 | 4.89 | 7.15 | 42.2 | 51.5 | 18.9 | 23.9 | 0.000 |
| SaO2 | 84.56 | 75.35 | 9.82 | 12.89 | 96 | 94 | 46 | 49 | 0.000 |
| NC | 39.45 | 43.74 | 3.79 | 3.88 | 46 | 51 | 33 | 35.5 | 0.000 |
| H – C3 | 37.79 | 41.18 | 6.04 | 5.80 | 50.09 | 55.41 | 26.69 | 27.99 | 0.006 |
| H – C4 | 38.45 | 41.78 | 5.64 | 5.79 | 51.18 | 56.38 | 27.71 | 29.89 | 0.007 |
| VAL | 69.86 | 73.99 | 6.85 | 7.06 | 82.97 | 86.9 | 53.84 | 58.38 | 0.007 |
| S – H | 111.32 | 116.35 | 8.52 | 8.82 | 127 | 129.53 | 93.06 | 95.36 | 0.008 |
| TGL | 75.51 | 79.64 | 6.23 | 7.54 | 63.55 | 65.26 | 87.2 | 98.11 | 0.010 |
| UW1 – UW2 | 10.02 | 10.85 | 1.56 | 1.96 | 6.5 | 6.7 | 13.67 | 15.93 | 0.018 |
| <H – H1 | 16.98 | 21.61 | 9.29 | 8.90 | 37.39 | 39.31 | -10.96 | 5.91 | 0.029 |
| MP – H | 21.43 | 24.58 | 6.68 | 6.00 | 32.91 | 36.74 | 2.06 | 10.96 | 0.032 |
| <Go–Gn–H | 30.09 | 34.21 | 8.51 | 8.67 | 47.95 | 48.09 | 8.85 | 13.25 | 0.037 |
| TT - ET | 73.23 | 76.64 | 6.28 | 7.76 | 86.3 | 95.14 | 59.93 | 60.87 | 0.037 |

## 11.8 Group differences with cut-off RDI 20

The following analyses were performed following the division of the population into two groups. These groups were determined by the RDI recorded for an individual subject in their polysomnographic study. Those subjects with an RDI < 20 will be referred to as Type III Type III snoring subjects and those with an RDI > 20 as Type III OSA subjects.

The two groups did not contain 50% of the sample each, with Type III snorers comprising 53 subjects and Type III OSA subjects numbering 41. This difference was not enough to preclude the use of a two tail t – test assuming equal variance. Statistical analysis was performed on the same variables as for the Pearson correlation (Section 12.2 and Section 12.3) and additionally sex and age distribution was considered for the two groups.

Only those measures that were statistically significant at the 0.01 or 0.05 level of significance will be discussed. The variables that are significantly different between the two groups of patients are reported in Table 11.8-1.

### 11.8.1 Body Mass Index (BMI)

The difference in BMI between the two groups was significant at the 0.01 level with a p-value of 0.000. Fifty subjects were in the Type III snoring group and had a mean BMI of 29.59 $kg/m^2$, whilst the Type III OSA group had a mean BMI of 35.65 $kg/m^2$.

### 11.8.2 Minimum Arterial Oxygen Saturation ($SaO_2$)

The average minimum $SaO_2$ for the Type III snoring group was 84.33 % whilst the average minimum $SaO_2$ for the Type III OSA group was 72.88 %. This was significantly different with a p-value of 0.000.

### 11.8.3 Neck Circumference (NC)

The neck circumference was significantly smaller in the Type III snoring group compared with the Type III OSA group. The average neck circumference in the Type III snoring group was40.09 cm whilst in the Type III OSA group it was 43.94 cm. This difference was highly significant with p=0.000.

### 11.8.4 Soft Palate Thickness (UW1 – UW2)

The thickness of the soft palate at its widest point was significantly smaller in the Type III snoring group compared with the subjects with Type III OSA (p=0.002). The average thickness of the soft palate's widest point was 9.90 mm in the Type III snoring group, whilst in the Type III OSA group this measurement was 11.06 mm.

### 11.8.5 Pharyngeal Length (VAL)

The length of the pharynx, as measured from the posterior nasal spine to the base of the epiglottis, was significantly shorter in patients who snore compared to those with Type III OSA (p=0.003). The average distance for subjects in the Type III snoring group was 69.96 mm compared to 74.30 mm in those subjects in the Type III OSA group.

### 11.8.6 Tongue Length (TGL)

Tongue length as measured from the tip of the tongue to the base of the epiglottis (vallecula) was significantly shorter in Type III snoring subjects than in those subjects with Type III

OSA. The mean tongue length for the Type III snoring group was 75.66 cm whilst the mean length was 79.92 cm in the Type III OSA subjects. This difference was significant at the 0.01 level of significance with p=0.004.

### 11.8.7 Tongue Length (TT – Et)

The distance from the tongue tip to the most superior point on the tip of the epiglottis was significantly greater in subjects with Type III OSA than their Type III snoring counterparts (p=0.021). The mean tongue length as measured between these landmarks was 73.37 mm in the Type III snoring group whilst the Type III OSA group had a mean distance of 76.80 mm.

### 11.8.8 Upper Incisal Angle (U1 – SN)

The angle formed between the cranial base (line joining points sella and nasion) and the upper incisor (line passing through the upper incisor tip and apex) was significantly greater in Type III OSA subjects than Type III snoring subjects. This indicates a greater proclination of the upper incisor in the Type III OSA subjects (p=0.030). The average upper incisal angulation in the Type III snoring group was $103.81^0$ whilst in the Type III OSA group it was $108.69^0$.

### 11.8.9 Distance Uvula Tip to Posterior Pharyngeal Wall (UT – PhW1)

The distance from the tip of the uvula to the posterior pharyngeal wall measured parallel to Frankfort Horizontal plane was increased in the Type III snoring group compared with the Type III OSA group (p=0.051). The mean distance between the uvula tip and the posterior pharyngeal wall was 19.03 mm in the Type III snoring group and 21.71 mm in the Type III OSA group.

**Table 11.8-1: Significant Variables with Group Separation RDI > 20**

| | Mean | | Standard Deviation | | Maximum | | Minimum | | p Value |
|---|---|---|---|---|---|---|---|---|---|
| | Snorer | OSA | Snorer | OSA | Snorer | OSA | Snorer | OSA | |
| BMI | 29.59 | 35.65 | 5.03 | 7.34 | 42.20 | 51.50 | 18.90 | 24.00 | 0.000 |
| SaO2 | 84.33 | 72.88 | 10.02 | 16.05 | 96.00 | 94.00 | 46.00 | 49.00 | 0.000 |
| Neck Circ | 40.09 | 43.94 | 4.02 | 3.79 | 49.00 | 51.00 | 33.00 | 36.00 | 0.000 |
| UW1– UW2 | 9.90 | 11.06 | 1.60 | 1.92 | 13.67 | 15.93 | 6.50 | 6.70 | 0.002 |
| VAL | 69.96 | 74.30 | 7.11 | 6.75 | 86.75 | 86.9 | 53.84 | 58.68 | 0.003 |
| TGL | 75.66 | 79.92 | 7.04 | 6.74 | 98.11 | 92.76 | 63.55 | 65.65 | 0.004 |
| TT – ET | 73.37 | 76.80 | 7.27 | 6.91 | 95.14 | 90.88 | 59.93 | 60.87 | 0.021 |
| <U1 – SN | 103.81 | 108.69 | 10.22 | 7.04 | 121.00 | 123.00 | 64.00 | 91.00 | 0.030 |
| UT – PhW1 | 19.03 | 21.71 | 5.72 | 7.55 | 33.99 | 41.2 | 10.57 | 4.72 | 0.051 |

## 11.9 Maxillary and Mandibular Antero-Posterior Position

The position of the maxilla and mandible with respect to the cranial base is considered "normal" if subnasale is located more anterior to nasion than supramentale. Individuals may exhibit maxillary or mandibular prognathism or retrognathism relative to the cranial base measured by the angle S-N-A or S-N-B. Alternatively the maxilla or mandible may be prognathic or retrognathic with respect to each other. The population studied was analysed for extreme anteroposterior position of the maxilla or mandible according to recognized cephalometric norms for a Caucasian population (Steiner, 1959).

The method used to determine the relationship in the anteroposterior direction of the maxilla and mandible was angle A-N-B. Subjects with a normal, or class I skeletal relationship were defined as those with an angle $0^0$ < A-N-B > $5^0$. Thirty-nine subjects (41.5%) had such a relationship. The largest group of patients were those where the mandible was located further anteriorly with respect to the maxilla, as defined by an angle A-N-B < $0^0$. Forty-two subjects (44.7%) had this relationship. The smallest group (thirteen subjects, 13.8%) had an angle A-N-B > $5^0$ indicating a prognathic maxilla relative to the mandible. Clinch (1951) reported and epidemiologic study on the incidence of malocclusion in the Australian Caucasian population. In his sample 87.0% were class I, 11.1% were class II and 1.9% were class III. This raw data shows a significantly greater number of subjects with a class III malocclusion compared with the expected incidence. There is no significant difference in the

incidence of a class II relationship with the increase in a class III skeletal relationship being at the expense of a class I pattern. These results are shown in Table 11.9-1.

A Chi-square analysis was undertaken to compare the distribution of obstructive sleep apnoea and snoring at the various diagnostic levels (RDI < 10, RDI < 15 and RDI < 20 for snoring groups) with the distribution of the subjects according to skeletal classification. There was no significant difference in the incidence of obstructive sleep apnoea or snoring subjects in each group compared with that which would be expected based upon the incidence of obstructive sleep apnoea in our sample. The raw data showing the division of subjects according to skeletal class and severity of snoring/obstructive sleep apnoea is shown in Table 11.9-1.

**Table 11.9-1: Skeletal Classification of Subjects and the Incidence of Snoring/Obstructive Sleep Apnoea.**

| | ANB < 0 | 0 < ANB < 5 | ANB > 5 |
|---|---|---|---|
| **Whole group** | 42 | 39 | 13 |
| **Skeletal Class I** | 0 | 39 | 0 |
| **Skeletal Class II** | 0 | 0 | 13 |
| **Skeletal Class III** | 42 | 0 | 0 |
| **RDI < 10 snorer** | 16 | 15 | 4 |
| **RDI > 10 OSA** | 26 | 25 | 9 |
| **RDI < 15 snorer** | 19 | 20 | 5 |
| **RDI > 15 OSA** | 23 | 19 | 8 |
| **RDI < 20 snorer** | 22 | 24 | 6 |
| **RDI > 20 OSA** | 20 | 15 | 7 |

Subjects with obstructive sleep apnoea and an RDI > 20 events per hour were separated into six groups based upon BMI and skeletal class. These groupings were then statistically analysed by a t-test to determine if BMI or skeletal class were the greater influence upon severity of obstructive sleep apnoea.

**Table 11.9-2: Influence of BMI on OSA Severity by Skeletal Class**

| | Mean RDI per subject | p Value |
|---|---|---|
| 0 ANB 5; BMI > 30 | 62.3 | |
| 0 ANB 5; BMI 30 | 36.6 | 0.037 |
| ANB < 5; BMI 30 | 42.75 | |
| ANB < 5; BMI 30 | 48.95 | 0.294 |
| ANB > 5; BMI > 30 | 48.95 | |
| ANB > 5; BMI 30 | 34.13 | 0.049 |

Twenty-one subjects had a retrognathic maxilla greater than one standard deviation below the mean for a Caucasian population (S-N-A < $80^0$). Five of these subjects had an angle $0^0$ < A-N-B < $5^0$ (skeletal class I); two had an angle A-N-B > $5^0$ (skeletal class II) whilst 14 had an angle A-N-B < $0^0$ (skeletal class III). Ten subjects with a retrognathic maxilla had an RDI > 20 (obstructive sleep apnoeic), a further ten subjects had an RDI < 10 (simple snorers) whilst only one subject had an 10 < RDI < 20 events/hour.

Forty-nine subjects had a prognathic maxilla greater than one standard deviation above the mean for a Caucasian population (S-N-A > 84). Twenty-eight of these subjects had an angle$0^0$ < A-N-B < $5^0$ (skeletal class I); eight had an angle A-N-B > $5^0$ (skeletal class II) whilst 13 had an angle A-N-B < $0^0$ (skeletal class III). Twenty five subjects with a retrognathic maxilla had an RDI > 20 (obstructive sleep apnoeic), a further 13 subjects had an RDI < 10 (simple snorers) whilst 11 subjects had an 10 < RDI < 20 events/hour.

The mandible was retrognathic (S-N-B < $78^0$) in 15 subjects at a level greater than one standard deviation from the Caucasian population mean. Four of these subjects were skeletal class I ($0^0$ < A-N-B < $5^0$); seven were skeletal class II (A-N-B > $5^0$) and four were skeletal class III (A-N-B < $0^0$). There were seven simple snorers (RDI < 10) in this group, seven subjects with obstructive sleep apnoea (RDI > 20) and one subject with a respiratory disturbance index 10 < RDI < 20.

Mandibular prognathism (S-N-B > 82) greater than one standard deviation from the Caucasian population mean was a finding in 60 subjects. Twenty-seven of these subjects had a class I skeletal relationship ($0^0$ < A-N-B < $5^0$); three had a class II skeletal relationship (A-N-B > $5^0$) and 30 had a class II skeletal relationship (A-N-B < $0^0$). The number of simple snorers in this group was 21 (RDI < 10), 26 subjects had obstructive sleep apnoea (RDI > 20)

whilst 13 subjects had a respiratory disturbance index 10 < RDI < 20. These findings are all shown in Table 11.9-3.

**Table 11.9-3: Subjects With Maxilla and/or Mandible Greater Than 1 SD from Population Mean**

| | SNA < 80 | SNA > 84 | SNB < 78 | SNB > 82 |
|---|---|---|---|---|
| **Whole group** | 21 | 49 | 15 | 60 |
| **Skeletal Class I** | 5 | 28 | 4 | 27 |
| **Skeletal Class II** | 2 | 8 | 7 | 3 |
| **Skeletal Class III** | 14 | 13 | 4 | 30 |
| **RDI < 10 snorer** | 10 | 13 | 7 | 21 |
| **RDI > 10 OSA** | 11 | 36 | 8 | 39 |
| **RDI < 15 snorer** | 10 | 18 | 7 | 29 |
| **RDI > 15 OSA** | 11 | 31 | 8 | 31 |
| **RDI < 20 snorer** | 11 | 24 | 8 | 34 |
| **RDI > 20 OSA** | 10 | 25 | 7 | 26 |

Ten subjects in the study population had a maxilla and mandible that were at least one standard deviation below the expected population mean. These ten subjects all exhibited traits as defined by lateral cephalometry of bimaxillary retrusion. Four of these subjects had a Skeletal Class I maxilla and mandible relationship, two had a Skeletal Class II relationship and four were in the Skeletal Class III group. Considering the same ten subjects with respect to RDI half had an RDI < 10, one had an 15 < RDI < 20 and the remaining four had an RDI greater than 20. These results are shown in Table 11.9-4. Three subjects (30%) had a BMI < 25 kg/m$^2$ and eight (80%) had a BMI < 30 kg/m$^2$.

**Table 11.9-4: Bimaxillary Retrusion (1 SD), Skeletal Class and RDI.**

| | RDI < 10 | 10 < RDI < 15 | 15 < RDI < 20 | RDI > 20 |
|---|---|---|---|---|
| **Total** | 5 | 0 | 1 | 4 |
| **Class I** | 3 | 0 | 1 | 0 |
| **Class II** | 1 | 0 | 0 | 0 |
| **Class III** | 1 | 0 | 0 | 4 |

Forty-one subjects had a maxilla and mandible that were at least one standard deviation above the expected population mean (bimaxillary prognathism). Thirty of these subjects had a skeletal class I relationship, three had a skeletal class II relationship and thirteen had a skeletal class III relationship. Twelve of these subjects exhibiting a trait for bimaxillary prognathism had an RDI < 10. Eight were skeletal class I and four were skeletal class III. Four had a respiratory disturbance index 10 < RDI <15, with three having a class I skeletal relationship and one a class I skeletal relationship. Five had 15 < RDI < 20, two class I and three class III. Twenty had an RDI > 20. Twelve of these subjects had a class I skeletal relationship, two had a class II relationship whilst the remaining eight had a class III skeletal relationship. These results are shown in Table 11.9-5. Thirty six subjects (88%) had a BMI > 25 kg/m$^2$ whilst twenty six (63%) had a BMI > 30 kg/m$^2$.

**Table 11.9-5: Bimaxillary Protrusion (1 SD), Skeletal Class and RDI.**

| | Total | RDI < 10 | 10 < RDI < 15 | 15 < RDI < 20 | RDI > 20 |
|---|---|---|---|---|---|
| **Total** | 41 | 12 | 4 | 5 | 20 |
| **Class I** | 25 | 8 | 3 | 2 | 12 |
| **Class II** | 3 | 0 | 1 | 0 | 2 |
| **Class III** | 13 | 4 | 0 | 3 | 6 |

Eleven subjects had a retrognathic maxilla greater than two standard deviations below the mean for a Caucasian population (S-N-A < $78^0$). Two of these subjects had an angle $0^0$ < A-N-B < $5^0$ (skeletal class I); one had an angle A-N-B > $5^0$ (skeletal class II) whilst eight had an angle A-N-B < $0^0$ (skeletal class III). Six subjects with a retrognathic maxilla had an RDI > 20 (obstructive sleep apnoeic), a further four subjects had an RDI < 10 (simple snorers) whilst only one subject had an 10 < RDI < 20 events/hour.

Thirty-four subjects had a prognathic maxilla greater than two standard deviations above the mean for a Caucasian population (S-N-A > $86^0$). Twenty-three of these subjects had an angle $0^0$ < A-N-B < $5^0$ (skeletal class I); five had an angle A-N-B > $5^0$ (skeletal class II) whilst six had an angle A-N-B < $0^0$ (skeletal class III). Fifteen subjects with a retrognathic maxilla had an RDI > 20 (obstructive sleep apnoeic), a further nine subjects had an RDI < 10 (simple snorers) whilst 10 subjects had an 10 < RDI < 20 events/hour.

The mandible was retrognathic (S-N-B < $76^0$) in eight subjects at a level greater than two standard deviations from the Caucasian population mean. One of these subjects were skeletal class I ($0^0$ < A-N-B < $5^0$); four were skeletal class II (A-N-B > $5^0$) and three were skeletal

class III (A-N-B < $0^0$). There were three simple snorers (RDI < 10) in this group, four subjects with obstructive sleep apnoea (RDI > 20) and one subject with a respiratory disturbance index 10 < RDI < 20.

Mandibular prognathism (S-N-B > $84^0$) greater than two standard deviations from the Caucasian population mean was a finding in 48 subjects. Twenty-one of these subjects had a class I skeletal relationship ($0^0$ < A-N-B < $5^0$); two had a class II skeletal relationship (A-N-B > $5^0$) and 25 had a class II skeletal relationship (A-N-B < $0^0$). The number of simple snorers in this group was 16 (RDI < 10), 21 subjects had obstructive sleep apnoea (RDI > 20) whilst 11 subjects had a respiratory disturbance index 10 < RDI < 20. These findings are all shown in Table 11.9-6.

**Table 11.9-6: Subjects With Maxilla and/or Mandible Greater Than 2 SD from Population Mean**

| | SNA < 78 | SNA > 86 | SNB < 76 | SNB > 84 |
|---|---|---|---|---|
| **Whole group** | 11 | 34 | 8 | 48 |
| **Skeletal Class I** | 2 | 23 | 1 | 21 |
| **Skeletal Class II** | 1 | 5 | 4 | 2 |
| **Skeletal Class III** | 8 | 6 | 3 | 25 |
| **RDI < 10 snorer** | 4 | 9 | 3 | 16 |
| **RDI > 10 OSA** | 7 | 25 | 5 | 32 |
| **RDI < 15 snorer** | 4 | 13 | 3 | 22 |
| **RDI > 15 OSA** | 7 | 21 | 5 | 26 |
| **RDI < 20 snorer** | 5 | 19 | 4 | 27 |
| **RDI > 20 OSA** | 6 | 15 | 4 | 21 |

Five subjects in the study population had a maxilla and mandible that were at least two standard deviations below the expected population mean. One of these subjects had a class I skeletal relationship ($0^0$ < A-N-B < $5^0$), one had a skeletal class II relationship (A-N-B > $5^0$) and three were in the skeletal class III group (A-N-B < $0^0$). Considering the same five subjects with respect to RDI the subject with a class I skeletal relationship was a snorer (RDI < 10); the subject with the class II skeletal relationship had a respiratory disturbance index in the range 15 < RDI < 20 whilst all three subjects with a class III skeletal relationship were obstructive sleep apnoeic (RDI > 20). These results are shown in Table 11.9-7. One of these subjects had a BMI < 25 kg/$m^2$, three had a BMI < 30 kg/$m^2$ and one had a BMI > 30 kg/$m^2$.

**Table 11.9-7: Bimaxillary Retrusion (2SD), Skeletal Class and RDI.**

| | Total | RDI < 10 | 10 < RDI < 15 | 15 < RDI < 20 | RDI > 20 |
|---|---|---|---|---|---|
| **Total** | 5 | 1 | 0 | 1 | 3 |
| **Class I** | 1 | 0 | 0 | 1 | 0 |
| **Class II** | 1 | 1 | 0 | 0 | 0 |
| **Class III** | 3 | 0 | 0 | 0 | 3 |

Twenty eight subjects had both maxilla and mandible that were at least two standard deviations above the expected population mean when measured by angle S-N-A and S-N-B respectively. Twenty of these subjects had a skeletal class I relationship, two had a skeletal class II relationship and six had a skeletal class III relationship. Seven of these subjects exhibiting a trait for bimaxillary prognathism had an RDI < 10, five skeletal class I and two skeletal class III). Four had 10 < RDI <15, three skeletal class I and one skeletal class II. Five had 15 < RDI < 20, two skeletal class I and three skeletal class III. Twelve had an RDI > 20, 10 skeletal class I, and one each with a skeletal class II and class III. These results are recorded in Table 11.9-8. Twenty-four subjects (86%) had a BMI > 25 kg/m$^2$ whilst sixteen (57%) had a BMI > 30 kg/m$^2$.

**Table 11.9-8: Bimaxillary Protrusion (2SD), Skeletal Class and RDI.**

| | Total | RDI < 10 | 10 < RDI < 15 | 15 < RDI < 20 | RDI > 20 |
|---|---|---|---|---|---|
| **Total** | 28 | 7 | 4 | 5 | 12 |
| **Class I** | 20 | 5 | 3 | 2 | 10 |
| **Class II** | 2 | 0 | 1 | 0 | 1 |
| **Class III** | 6 | 2 | 0 | 3 | 1 |

Six subjects had a retrognathic maxilla greater than three standard deviations below the mean for a Caucasian population (S-N-A < 76$^0$). One of these subjects had an angle 0$^0$ < A-N-B < 5$^0$ (skeletal class I); one had an angle A-N-B > 5$^0$ (skeletal class II) whilst four had an angle A-N-B < 0$^0$ (skeletal class III). Three subjects with a retrognathic maxilla had an RDI > 20 (obstructive sleep apnoeic), a further two subjects had an RDI < 10 (simple snorers) whilst only one subject had an 10 < RDI < 20 events/hour.

Twenty-two subjects had a prognathic maxilla greater than three standard deviations above the mean for a Caucasian population (S-N-A > 88$^0$). Fifteen of these subjects had 0$^0$ < A-N-

B < $5^0$ (skeletal class I); four had an angle A-N-B > $5^0$ (skeletal class II) whilst three had an angle A-N-B < $0^0$ (skeletal class III). Ten subjects with a retrognathic maxilla had an RDI > 20 (obstructive sleep apnoeic), a further seven subjects had an RDI < 10 (simple snorers) whilst five subjects had an 10 < RDI < 20 events/hour.

The mandible was retrognathic (S-N-B < $74^0$) in five subjects at a level greater than three standard deviations from the Caucasian population mean. One of these subjects were skeletal class I ($0^0$ < A-N-B < $5^0$); two were skeletal class II (A-N-B > $5^0$) and two were skeletal class III (A-N-B < $0^0$). There were two simple snorers (RDI < 10) in this group, two subjects with obstructive sleep apnoea (RDI > 20) and one subject with a respiratory disturbance index 10 < RDI < 20.

Mandibular prognathism (S-N-B > $86^0$) greater than three standard deviations from the Caucasian population mean was a finding in 28 subjects. Thirteen of these subjects had a class I skeletal relationship ($0^0$ < A-N-B < $5^0$); two had a class II skeletal relationship (A-N-B > $5^0$) and 13 had a class II skeletal relationship (A-N-B < $0^0$). The number of simple snorers in this group was six (RDI < 10), fourteen subjects had obstructive sleep apnoea (RDI > 20) whilst eight subjects had a respiratory disturbance index 10 < RDI < 20. These findings are shown in Table 11.9-9 and are extremes at least three standard deviations from the control population mean as used by Steiner (1959).

**Table 11.9-9: Subjects With Maxilla and/or Mandible Greater Than 3 SD from Population Mean**

| | SNA < 76 | SNA > 88 | SNB < 74 | SNB > 86 |
|---|---|---|---|---|
| **Whole group** | 6 | 22 | 5 | 28 |
| **Skeletal Class I** | 1 | 15 | 1 | 13 |
| **Skeletal Class II** | 1 | 4 | 2 | 2 |
| **Skeletal Class III** | 4 | 3 | 2 | 13 |
| **RDI < 10 snorer** | 2 | 7 | 2 | 6 |
| **RDI > 10 OSA** | 4 | 15 | 3 | 22 |
| **RDI < 15 snorer** | 2 | 6 | 2 | 11 |
| **RDI > 15 OSA** | 4 | 16 | 3 | 17 |
| **RDI < 20 snorer** | 3 | 12 | 3 | 14 |
| **RDI > 20 OSA** | 3 | 10 | 2 | 14 |

Four subjects in the whole study population had a maxilla and mandible that were at least three standard deviations below the expected population mean. One of these subjects had a Skeletal Class I maxilla and mandible relationship, one had a Skeletal Class II relationship

and two were in the Skeletal Class III group. Considering the same four subjects with respect to RDI, the subject with a class I skeletal relationship had an RDI < 10, the subject with a class II skeletal relationship had a respiratory disturbance index 15 < RDI < 20 and the remaining two had a skeletal class III relationship and an RDI > 20. These results are shown in Table 11.9-10. One of these subjects (25%) had a BMI < 25 kg/m$^2$ and three (75%) had a BMI < 30 kg/m$^2$.

**Table 11.9-10: Bimaxillary Retrusion (3SD), Skeletal Class and RDI**

| | Total | RDI < 10 | 10 < RDI < 15 | 15 < RDI < 20 | RDI > 20 |
|---|---|---|---|---|---|
| **Total** | 4 | 1 | 0 | 1 | 2 |
| **Class I** | 1 | 0 | 0 | 1 | 0 |
| **Class II** | 1 | 1 | 0 | 0 | 0 |
| **Class III** | 2 | 0 | 0 | 0 | 2 |

Fifteen subjects had both maxilla and mandible that were at least three standard deviations above the expected population mean when measured by angle S-N-A and S-N-B respectively. Ten of these subjects had a skeletal class I relationship, two had a skeletal class II relationship and three had a skeletal class III relationship. Three of these subjects exhibiting a trait for bimaxillary prognathism had an RDI < 10, three had 10 < RDI <15, two had 15 < RDI < 20 and seven had an RDI > 20. These results are shown in Table 11.9-11. Thirteen of these subjects (87%) had a BMI > 25 and ten (67%) had a BMI > 30 kg/m$^2$.

**Table 11.9-11: Bimaxillary Protrusion (3SD), Skeletal Class and RDI.**

| | Total | RDI < 10 | 10 < RDI < 15 | 15 < RDI < 20 | RDI > 20 |
|---|---|---|---|---|---|
| **Total** | 15 | 3 | 3 | 2 | 7 |
| **Class I** | 10 | 2 | 2 | 0 | 6 |
| **Class II** | 2 | 0 | 1 | 0 | 1 |
| **Class III** | 3 | 1 | 0 | 2 | 0 |

The anteroposterior position of the maxilla and mandible, as measured by angle S-N-A and S-N-B respectively, and the severity of UAW obstruction, as measured by RDI was compared to the relationship of the maxilla and mandible as defined by angle A-N-B. This relationship quantifies the degree of maxillary or mandibular prognathism or retrognathism and relates this

to the interrelationship of the jaws. The inclusion of RDI in the criteria determined if there was a trend toward a greater incidence of OSA in subjects who had jaw position greater than one standard deviation from that expected for the general population.

Table 11.9-12 shows these relationships for patients who have a maxilla or mandible greater than one standard deviation from the expected mean in the anteroposterior direction. The maxilla and mandible are located anteroposteriorly by measuring angle S-N-A and angle S-N-B respectively, the mean and standard deviation is that reported by Steiner (1959) for these cephalometric measures.

There were 39 subjects with a skeletal class I relationship ($0^0$ < A-N-B < $5^0$), of whom 25 (64.1%) had an RDI > 10. Only one subject (4.2%) had a maxilla that was retrognathic at one standard deviation from the mean (S-N-A < $80^0$), although 19 (79.2%) had a relatively prognathic maxilla (S-N-A > $84^0$). Relative mandibular prognathism (S-N-B > $82^0$) occurred in 10 subjects (40.0%) and was more common than mandibular retrognathism (S-N-B < $78^0$) which occurred in one subject (4.0%).

There were thirteen subjects who had a skeletal class II relationship (A-N-B > $5^0$) of whom 9 (69.2%) had an RDI > 10. Three of these subjects (23.1%) exhibited mandibular retrognathism and had an RDI > 20. No subject exhibited a retrognathic maxilla, however eight (61.5%) had a prognathic maxilla. Three subjects had a bimaxillary protrusion with the maxilla being more protrusive than the mandible, resulting in the class II skeletal pattern.

Subjects who had a skeletal class III relationship (A-N-B < $0^0$) numbered 42, of whom 26 (61.9%) had an RDI > 10. All subjects who had a retrognathic maxilla (n = 10, 23.8%) had greater than 20 UAW obstructive events per hour of sleep. Similarly all subjects who had a retrognathic mandible (n = 4, 9.5%) had greater than 20 obstructive events per hour of sleep. In this group of patients the mandible was more likely to be prognathic (n = 19, 45.2%) than the maxilla (n = 9, 21.4%).

Maxillary retrognathism (S-N-A < $80^0$) with a normally positioned mandible ($78^0$ < S-N-B < $82^0$) occurred in 11 subjects. Five of these subjects had an RDI < 10 and six had an RDI > 20. Ten subjects had bimaxillary retrusion defined by the maxilla and mandible being greater than one standard deviation below the mean. Five of these subjects had an RDI < 10, one was in the range 10 < RDI < 20 with four having an RDI > 20. Mandibular retrognathism (S-N-B < $78^0$) with a normally positioned maxilla (S-N-A > $80^0$) was present in five subjects. Two of these had an RDI < 10 whilst three had an RDI > 20.

Mandibular prognathism (S-N-B > $82^0$) with a normally positioned maxilla ($80^0$ < S-N-A < $84^0$) occurred in 19 subjects. Nine of these were simple snorers (RDI < 10), six had an RDI > 20 and the remaining four were in the range 10 < RDI < 20. Forty-one subjects had

bimaxillary protrusion defined by the maxilla and mandible being greater than one standard deviation above the mean. Twelve of these subjects had an RDI < 10, nine were in the range 10 < RDI < 20 with 20 having an RDI > 20. Maxillary prognathism (S-N-A > $84^0$) with a normally positioned mandible (S-N-B < 82) was present in eight subjects. One was a simple snorer (RDI < 10), three were in the range 10 < RDI < 20 whilst five had an RDI > 20.

**Table 11.9-12: Comparison of RDI and Anteroposterior Jaw Position Greater Than 1 SD From Population Mean**

| | 0 < ANB < 5 | ANB > 5 | ANB < 0 |
|---|---|---|---|
| **RDI > 10 and SNA < 80** | 1 | 0 | 10 |
| **RDI > 10 and SNA > 84** | 19 | 8 | 9 |
| **RDI > 15 and SNA < 80** | 1 | 0 | 10 |
| **RDI > 15 and SNA > 84** | 15 | 6 | 9 |
| **RDI > 20 and SNA < 80** | 0 | 0 | 10 |
| **RDI > 20 and SNA > 84** | 13 | 6 | 6 |
| **RDI > 10 and SNB < 78** | 1 | 3 | 4 |
| **RDI > 10 and SNB > 82** | 10 | 3 | 19 |
| **RDI > 15 and SNB < 78** | 1 | 3 | 4 |
| **RDI > 15 and SNB > 82** | 7 | 2 | 15 |
| **RDI > 20 and SNB < 78** | 0 | 3 | 4 |
| **RDI > 20 and SNB > 82** | 7 | 2 | 12 |

Table 11.9-13 shows the anteroposterior relationship of the maxilla and mandible where either jaw is greater than two standard deviations from the expected mean in the anteroposterior direction. The maxilla and mandible are located anteroposteriorly by measuring angle S-N-A and angle S-N-B respectively, the mean and standard deviation is that reported by Steiner (1959) for these cephalometric measures.

There were 39 subjects in the group $0^0$ < A-N-B < $5^0$, of whom 25 (64.1%) had an RDI > 10. Only one subject (4%) had a maxilla that was retrognathic at two standard deviations from the mean, although 16 (64%) had a relatively prognathic maxilla. Relative mandibular prognathism (S-N-B > $84^0$) was more common than mandibular retrognathism. Fifteen subjects (60%) had an S-N-B > $84^0$ , with only one exhibiting mandibular retrognathism.

There were thirteen subjects who had a skeletal class II relationship (A-N-B > $5^0$) of whom nine (69.2%) had an RDI > 10. One of these subjects (11.1%) exhibited mandibular retrognathism and had an RDI > 20. None of these subjects had a retrognathic maxilla,

however three (33%) had a prognathic maxilla. One subject had bimaxillary protrusion with the maxilla being more protrusive than the mandible, resulting in the class II skeletal pattern.

Subjects who had a skeletal class III relationship (A-N-B < $0^0$) numbered 42, of whom 26 (61.9%) had an RDI > 10. All subjects who had a retrognathic maxilla (n = 6, 23.1%) had greater than 20 UAW obstructive events per hour of sleep. Similarly all subjects who had a retrognathic mandible (n = 3, 11.5%) had greater than 20 obstructive events per hour of sleep. In this group of patients the mandible was more likely to be prognathic (n = 15, 57.7%) than the maxilla (n = 4, 15.4%).

**Table 11.9-13: Comparison of RDI and Anteroposterior Jaw Position Greater Than 2 SD From Population Mean**

| | 0 < ANB < 5 | ANB > 5 | ANB < 0 |
|---|---|---|---|
| RDI > 10 and SNA < 78 | 1 | 0 | 6 |
| RDI > 10 and SNA > 86 | 16 | 5 | 4 |
| RDI > 15 and SNA < 78 | 1 | 0 | 6 |
| RDI > 15 and SNA > 86 | 13 | 3 | 4 |
| RDI > 20 and SNA < 78 | 0 | 0 | 6 |
| RDI > 20 and SNA > 86 | 11 | 3 | 1 |
| RDI > 10 and SNB < 76 | 1 | 1 | 3 |
| RDI > 10 and SNB > 84 | 15 | 2 | 15 |
| RDI > 15 and SNB < 76 | 1 | 1 | 3 |
| RDI > 15 and SNB > 84 | 12 | 1 | 13 |
| RDI > 20 and SNB < 76 | 0 | 1 | 3 |
| RDI > 20 and SNB > 84 | 10 | 1 | 10 |

Table 11.9-14 shows the anteroposterior relationship of the maxilla and mandible where either jaw is greater than three standard deviations from the expected mean in the anteroposterior direction. The maxilla and mandible are located anteroposteriorly by measuring angle S-N-A and angle S-N-B respectively, the mean and standard deviation is that reported by Steiner (1959) for these cephalometric measures.

There were 39 subjects in the group $0^0$ < A-N-B < $5^0$, of whom 25 (64.1%) had an RDI > 10. Only one subject (4%) had a maxilla that was retrognathic at three standard deviations from the mean, although 10 (40%) had a relatively prognathic maxilla. Relative mandibular

prognathism (S-N-B > $86^0$) was more common than mandibular retrognathism. Ten subjects (40%) had an S-N-B > $86^0$, with only one (4%) exhibiting mandibular retrognathism.

There were thirteen subjects who had a skeletal class II relationship (A-N-B > $5^0$) of whom 9 (69.2%) had an RDI > 10. None of these subjects exhibited maxillary or mandibular retrognathism. Four subjects (44.4%) had a prognathic maxilla and two (22.2%) exhibited mandibular prognathism. Two subjects (22.2%) had bimaxillary protrusion with the maxilla being more protrusive than the mandible, resulting in the class II skeletal pattern.

Subjects who had a skeletal class III relationship (A-N-B < $0^0$) numbered 42, of whom 26 (61.9%) had an RDI > 10. All subjects who had a retrognathic maxilla (n = 3, 11.5%) had greater than 20 UAW obstructive events per hour of sleep. Similarly all subjects who had a retrognathic mandible (n = 2, 7.7%) had greater than 20 obstructive events per hour of sleep. In this group of patients the mandible was more likely to be prognathic (n = 10, 38.5%) than the maxilla (n = 2, 7.7%).

**Table 11.9-14: Comparison of RDI and Anteroposterior Jaw Position Greater Than 3 SD From Population Mean**

| | 0 < ANB < 5 | ANB > 5 | ANB < 0 |
|---|---|---|---|
| RDI > 10 and SNA < 76 | 1 | 0 | 3 |
| RDI > 10 and SNA > 88 | 10 | 4 | 2 |
| RDI > 15 and SNA < 76 | 1 | 0 | 3 |
| RDI > 15 and SNA > 88 | 8 | 2 | 2 |
| RDI > 20 and SNA < 76 | 0 | 0 | 3 |
| RDI > 20 and SNA > 88 | 8 | 2 | 0 |
| RDI > 10 and SNB < 74 | 1 | 0 | 2 |
| RDI > 10 and SNB > 86 | 10 | 2 | 10 |
| RDI > 15 and SNB < 74 | 1 | 0 | 2 |
| RDI > 15 and SNB > 86 | 7 | 1 | 9 |
| RDI > 20 and SNB < 74 | 0 | 0 | 2 |
| RDI > 20 and SNB > 86 | 7 | 1 | 6 |

Analysis of cranial base length according to skeletal class revealed those subjects with a Class II skeletal relationship had a significantly shorter distance S – N than subjects with a Class I skeletal relationship or those with a Class III skeletal relationship. No such difference was evident for the angle Ba – S – N for these groupings. Similarly there was no significant

difference between subgroups based on RDI for the length or angulation of the cranial base. The results are shown in Table 11.9-15.

**Table 11.9-15 Skeletal Class and Cranial Base Length (S – N)**

| | Mean | Standard Deviation | Minimum | Maximum |
|---|---|---|---|---|
| **0 < ANB < 5** | 77.52 | 4.28 | 64.5 | 83.5 |
| **ANB > 5** | 77.78 | 4.11 | 69.5 | 84.5 |
| **ANB < 0** | 74.67 | 4.19 | 67.5 | 81.0 |

Tsuchiya et al (1992) first suggested that a subgroup of OSA patients have a significant skeletal discrepancy in the absence of an elevated BMI that contributes to UAW obstruction. Table 11.9-16 shows the relationship between BMI and RDI in the current study. Three subjects out of nine (33%) with a BMI < 25 kg/m$^2$ had an RDI > 10. Only one of these subjects had an RDI > 20. Those subjects who were in the range 25 < BMI < 30 kg/m$^2$ numbered 29, of whom 14 (48.3%) had an RDI > 10, falling to 11 (37.9%) with an RDI > 20. For those 52 obese subjects with a BMI > 30 kg/m$^2$, 40 (76.9%) had an RDI > 10 and 29 (55.8%) had an RDI > 20. The numbers are too small to allow statistical analysis to be performed between groups however there is a trend for an increased RDI as BMI increases.

**Table 11.9-16: Distribution of Subjects by RDI and BMI.**

| | BMI < 25 | 25 < BMI < 30 | BMI > 30 |
|---|---|---|---|
| **RDI < 10** | 6 | 15 | 12 |
| **10 < RDI < 15** | 1 | 3 | 6 |
| **15 < RDI < 20** | 1 | 1 | 5 |
| **RDI > 20** | 1 | 10 | 29 |

Considering the nine subjects with a BMI < 25 kg/m$^2$, three had an RDI > 10. All three of these subjects also had a maxilla and/or mandible that measured outside three standard deviations from the mean for a Caucasian population. One subject exhibited bimaxillary retrognathism (S-N-A < 74 / S-N-B < 76), one subject exhibited bimaxillary prognathism (S-N-A >88 / S-N-B > 86) with the third subject having a prognathic mandible (S-N-B > 86). Using an RDI > 15 as the diagnostic level for OSA resulted in retention of the subject with bimaxillary retrognathia and the subject with relatively more prognathism of the mandible than the maxilla. The patient with the most severe OSA (RDI > 20) was the subject who had

bimaxillary protrusion but greater mandibular prognathism than exhibited in the maxilla, resulting in a class III skeletal pattern. These results are shown in Table 11.9-17.

**Table 11.9-17: BMI < 25 kg/m$^2$**

| | RDI < 10 | 10 < RDI < 15 | 15 < RDI < 20 | RDI > 20 |
|---|---|---|---|---|
| **Total** | 6 | 1 | 1 | 1 |
| **SNA < 76** | 0 | 0 | 1 | 0 |
| **SNA > 88** | 1 | 1 | 0 | 0 |
| **SNB < 74** | 0 | 0 | 1 | 0 |
| **SNB > 86** | 1 | 1 | 0 | 1 |
| **SNA < 76 / SNB < 74** | 0 | 0 | 1 | 0 |
| **SNA > 88 / SNB > 86** | 1 | 1 | 0 | 0 |

Considering the twenty-nine subjects with a 25 < BMI < 30 kg/m$^2$, 14 had an RDI > 10 and 11 had an RDI > 20. Seven (50%) had a maxilla and/or mandible that measured outside three standard deviations from the mean for a Caucasian population (Table 11.9-18). One subject exhibited bimaxillary retrognathism (S-N-A < 74 / S-N-B < 76) and an RDI > 20. Two subjects exhibited bimaxillary prognathism (S-N-A >88 / S-N-B > 86) and an RDI in the range 10 < RDI < 15. One subject had a retrognathic maxilla (S-N-A < 76), two had a prognathic maxilla (S-N-A > 88) with the remaining subject having a prognathic mandible (S-N-B > 86). Five subjects had an RDI > 20, including the subject with bimaxillary retrognathia.

**Table 11.9-18: 25 < BMI < 30 kg/m$^2$**

| | RDI < 10 | 10 < RDI < 15 | 15 < RDI < 20 | RDI > 20 |
|---|---|---|---|---|
| **Total** | 15 | 3 | 1 | 10 |
| **SNA < 76** | 2 | 0 | 0 | 2 |
| **SNA > 88** | 1 | 2 | 0 | 2 |
| **SNB < 74** | 2 | 0 | 0 | 1 |
| **SNB > 86** | 0 | 2 | 0 | 1 |
| **SNA < 76 / SNB < 74** | 1 | 0 | 0 | 1 |
| **SNA > 88 / SNB > 86** | 0 | 2 | 0 | 0 |

Considering the fifty-two subjects with a > 30 kg/m$^2$, 40 had an RDI > 10 and 29 had an RDI > 20. Twenty (69.0%) had a maxilla and/or mandible that measured outside three standard deviations from the mean for a Caucasian population (Table 11.9-19). One subject exhibited bimaxillary retrognathism (S-N-A < 74 / S-N-B < 76) and an RDI > 20. Nine subjects exhibited bimaxillary prognathism (S-N-A >88 / S-N-B > 86), two had an RDI in the range 15 < RDI < 20 with the remainder having an RDI > 20. Two subjects had a prognathic maxilla (S-N-A > 88) and eight had a prognathic mandible (S-N-B > 86). Fourteen subjects had an RDI > 20, including the subject with bimaxillary retrognathia.

**Table 11.9-19: BMI > 30 kg/m$^2$**

| | RDI < 10 | 10 < RDI < 15 | 15 < RDI < 20 | RDI > 20 |
|---|---|---|---|---|
| **Total** | 12 | 6 | 5 | 29 |
| **SNA < 76** | 0 | 0 | 0 | 1 |
| **SNA > 88** | 1 | 1 | 2 | 8 |
| **SNB < 74** | 0 | 0 | 0 | 1 |
| **SNB > 86** | 3 | 2 | 3 | 12 |
| **SNA < 76 / SNB < 74** | 0 | 0 | 0 | 1 |
| **SNA > 88 / SNB > 86** | 1 | 0 | 2 | 7 |

# Chapter 12

# Discussion

## 12.1 Patient Selection and Organisational Issues

Prior to commencement of data collection a written protocol and overview of the research project was circulated to the Chest Clinic, Royal Adelaide Hospital, the Dental Radiography Unit, Adelaide Dental Hospital and to the clinicians of the Oral and Maxillofacial Surgery Unit, Royal Adelaide Hospital and Adelaide Dental Hospital.

One hundred and four patients were referred from the Thoracic Medicine Department, Royal Adelaide Hospital to the Oral and Maxillofacial Surgery Unit, Royal Adelaide Hospital for inclusion in the study. The Thoracic Medicine Physician, prior to referring the patient, discussed the purpose of the study and the need for a radiograph with the patient.

In order to minimize bias an attempt was made to obtain consecutive patients, however a number of patients were either unable or unwilling to be enrolled. Reasons given for not participating included concerns over radiation exposure, concerns over privacy and a lack of time to attend another consultative clinic that may have no direct bearing on their management.

There was no compulsion for patients to enroll and not all Thoracic Medicine Consultants in the clinic referred patients for the study. Reasons given by Thoracic Medicine Consultants for non-referral were principally concerned with unnecessary radiation exposure of the patients and doubt over the usefulness of the lateral cephalometric radiograph in the management of patients with OSA. Since preliminary results of this thesis have been discussed with the Director of the Chest Clinic the latter concerns have been somewhat alleviated, however too late for alteration of the pattern of referrals for this study. A record was not kept of the number of patients approached for inclusion into the study however personal communication with the referring physicians indicates the majority of patients approached agreed to participate in the study.

The Chest Clinic and the Adelaide Dental Hospital are within the Royal Adelaide Hospital Health Campus and are located approximately two hundred metres apart and several patients who presented for radiographic examination complained of the inconvenience of attending a separate building for further investigations. Others complained of difficulty locating the Oral and Maxillofacial Surgery Unit, despite the provision of a marked map and detailed written directions and instructions to follow upon presentation to the Adelaide Dental Hospital. A number of patients were turned away by the receptionists at the Adelaide Dental Hospital as they were deemed ineligible for consultation in the Adelaide Dental Hospital, despite written notification being given to the staff prior to commencement of the study.

This latter problem was rectified very early within the study period and these subjects subsequently had a lateral cephalometric radiograph taken and were included in the study.

The problem of incomplete records is not uncommon, as mentioned in other research performed within our unit (Ching Thesis 1995). He noted that even in the most tightly controlled surgical units data collection and record keeping are often inadequate and are indeed a hindrance to accurate and concise research.

A comparison of our study population with age and weight matched controls from the South Australian population may have produced results showing greater potential utility of lateral cephalometric radiographs in the diagnosis of patients with OSA. Preliminary inquiries to the ethics committee indicated approval to record lateral cephalometric radiographs on a control population with no history of snoring would be unlikely to succeed.

The options for a control population were therefore limited to three choices:

1. Cephalometric radiographs from a South Australian population that were taken for orthognathic surgery or orthodontic diagnosis and planning;
2. the use of published cephalometric norms from the national and international literature (matched or unmatched for gender, age or BMI); or
3. the use of internal controls within the population sampled.

A possible source of lateral cephalometric radiographs taken on the same machine was presurgical orthognathic cases. A number of reasons precluded the use of this ready source of lateral cephalometric radiographs.

1. Age difference between the two groups. The patient undergoing corrective orthognathic surgery in the Oral and Maxillofacial Surgery Unit at the Adelaide Dental Hospital has a mean age of 23 years (Ching Thesis, 1995). The average age for the population referred from the Chest Clinic for inclusion in this study was 50.5 years. Lewis and Roche (1988) reported late growth in the craniofacial skeleton extending into the fifth decade in some subjects however the authors noted that the magnitude of growth was small.
2. BMI differences between the two groups. The study population was markedly obese, with the average BMI being 32.28 +/- 0.72 $kg/m^2$. A far greater percentage of subjects were obese (BMI > 30 $kg/m^2$) compared with the expected population average. The 1989-90 National Health Survey revealed 44% of Australian males over the age of 18 and 30% of females had a BMI > 25 $kg/m^2$ and were considered `obese. The study population had 85% (60/71) males with a BMI > 25 $kg/m^2$ and 95% (21/22) of females being obese. Relaxing the criteria for obesity to a cut-off of

30 kg/m$^2$ still sees 58% (41/71) of males and 50% (11/22) of females in the study population being classified as obese.

3. The orthognathic surgical candidate does differ from the general population in the relationship of their maxilla and mandible to each other and to the cranial base. Indeed orthognathic surgery is undertaken to correct such anomalies. The use of a population known to have a different incidence of maxillo-mandibular relationship to the general population would defeat the aim of comparison of our subject population to the "normal" population.

A decision was therefore taken to use internal controls within the population studied and to compare the study population with published cephalometric norms for a Caucasian population.

Males were over represented in the study population when compared to the general population. Seventy seven percent of the subjects were male compared with 49.2% of the South Australian population in the 1996 census. This bias with an increased number of men being referred for assessment of possible obstructive sleep apnoea is not unexpected. The literature also reports an increased incidence of men with obstructive sleep apnoea compared to women (Ancoli-Israel, 1989; Guilleminault and Dement, 1978; Kales et al, 1985; and Young et al, 1993).

This study did not attempt to discriminate between subjects on the basis of racial origin, sex or the presence or absence of teeth. Measurements from cephalometric radiographs have been found to vary according to racial origin by a number of authors (Alcade et al, 1998; Cooke and Wei, 1988; Lee et al, 1997; Miyajima et al, 1996; Park et al, 1989; Redline et al, 1997; and Shen et al, 1994). The purpose of this study was to determine if the lateral cephalometric radiograph may be useful alone or as an adjunct to other diagnostic tests in determining the likelihood of a person to suffer obstructive sleep apnoea. Further studies could be done with an homogenous population with respect to racial origin and sex to determine if the lateral cephalometric radiograph becomes more or less sensitive as a screening tool. The use of internal controls i.e. using one part of the study group and comparing them with the remainder meant these factors should have little influence over the findings. Additionally the purpose of this study was to look at a representative sample of the South Australian population referred for assessments of snoring or obstructive sleep apnoea. The population studied reflects that referred to the largest public sleep disorders clinic in the state, with no known influences over those practitioners referring to the clinic.

The use of a more homogenous population e.g. the Caucasian male might have produced different results. However the aim of assessing the usefulness of the lateral cephalometric

radiograph as a screening tool for the population suspected of obstructive sleep apnoea would not have been met.

## 12.2 Materials and methods

Lateral cephalometric radiographs when taken with correct exposure do allow visualization of both skeletal and soft tissue profiles. The use of these radiographs in the assessment of subjects regarding possible UAW anomalies necessitates production of a radiographic image that allows accurate location of cephalometric landmarks. Problems of image quality will inevitably arise and it is imperative that the radiographer is cogniscant of the fact that the radiograph must extend inferiorly to include the hyoid and be "soft" enough to allow soft tissue detail without compromising the skeletal image. The use of a radiopaque mouth wash prior to imaging would allow greater definition of the soft tissue outlines of the UAW however there is an added cost and risk of adverse reaction by a few patients to the use of these materials.

CT and MRI have both been utilized to gain a three dimensional view of the airway. CT was used as a dynamic imaging modality with exposure of the upper airway occurring during patient respiration (Schwab, Gefter, Hoffman et al, 1993 and Schwab, Gefter, Pack et al, 1993). Cine-CT was used by Kuna et al (1988) to visualize the upper airway during Muellers manouvre. MRI can only be used to measure the static airway due to the time taken for image generation, and this modality has been used in obstructive sleep apnoeic patients by Schwab et al (1995). Lateral cephalometric radiographs only allow visualisation of the upper airway in a static position. Thus alterations that may occur as a result of respiration or swallowing may not be identified. The stage of the respiratory cycle at which the lateral cephalometric radiograph is exposed has been investigated. Loube et al (1995) found no variation in any of the parameters measured from cephalometric radiographs taken either during inspiration or expiration. deBerry-Borowiecki et al (1988) exposed the lateral cephalometric radiographs during the expiratory phase stating that this was the time of maximal relaxation of all structures, particularly the tongue. This study utilised the same technique.

The measurement of the upper airway in a lateral dimension by CT and MRI has been reported. A decreased diameter was found in obstructive sleep apnoeic subjects on CT by Kuna et al (1988) and Shephard et al (1990). Schwab et al (1990) used MRI and identified a similar narrowing of the upper airway in a lateral direction. Obviously a lateral cephalometric radiograph cannot detect such altered dimensions.

Lowe et al (1995) used CT to determine the volume of the upper airway in obstructive sleep apnoeic subjects and detected larger soft tissue volumes and smaller upper airway volumes in obstructive sleep apnoeic subjects. Lateral cephalometric radiographs can allow measurement of similar alterations in area, but obviously determining tissue and space volume is not possible.

CT, MRI and lateral cephalometry cannot be used whilst the patient is sleeping and therefore rely on identifying abnormalities in upper airway dimension of awake patients. Lateral cephalometric radiography has the same limitation. MRI is better at visualizing the soft tissues surrounding the upper airway and does not expose the patient to radiation, however it is a more expensive modality and is not as widely available as CT. Both of these modalities are limited to some extent by the obesity of the patient, with 300 pounds being the upper weight limit to allow the patient to fit the machine and/or the table.

During this study it was noted radiograph quality varied and this impacted upon the ability to locate accurately two soft tissue points in particular, the tip of the tongue and the tip of the uvula. The use of a radiopaque mouthwash could have allowed more precise location of these two important cephalometric landmarks.

Image quality may have been variable for a number of reasons. Although a radiographer supervises the production of these images, the Adelaide Dental Hospital is an undergraduate teaching institution. Final year dental students are rostered to the Dental Radiology Unit in order to learn the skills necessary to take both intraoral and extraoral radiographs pertinent to the practice of dentistry. In such a situation it is inevitable that there will be variation in image quality. A single qualified radiographer would ideally undertake the production of adequate lateral cephalometric radiographs for the purposes of this study.

The position of the patient when the lateral cephalometric radiograph is taken has also been investigated. Obviously people are in a supine position during sleep and the standard lateral cephalometric radiograph is taken in the upright (standing or seated) position. There is disagreement in the literature as to whether patient position influences the position of the hard and soft tissues. Hoffstein et al (1991) and Pracharktam et al (1994) found no significant alteration in parameters measured on lateral cephalometric radiographs in changing from upright to supine. There are reports of anterosuperior movement of the hyoid when supine (Ono et al, 1996) with a corresponding increase in oropharyngeal airway space (Yildirim et al, 1991). The nasopharyngeal airway has been reported to decrease (Pae et al, 1994; and Yildirim et al, 1991) and in contrast to Yildirim et al (1991) the oropharyngeal airway space was also found to decrease by Pae et al (1994). This latter group also found the cross-sectional area of the tongue increased in obstructive sleep apnoeic subjects but not in controls.

Neck circumference was recorded with the aim of determining the predictive value of this measure for elevated BMI. This study did find that an increased neck circumference correlated with an increased BMI, in agreement with Katz et al (1990); Lowe et al (1995); Ono et al (1996) and Zucconi et al (1993). There was also a correlation between increasing neck circumference and an increased incidence of obstructive sleep apnoea. This correlation has also been reported by Davies et al (1992); Hoffstein and Mateika (1992); Katz et al (1990); and Ferguson et al (1995).

Records were incomplete for twenty-two of the ninety-four subjects (18%) with respect to neck circumference. Neck circumference was to be measured at the time the lateral cephalometric radiograph was taken by the clinician ordering the radiograph. Subjects did not always return to the Oral and Maxillofacial Surgery unit following the taking of the radiograph, being discharged by the radiology unit, who subsequently returned the casenotes of the subject to the unit. The absence of a complete set of records for these twenty two subjects does not impact upon the central aim of this thesis, that being the utilization of lateral cephalometric radiographs in the assessment of the obstructive sleep apnoeic patient.

## 12.3 Criteria For Diagnosis of Obstructive Sleep Apnoea

In this study the respiratory disturbance index (RDI) alone, as measured during an overnight polysomnographic study, was used as the criteria for diagnosis of obstructive sleep apnoea. The principle reason for use of this diagnostic criterion alone was the relative simplicity of the measure and the fact most previous studies in the literature have relied upon this measure. The difficulty in the use of this parameter arises when deciding at what level of RDI is a subject diagnosed with obstructive sleep apnoea. This issue is yet to be satisfactorily resolved in the literature, hence other diagnostic criterion have been used in addition to or in place of RDI. Such criteria include the apnoea index and the minimum arterial oxygen saturation.

A definition of OSA arising from the 1990 meeting of the American Sleep Disorders Association described OSA as being " . . . characterized by repetitive episodes of UAW obstruction that occur during sleep, usually associated with a reduction in blood oxygen saturation..." with associated features of daytime sleepiness and snoring (Thorpy, 1998).

The consensus in the literature is a RDI > 10 events per hour or an AI > 5 events per hour is abnormal. Several studies that have undertaken cephalometric evaluation of obstructive sleep apnoeic patients have used an RDI > 10 as the diagnostic level for obstructive sleep apnoea (Bacon et al, 1989; Johns et al, 1998; Lowe et al, 1996; Maltais et al, 1991; Mochizuki et al, 1996; Ono et al, 1996; Ozbek et al, 1998; Tangugsorn et al, 1995a; Tangugsorn et al,

1995b and Zucconi et al, 1993). The analysis of the data was initially performed by dividing the pool of subjects into two groups separated at this level of RDI. The problems with this separation are twofold. Firstly the subjects with a RDI close to 10 are by all definitions at worst mild obstructive sleep apnoeics, and many would argue a cut off RDI > 20 better defines an obstructive sleep apnoeic subject, particularly as this level of respiratory disturbance during sleep has been reported to result in an increased mortality (He et al, 1988). The possibility of skewing the results by including patients who may indeed not be true obstructive sleep apnoeics and thus not recognizing significant measurable differences on cephalometric radiographs of patients with more severe obstructive sleep apnoea is possible. Secondly the mortality associated with obstructive sleep apnoea is reported to be most relevant in those subjects less than 50 years of age with an RDI > 20 (He et al, 1988). The relevance of using this lower cut-off RDI can again be questioned on the grounds of limited clinical significance. The value of treating patients with 10 < RDI < 20 is of questionable importance and beyond the scope of this thesis.

The arbitrary point RDI = 15 was used for the second series of analyses because this level divided the group into even numbers for the purposes of data analysis (n = 45 RDI < 15 and n = 48 RDI > 15). Some authors have considered this level of respiratory disturbance to be a suitable demarcation point between snorer/mild apnoeic subjects and apnoeic patients (Ferguson et, 1996; Mayer et al, 1996; and Schwab et al, 1995).

Arguably the most significant clinical level of respiratory disturbance to be analyzed in the study of patients with obstructive sleep apnoea is an RDI > 20. This level of respiratory disturbance has been positively linked with the development of significant morbidity and increased mortality if untreated (Benaim et al, 1992; He et al, 1988; Partinen et al, 1988; and Pracharktam et al, 1996). The majority of the literature reporting on the treatment of obstructive sleep apnoea where a lateral cephalometric radiograph has been obtained as a pretreatment investigation have used an RDI > 20 as the minimum RDI indicating the need for treatment (Conradt et al, 1997; Hochban et al, 1994; and Powell and Riley, 1993). Interestingly these same studies commonly use a criteria for success or otherwise of their treatment an RDI < 10 (Conradt et al, 1997; Hochban et al, 1994; and Johnson and Chinn, 1994).

## 12.4 Differences In Lateral Cephalometric Radiograph Measures Between Snorers and OSA Subjects

There were differences in which cephalometric measures were significantly altered between subjects relative to the respiratory disturbance index. In contrast measures of neck

circumference and BMI were correlated with increasing severity of obstructive sleep apnoea as defined by the apnoea-hypopnoea index. The discussion will consider each discrete anatomic area analysed by linear and angular variables.

### 12.4.1 Soft Palate

The soft palate has long been of interest to clinicians managing the often complex and frequently inter-related problems of snoring and obstructive sleep apnoea. The altered dimensions of the soft palate are one of the most consistent findings in studies of OSA patients using cephalometry. Many investigators have postulated the dimensions of the soft palate are greater in patients who snore or suffer OSA thus predisposing them to UAW occlusion at this level. Fujita et al (1981) used direct visualisation to determine soft palate dimensions and reported an increased size in snoring patients. This increased length has been used to justify uvulopalatopharyngoplasty (UPPP) in the treatment of both snoring and obstructive sleep apnoeic patients (Fujita et al, 1981 and Riley et al, 1987). The justification is the assumption that excessive length or thickness of the soft palate is instrumental in the development of obstructive episodes. Removal of this redundant soft tissue should prevent the development of an obstructive event, thereby alleviating the potential serious consequences of obstructed breathing during sleep.

The length, thickness, area and volume of the soft palate have been of interest to investigators involved in the diagnosis and treatment of OSA since this time. The measurements of the soft palate from subjects in the current study are presented in Table 12.4-1 and are compared with results from the literature review.

Soft palate length (PNS – P) is reported to be significantly increased in patients with obstructive sleep apnoea syndrome (Bacon et al, 1989; deBerry-Borowiecki et al, 1988; Hochban and Brandenburg, 1994; Lowe et al, 1996; Lyberg et al, 1989b; Maltais et al, 1991; Mayer et al, 1996; Mochizuki et al, 1996; Ono et al, 1996; Pracharktam et al, 1996; Sakakibara et al, 1999; Strelzow et al, 1988; Tangugsorn et al, 1995b and Zucconi et al, 1993). The increased soft palate length in obstructive sleep apnoea is suggested to cause obstruction by falling across the nasopharynx during sleep, particularly if the patient is supine. In this study the length of the soft palate from the posterior nasal spine to the tip of the uvula (PNS – P) was not significantly increased in OSA subjects. The p value of 0.062 was approaching significance. This result is in agreement with Andersson and Brattstrom et al (1991); Battagel and L'Estrange (1996); Johns et al (1998); Mayer and Meier-Ewert (1995) and Pracharktam et al (1994) who also found no increase in soft palate length in obstructive sleep apnoeic subjects compared to a control population.

Although this study did not find a significant difference in soft palate length between snorers and OSA subjects this dimension may play a role in the aetiology of obstruction in some patients. Treatment modalities aimed at the reduction in soft palate dimension, such as UPPP have been demonstrated to achieve a reduction in the RDI. However a comprehensive review of the literature by Schechtman et al (1995) reported an overall success rate of 54% using the criteria for success the reduction of the RDI by 50%. These results are disturbing as 46% of patients continue to register an RDI that maintains an elevated risk of comorbidities such as CVA, MI or impaired concentration.

One of the problems in determining the length of the soft palate from a lateral cephalometric radiograph is visualising the tip of the uvula. This study did not use a contrast dye mouthwash, such as Barium Sulfate Oesophageal Cream, prior to completion of the radiograph that would have facilitated location of this cephalometric landmark. The literature on the use of cephalometric radiographs does not usually mention the use of a radiopaque marker to assist in the definition of soft tissue outlines, however Hans and Goldberg (1995) do suggest the use of a radiopaque marker.

The greatest thickness of the soft palate was also measured on all lateral cephalometric radiographs. This study found a significantly increased maximum soft palate width (UW1 – UW2) as RDI increased for all patient groupings according to RDI. This result is supported by the findings of deBerry-Borowiecki et al (1988); Hochban and Brandenburg (1994); Lowe et al (1996); Lyberg et al (1989b); Ono et al (1996); Strelzow et al (1988) and Tangugsorn et al (1995b). This is in contrast to Andersson and Brattstrom (1991); Battagel and L'Estrange (1996); Bacon et al (1989); Mayer et al (1996) and Sakakibara et al (1999) who found no such difference in soft palate width.

Thickness of the soft palate is positively correlated with BMI, suggesting that this functional area is affected by an increase in weight, presumably by fat deposition. Increased soft palate thickness may result in a narrowed distance between the soft palate and the posterior pharyngeal wall. This would be expected to increase the tendency for obstruction at the nasopharyngeal level of the upper airway. This will be discussed in section 12.4.2. Alternatively, the thickened soft palate may protrude further into the oral cavity and in this instance there may be no increased tendency for obstruction of the UAW. A positive correlation between RDI and soft palate thickness, as found in this study, would support the hypothesis that soft palate dimensions do play a role in the aetiology of OSA.

**Table 12.4-1: Soft Palate Linear and Angular Dimensions**

| | | | UD | UL | UV | ANS-PNS-UT | CL |
|---|---|---|---|---|---|---|---|
| Andersson et al (1991) | A vs C | U | | NS | | | |
| Battagel et al (1996) | A vs C | U | | NS | 0.014 | NS | |
| Bacon et al (1989) | A vs C | U | | | 0.05 | NS | |
| deBerry-Borowiecki et al (1988) | A vs C | U | 0.000 | 0.010 | 0.000 | | |
| Hochban et al (1994) | A vs C | U | 0.005 | 0.0001 | | NS | |
| Johns et al (1998) | A vs S | U | | NS | | | |
| Lowe et al (1996) I | A vs C | U | 0.007 | 0.002 | 0.000 | | |
| Lowe et al (1996) I | A vs C | S | NS | NS | NS | | |
| Lowe et al (1996) II | A vs C | U | 0.023 | 0.009 | 0.001 | | |
| Lowe et al (1996) II | A vs C | S | 0.034 | NS | 0.031 | | |
| Lowe et al (1996) III | A vs C | U | NS | NS | NS | | |
| Lowe et al (1996) III | A vs C | S | NS | NS | NS | | |
| Lyberg et al (1989b) | A vs C | U | 0.001 | 0.001 | 0.001 | 0.01 | 0.01 |
| Maltais et al (1991) | A vs C | U | | 0.01 | | | |
| Mayer et al (1995) | A vs C | U | | NS | | | |
| Mayer et al (1996) | A vs S | U | NS | 0.01 | | | |
| Mochizuki et al (1996) | A vs C | U | | 0.01 | 0.01 | | |
| Ono et al (1996) | A vs C | U | 0.05 | 0.05 | 0.05 | | |
| Ono et al (1996) | A vs C | S | NS | 0.05 | 0.05 | | |
| Pracharktam et al (1994) | A vs S | U | | NS | NS | | |
| Pracharktam et al (1996) | A vs S | U | | 0.013 | | | |
| Sakakibara et al (1999) | A1 vs C | U | NS | 0.02 | 0.001 | | |
| Sakakibara et al (1999) | A2 vs C | U | NS | 0.001 | 0.001 | | |
| Sakakibara et al (1999) | A1 vs A2 | U | NS | NS | NS | | |
| Strelzow et al (1988) | A vs C | U | 0.05 | 0.05 | 0.05 | | |
| Tangugsorn et al (1995b) | A vs C | U | 0.001 | 0.001 | 0.001 | 0.05 | 0.001 |
| Zucconi et al (1992) | A vs S | U | | 0.05 | | | |
| **Sherring (2001) RDI > 10** | **A vs S** | **U** | **0.01** | **NS** | | | |
| **Sherring (2001) RDI > 15** | **A vs S** | **U** | **0.02** | **NS** | | | |
| **Sherring (2001) RDI > 20** | **A vs S** | **U** | **0.002** | **NS** | | | |

A = obstructive sleep apnoea subject; C = non-snoring, non-apnoeic subject; S = snoring, non-apnoeic subject; A1 = non-obese obstructive sleep apnoea subject; A2 = Obese obstructive sleep apnoea subject.

## 12.4.2 Nasopharynx

The nasopharyngeal airway lies behind the soft palate and has been measured in its anteroposterior dimension and area by a number of authors using lateral cephalometric radiographs. If the distance between the posterior edge of the soft palate was narrowed then it is conceivable obstruction may occur, even if the dimension of the soft palate were not increased. Thus in considering obstruction at this level of the UAW it may be wise to

consider not only the individual dimension of the soft palate but also the nasopharyngeal dimensions.

This study did not find a significant difference in the anteroposterior dimension of the nasopharyngeal airway using measurements taken at five different levels. One of the problems when comparing the nasopharyngeal airway width between studies is the use of different reference planes and cephalometric landmarks by different authors. The uvula tip is a relatively simple landmark to identify on most radiographs and was chosen as one of the cephalometric landmarks on the soft palate. The second cephalometric point on the soft palate was the point of greatest convexity as reported by deBerry-Borowiecki et al (1988). This point correlates with the posterior superior point on the soft palate as reported by Lowe et al (1986). The third landmark used was the posterior nasal spine. This landmark is simple to locate on a cephalometric image, is easily reproducible and has been used by a number of authors (Andersson and Brattstrom, 1991; deBerry-Borowiecki et al, 1988; Hochban et al, 1994; Lyberg et al, 1989b; Sakakibara et al, 1999; Solow et al, 1996; Strelzow et al, 1988 and Tangugsorn et al, 1995b). These factors make it a useful landmark to allow comparison between studies.

The reference planes used in this study to consider the width of the nasopharyngeal airway were constructed parallel to Frankfort Horizontal and a line joining point B and gonion. Lines parallel to these two reference planes were constructed to pass between the three landmarks mentioned in the previous paragraph and extended posteriorly to intersect with the posterior pharyngeal wall. Two planes were chosen to allow comparison with a larger number of studies in the literature.

The distance between the posterior pharyngeal wall and the most posterior superior point on the soft palate parallel to Frankfort Horizontal (UT – PhW1) was significant if an RDI > 20 defined OSA subjects. This dimension was not significantly decreased if the subjects were divided based on a lower RDI. This suggests that in the group studied nasopharyngeal airway narrowing is significant only as RDI increases, however there was no significant correlation between RDI and this distance for the whole group. Lowe et al (1996) found this distance significantly decreased in all subjects with an RDI > 10. deBerry-Borowiecki et al (1988) used a more complex criteria for determining OSA however they too found this distance significantly decreased in all subjects with OSA.

The distance from the tip of the uvula to the posterior pharyngeal wall along the line Go – B (UT – PhW5) was not significantly decreased irrespective of the RDI used to define OSA. However if an RDI > 20 is used to define OSA subjects then a p value of 0.051 approaches significance. This finding is supported by Johns et al (1998) and Mayer and Meier-Ewert

(1995). The inference from this finding is obstruction of the nasopharyngeal airway at the level of the uvula is likely to be a function of soft palate length rather than nasopharyngeal airway width.

Narrowing of the UAW at any point has been postulated to cause pressure changes that may predispose to obstruction at another site. The distance from the posterior nasal spine is the most superior point of the pharyngeal airway, and the distance to the posterior pharyngeal wall from this point was measured. The distance from the posterior nasal spine to the posterior pharyngeal wall differs from other measures of nasopharyngeal airway width because a fixed bony point is being used. The superior constrictor arises from the base of skull at the medial pterygoid plate and the pterygoid hamulus and is unlikely to have enough elasticity to occlude totally at this level. This study did not find significant narrowing of the nasopharyngeal airway at this point (PNS – PhW3), in agreement with deBerry-Borowiecki et al (1988).

A number of explanations have been offered to explain the inability to measure a decreased nasopharyngeal airway width on a lateral cephalometric radiograph yet determine by other measures that this is indeed the principle site of UAW obstruction. In some cases this has been attributed to the simple assumption that OSA patients do not have narrowed upper airways during wakefulness and therefore no abnormality can be visualized on subjects who are awake during imaging (Hans and Goldberg, 1995). Schwab et al (1995) suggest a coronal narrowing of the UAW with no corresponding reduction in the sagittal dimension. They utilised MRI to assess the lateral dimension of the UAW. In contrast Riley et al (1987) found a significant correlation between the posterior airway space measured by cephalometrics and the volume of the pharyngeal airway. The results of the current study are shown with the results from the literature review in Table 12.4-2. The reliability of lateral cephalometric radiographs for predicting the nasopharyngeal airway as the site of obstruction if only pharyngeal airway space is measured is not clear as the conflicting reports in the literature indicate.

**Table 12.4-2: Nasopharyngeal Airway Dimensions**

| | | | PNS – PhW | UP-PhW | UT-PhW | Area |
|---|---|---|---|---|---|---|
| Andersson et al (1991) | A vs C | U | 0.001[1] | | | |
| Battagel et al (1991) | A vs C | U | | 0.000[2] | 0.005[2] | |
| Battagel et al (1991) | A vs C | U | | 0.000[2,3] | | |
| Battagel et al (1991) | A vs C | U | | 0.000[4] | | |
| deBerry-Borowiecki et al (1988) | A vs C | U | NS[5] | 0.002[5] | 0.005[5] | NS |
| Hochban et al (1994) | A vs C | U | 0.0001[1] | | 0.001[6] | |
| Hochban et al (1994) | A vs C | U | 0.0001[7] | | | |
| Johns et al (1998) | A vs S | U | | 0.01[8] | NS[5] | |
| Lowe et al (1996) I | A vs C | U | | 0.000[9] | 0.000[9] | NS |
| Lowe et al (1996) I | A vs C | S | | 0.035[9] | 0.027[9] | NS |
| Lowe et al (1996) II | A vs C | U | | 0.000[9] | 0.01[9] | NS |
| Lowe et al (1996) II | A vs C | S | | 0.001[9] | NS[9] | NS |
| Lowe et al (1996) III | A vs C | U | | NS[9] | NS[9] | NS |
| Lowe et al (1996) III | A vs C | S | | NS[9] | NS[9] | NS |
| Lyberg et al (1989b) | A vs C | U | 0.001[9] | | 0.001[9] | |
| Mayer et al (1995) | A vs C | U | | | NS[9] | |
| Mochizuki et al (1996) | A vs S | U | | NS[7] | | |
| Ono et al (1996) | A vs C | U | | | | NS |
| Pracharktam et al (1994) | A vs S | U | | 0.05[9] | | |
| Pracharktam et al (1996) | A vs S | U | | NS[9] | | |
| Sakakibara et al (1999) | A1 vs C | U | 0.001[7] | 0.001[7] | 0.001[7] | |
| Sakakibara et al (1999) | A2 vs C | U | 0.001[7] | 0.001[7] | 0.001[7] | |
| Sakakibara et al (1999) | A1 vs A2 | U | NS[7] | NS[7] | NS[7] | |
| Solow et al (1996) | A vs C | U | 0.01[9] | 0.001[10] | 0.001[10] | |
| Solow et al (1996) | A vs C | U | 0.001[7] | | | |
| Strelzow et al (1988) | A vs C | U | NS[10] | 0.05[10] | 0.05[10] | 0.05 |
| Tangugsorn et al (1995b) | A vs C | U | 0.001[7] | | 0.001[7] | |
| **Sherring (2001) RDI >10** | **A vs S** | **U** | **NS[5]** | **NS[5]** | **NS[5]** | |
| **Sherring (2001) RDI >10** | **A vs S** | **U** | | **NS[9]** | **NS[9]** | |
| **Sherring (2001) RDI > 15** | **A vs S** | **U** | **NS[5]** | **NS[5]** | **NS[5]** | |
| **Sherring (2001) RDI > 15** | **A vs S** | **U** | | **NS[9]** | **NS[9]** | |
| **Sherring (2001) RDI > 20** | **A vs S** | **U** | **NS[5]** | **NS[5]** | **0.05[5]** | |
| **Sherring (2001) RDI > 20** | **A vs S** | **U** | | **NS[9]** | **NS[9]** | |

A = obstructive sleep apnoea subject; C = non-snoring, non-apnoeic subject; S = snoring, non-apnoeic subject; A1 = non-obese obstructive sleep apnoea subject; A2 = Obese obstructive sleep apnoea subject.

1 Distance along extension of line ANS-PNS; 2 Distance along a horizontal line; 3 The most posterior point on the dorsum of the soft palate; 4 Distance along a horizontal line through the lower incisor tip; 5 Distance along a line parallel to FH; 6 Distance along extension of the occlusal plane; 7 Distance along line PNS-Ba (or parallel) between soft palate and pharyngeal wall; 8 Distance along line parallel to FH at midpoint of soft palate; 9 Distance along line parallel to Go-B; 10 Reference line not defined

### 12.4.3 Oropharynx

Narrowing of the posterior airway space at the level of the oropharynx has also been investigated using lateral cephalometric radiographs. The oropharyngeal airway width is determined by the relationship of the dorsal surface of the tongue base to the posterior pharyngeal wall. Loss of muscle tone that occurs during sleep (Chandler, 1988) has been postulated as a cause for increased narrowing or occlusion of the UAW due to a "slumping" of the tongue whilst supine (Riley et al, 1993).

There is disagreement in the literature regarding the ability of a lateral cephalometric radiograph taken on an awake patient in the upright position to accurately show the degree of UAW narrowing that may be present during sleep. Pracharktam et al (1994) reported a significant narrowing of the nasopharyngeal airway in both snoring and apnoeic patients when changed from the upright to supine position. They reported no other parameter to be significantly altered between upright and supine cephalometric radiographs. Horner et al (1989) imaged sleeping, supine OSA subjects with conventional CT and found UAW obstruction occurred by posterior displacement of the soft palate and tongue. These parameters may be analysed by cephalometry, however lateral pharyngeal airway collapse was also noted which cannot be seen on a lateral view.

The present study found no significant difference in the oropharyngeal airway dimension measured from the dorsal tongue to the posterior pharyngeal wall along an extension of the line Go-B (TP – PhW6). This finding is in agreement with Johns et al (1998); Mayer G and Meier-Ewert K (1995); Mayer et al (1996); Mochizuki et al (1996); Pracharktam et al (1994), and Solow et al (1996). Only one study in the literature considered the effect of body position on this parameter. Lowe et al (1996) found no significant difference in this dimension measured from supine radiographs. In contrast a number of authors (Battagel and L'Estrange, 1996; Lowe et al, 1996; Lyberg et al, 1989b; Maltais et al, 1991; and Sakakibara et al, 1999) have measured a significant decrease in the distance between the tongue and the pharyngeal wall at the level of an extension of Go-B. These results are shown in Table 12.4-3

The literature is not clear on the effectiveness of measuring the width of the UAW on lateral cephalometric radiographs to aid diagnosis of the site of obstruction. The current study found little difference in the width of the oropharyngeal airway between OSA subjects and snoring subjects, irrespective of the RDI used to define OSA. The value of a lateral cephalometric radiograph alone in predicting the likely site of pharyngeal obstruction is limited because of the possibility of lateral wall narrowing or collapse. Analysis to determine if there is a relationship between any of the soft palate or tongue dimensions, particularly

length, and pharyngeal width may be of greater benefit. Pharyngeal width, soft palate length or tongue length alone may not be significantly different in obstructive sleep apnoeic subjects. However in subjects being investigated for obstructive sleep apnoea an increased soft palate or tongue length and a decreased pharyngeal width in combination may produce a greater degree of narrowing and risk of obstruction.

**Table 12.4-3: Oropharyngeal Airway Dimensions**

| | | | PAS (OP) | PAS(Go-B) | PAS (Go-Gn) | Area |
|---|---|---|---|---|---|---|
| Battagel et al (1991) | A vs C | U | | 0.036[1] | | |
| Hochban et al (1994) | A vs C | U | 0.002 | | 0.005 | |
| Johns et al (1998) | A vs S | U | | NS | | |
| Lowe et al (1996) I | A vs C | U | | 0.001 | | 0.000 |
| Lowe et al (1996) I | A vs C | S | | NS | | NS |
| Lowe et al (1996) II | A vs C | U | | 0.015 | | 0.007 |
| Lowe et al (1996) II | A vs C | S | | NS | | NS |
| Lowe et al (1996) III | A vs C | U | | NS | | NS |
| Lowe et al (1996) III | A vs C | S | | NS | | NS |
| Lyberg et al (1989b) | A vs C | U | | 0.051 | | NS |
| Maltais et al (1991) | A vs C | U | | 0.051 | | |
| Mayer et al (1995) | A vs C | U | | NS | | |
| Mayer et al (1996) | A vs S | U | | NS | | |
| Mochizuki et al (1996) | A vs S | U | | NS | | |
| Ono et al (1996) | A vs C | U | | | | NS |
| Pracharktam et al (1994) | A vs S | U | | NS | | |
| Sakakibara et al (1999) | A1 vs C | U | | 0.001[1] | | |
| Sakakibara et al (1999) | A2 vs C | U | | 0.001[1] | | |
| Sakakibara et al (1999) | A1 vs A2 | U | | NS | | |
| Solow et al (1996) | A vs C | U | | NS[1] | | |
| Tangugsorn et al (1995b) | A vs C | U | | | 0.001 | 0.01 |
| Zucconi et al (1992) | A vs S | U | | | NS | |
| **Sherring (2001) RDI > 10** | **A vs S** | **U** | | **NS** | | |
| **Sherring (2001) RDI > 15** | **A vs S** | **U** | | **NS** | | |
| **Sherring (2001) RDI > 20** | **A vs S** | **U** | | **NS** | | |

A = obstructive sleep apnoea subject; C = non-snoring, non-apnoeic subject; S = snoring, non-apnoeic subject; A1 = non-obese obstructive sleep apnoea subject; A2 = Obese obstructive sleep apnoea subject.

1 The narrowest distance between the tongue and the posterior oropharyngeal wall.

## 12.4.4 Tongue

Increased tongue length, similar to soft palate length, may contribute to an increased RDI during sleep because of loss of muscle tone and a posterior resting position. Locating the

tongue tip and measuring the distance to the tip of the epiglottis (TT – Et) or the vallecula (TT – Eb, or TGL) is the most widely reported measure of tongue length.

The difficulty with determining tongue length from a standard lateral cephalometric radiograph again lies with difficulty in locating the tongue tip. This is a similar problem already discussed with respect to the soft palate tip. A modified radiographic technique, utilizing a radiopaque dye would assist in improving the accurate identification of this point. Accurate recording of the rest position of the tongue requires a strict protocol when obtaining a lateral cephalometric radiograph. The tongue is a particularly mobile structure within the UAW and the protocol followed in this study was to have the patient swallow and exhale prior to exposure of the radiographic film. This protocol is used for all lateral cephalometric radiographs taken within Dental Radiology Unit, Adelaide Dental Hospital. Exhalation prior to exposure is reported to result in maximal relaxation of all structures of the UAW (deBerry-Borowiecki et al, 1988). The tongue tip (TT) was the least reproducible point on the horizontal axis in this study and was the third least reliable point on the vertical axis after points posterior pharyngeal wall 3 (PhW3) and posterior pharyngeal wall 4 (PhW4). There was not a significant difference between the first and second determinations of point TT. This problem with reproducibility of this point means longitudinal comparison of changes in the position of this landmark, for example following surgery aimed at altering tongue position, is difficult. The use of a radiopaque dye prior to exposure of the lateral cephalometric film would assist in accurate location of the tongue tip. Given the mobility of the tongue, the point on the tongue identified as the "tip" may still vary between cephalometric radiographs.

This study found a significant increase in the length of the tongue measured from the tip of the tongue to vallecula (TGL) for OSA subjects as defined by an RDI > 15 and an RDI > 20 (Table 12.4-4). The OSA subjects as defined by an RDI > 10 approached significance with a p value of 0.057. These findings are in agreement with deBerry-Borowiecki et al (1988); Hochban and Brandenburg (1994); Lowe et al (1996); Pracharktam et al (1994); Pracharktam et al (1996); Sakakibara et al (1999); and Strelzow et al (1988) who also found an increased tongue length in all patients diagnosed with OSA irrespective of severity.

Lowe et al (1996) found no significant difference in tongue length, from the tongue tip to vallecula, between OSA subjects with a Class I or Class II dental malocclusion on supine or upright radiographs. Interestingly they did find a significant difference for OSA subjects with a Class III dental malocclusion on supine cephalometric radiographs, but not on upright radiographs. They concluded that this group of patients had an elongated airway secondary to increased tongue length, but did not proffer an aetiology for the increase in tongue length seen. One hypothesis is in subjects with a Class III dental malocclusion the mandible may be

lengthened. This increase in mandibular length would influence the dimensions of the attached musculature, including the tongue. The tongue may be lengthened due to the increased mandibular length.

Studies by deBerry-Borowiecki et al (1988) and Hochban and Brandenburg (1994) did not find a significant increase in tongue length as measured from the tongue tip to the tip of the epiglottis (TT – Et). This was true for the present study if OSA was defined as an RDI > 10. If however, an RDI > 15 or an RDI > 20 was used as the diagnostic criteria for OSA there was a significant increase in tongue length as measured by this parameter.

The findings of this study suggest that as OSA severity increases, so to does tongue length. This would be consistent with the hypothesis that in some OSA patients loss of tongue muscle tone during sleep allows the tongue to fall across the airway, producing an obstructive episode.

**Table 12.4-4: Tongue Dimensions**

| | | | V - TT | Depth | Height | Length | Area | Proportion |
|---|---|---|---|---|---|---|---|---|
| Battagel et al (1996) | A vs C | U | | | | | NS | 0.01[9] |
| deBerry-Borowiecki et al (1988) | A vs C | U | 0.003 | 0.000[1] | | NS[2] | 0.002 | |
| deBerry-Borowiecki et al (1988) | A vs C | U | 0.0083 | NS[4] | | NS[5] | | |
| Hochban et al (1994) | A vs C | U | 0.000[1] | 0.002[6] | 0.000[1] | 0.01[7] | | |
| Hochban et al (1994) | A vs C | U | 0.005[8] | 0.0001[9] | | NS[10] | | |
| Hochban et al (1994) | A vs C | U | NS[3] | 0.0001[11] | | | | |
| Hochban et al (1994) | A vs C | U | | 0.01[12] | | | | |
| Johns et al (1998) | A vs S | U | | 0.01[13] | NS | | | |
| Lowe et al (1996) I | A vs C | U | 0.045 | | NS | | NS | |
| Lowe et al (1996) I | A vs C | S | NS | | NS | | NS | |
| Lowe et al (1996) II | A vs C | U | 0.045 | | 0.015 | | NS | |
| Lowe et al (1996) II | A vs C | S | NS | | NS | | NS | |
| Lowe et al (1996) III | A vs C | U | NS | | 0.030 | | NS | |
| Lowe et al (1996) III | A vs C | S | 0.041 | | NS | | NS | |
| Lyberg et al (1995b) | A vs C | U | NS | 0.011[6] | NS | NS[14] | NS | |
| Mayer et al (1995) | A vs C | U | | NS[6] | | | | |
| Mochizuki et al (1996) | A vs S | U | | | | | 0.05 | |
| Pracharktam et al (1994) | A vs S | U | 0.01[15] | | | | NS | |
| Pracharktam et al (1996) | A vs S | U | 0.006[15] | | | | 0.004 | 0.01 |
| Sakakibara et al (1999) | A1 vs C | U | NS | 0.001 | 0.02 | | 0.001 | |
| Sakakibara et al (1999) | A2 vs C | U | 0.001 | 0.001 | 0.001 | | 0.001 | |
| Sakakibara et al (1999) | A1 vs A2 | U | 0.001 | NS | NS | | 0.001 | |
| Strelzow et al (1988) | A vs C | U | 0.05[15] | 0.05[12] | | 0.05[10] | 0.05 | |
| Tangugsorn et al (1995b) | A vs C | U | NS | 0.05[15] | | | 0.001 | |
| **Sherring (2001) RDI>10** | **A vs S** | **U** | **NS[3]** | **0.05[13]** | | | | |
| **Sherring (2001) RDI>10** | **A vs S** | **U** | | **NS[15]** | | | | |
| **Sherring (2001) RDI>15** | **A vs S** | **U** | **0.037[3]** | **0.007[13]** | | | | |
| **Sherring (2001) RDI>15** | **A vs S** | **U** | | **0.010[15]** | | | | |
| **Sherring (2001) RDI>20** | **A vs S** | **U** | **0.021[3]** | **0.003[13]** | | | | |
| **Sherring (2001) RDI>20** | **A vs S** | **U** | | **0.004[15]** | | | | |

A = obstructive sleep apnoea subject; C = non-snoring, non-apnoeic subject; S = snoring, non-apnoeic subject; A1 = non-obese obstructive sleep apnoea subject; A2 = Obese obstructive sleep apnoea subject.

1 PNS-TB; 2 B-TB; 3 TT – ET; 4 Go-TB; 5 Gn-TB; 6 TB – PNS; 7 V – Me; 8 TT – TB; 9 V – S; 10 TB – B; 11 V – ANS; 12 TB – ANS; 13 PNS – EB; 14 V – C spine parallel to FH; 15 TT – EB; 16 V – FH.

### 12.4.5 Hyoid Bone

The location of the hyoid in relation to the mandible, the cervical spine and to maxillary and cranial reference points is important because the hyoid is a point of attachment for many of the muscles involved with the UAW. This hyoid is suspended by the muscles and ligaments, which are attached to it. The bone is freely mobile, being felt and seen to alter position with breathing, swallowing and movement of the tongue. Investigators concerned with oropharyngeal or hypopharyngeal obstruction have thoroughly documented hyoid bone position on lateral cephalometric radiographs.

The hyoid tended to be inferiorly placed in subjects diagnosed with OSA in this study compared with the snoring controls. This was reflected in an increased distance from the mandibular plane to the hyoid (MP – H) in the RDI > 10 and RDI > 15 groups. Interestingly the group diagnosed with OSA by an RDI > 20 had no significant difference in vertical hyoid position compared with the snoring group. The increase in distance between the mandible and the hyoid is supported in this study by the angular measurement between Go-Gn-H and C3-RGn-H. There was also significant correlation between RDI and the distance MP-H across all ninety-four subjects with the distance increasing as RDI increased

The increased MP-H distance reported in this study are in agreement with Andersson and Brattstrom (1991); Hochban and Brandenburg (1994); Lowe et al (1996); Lyberg et al (1989b); Maltais et al (1991); Mayer et al (1996); Mochizuki et al (1996); Pracharktam et al (1994); Pracharktam et al (1996); Sakakibara et al (1999); Tangugsorn et al (1995a) and Tsuchiya et al (1992). Nelson M and Hans S (1997) found the distance MP-H to be of greater predictive value of OSA severity in obese subjects compared to subjects with OSA who were not obese. These results are shown in Table 12.4-5.

MP-H distance provides only indirect information regarding the relationship of the hyoid bone to the inferior border of the mandible. This distance would appear increased if the mandibular plane angle was horizontal, or decreased if the gonial angle was increased.

Measuring the position of the hyoid bone in relation to gnathion, gonion and as the angle Go-Gn-H documents it position more accurately and is independent of patient differences in mandibular plane angle. The distance Gn-H and Go-H was considered in the present study. For groups separated by an RDI > 10 the distance Go-H was significantly increased. deBerry-Borowiecki et al (1988) and Strelzow et al (1988) also found this distance increased for subjects defined as OSA. These distances were not significantly increased in the other subjects groups, although for RDI > 15 the distance Go-H approached significance (p=0.071).

Angular measures of hyoid depth that were considered in the present study were the angle Go-Gn-H and the angle C3-RGn-H. The angular measure of hyoid depth (C3-RGn-H) was increased if an RDI > 10 or and RDI > 15 was used to define OSA. This is supported by the study of Pracharktam et al (1994). The angle Go-Gn-H was only significant if the population studied was divided into OSA subjects and snorers at an RDI > 15 level. deBerry-Borowiecki et al (1988); Hochban and Brandenburg (1994), and Strelzow et al (1988) reported an increased angle Go-Gn-H which supports the findings of this study (Table 12.4-6).

Other measures of the vertical position of the hyoid relate it to the cranial base. The distance from sella to hyoid (S – H) and articulare to hyoid (Ar – H) were all increased in OSA subjects in this study. These increases occurred when RDI > 10 was used as the diagnostic criteria for OSA and became less important as OSA severity increased. Only the distance S-H was significantly increased in OSA subjects if RDI > 15 was the defining value. When RDI > 20 was used to differentiate between OSA subjects and snorers there was no significant increase in hyoid depth between the two groups, however this distance did approach significance (p=0.057).

The findings of the present study are in agreement with deBerry-Borowiecki et al (1988); Hochban and Brandenburg, 1994; Johns et al (1998); Strelzow et al (1988) and Tangugsorn et al (1995a). In contrast Lyberg et al (1989a) found no significant difference in the distance S-H between OSA subjects and non-snoring controls. These results are shown in Table 12.4-5. The finding that vertical hyoid position is less important as RDI increases may indicate hyoid position is more important for mild OSA subjects and decreases in importance as RDI increases.

The angular measure of hyoid position in the vertical plane was also considered in this study using the angle between the cranial base (S-N) and hyoid. No significant difference for this angle was found for any of the groups. This contrasts with the findings of deBerry-Borowiecki et al (1988) and Hochban and Brandenburg (1994) who found a significantly increased angle in OSA subjects. findings agreed with those of this study. The difficulty of using this angular measure is it also has a horizontal component and is altered by the cranial base angle. Those subjects with a steep cranial base (compared to Frankfort Horizontal) will tend to have a decreased angle S-N-H compared to those with a more shallow cranial base angle where the hyoid is in the same horizontal position. A similar problem arises with the angular measure between the mandibular plane and the hyoid (angle Go-Gn-H). The reliability of these angles for determining the vertical position of the hyoid is questionable if no consideration is given to the steepness of the cranial base or mandibular plane.

The anteroposterior position of the hyoid (or horizontal position) has been considered. One of the problems with the assessment of this parameter is the tangential nature of many of the distances measured. Some of the landmarks used include point B (Battagel and L'Estrange, 1996 and), retrognathion (Lowe et al, 1996; Pracharktam et al, 1994 and Tsuchiya et al, 1992), menton (Hochban and Brandenburg, 1994) and gnathion (Strelzow et al, 1988). The current study found no significant difference between OSA subjects and snoring subjects for the distance Me-H. This is in contrast to Hochban and Brandenburg (1994) who found this distance significantly increased in OSA subjects. There was a correlation between BMI and the distance Me-H, with this distance increasing as BMI increased. This suggests a more posteriorly positioned hyoid in obese subjects. This finding is also reported by Tsuchiya et al (1992) who reported a more posteriorly positioned hyoid in subjects with OSA who had a low RDI and elevated BMI. There is no true horizontal assessment of hyoid position using these measures because most relate to a landmark on the anterior maxilla or mandible. All these constructed lines have a significant vertical component and may therefore diminish the sensitivity of the horizontal position of the hyoid because of the vectors involved.

A better estimate of the anteroposterior position of the hyoid is by its relationship to the cervical spine. The horizontal distance to a point or reference line on the cervical spine allows a true horizontal measurement to be taken, with little or no vertical component. The third cervical vertebra is on the same horizontal plane as the hyoid in most subjects and is likely to provide the most reliable landmark for evaluation of this dimension.

The present study found the hyoid to be anteriorly placed in OSA subjects compared with snorers for groups divided by RDI > 10 and RDI > 15 as measured by the distance H-C4 and H-C3. This finding is in agreement with Lowe et al (1996) who also found an increased distance for OSA subjects with a Class I or Class III dental malocclusion on upright cephalometric radiographs. This group reported no significant difference for subjects with a Class II dental malocclusion. Sakakibara et al (1999) found the hyoid to be significantly further from the cervical spine in obese OSA subjects compared to normal controls and non-obese OSA subjects. Lyberg et al (1989a) found no significant difference in these dimensions between OSA subjects and non-snoring controls. Tsuchiya et al (1992) found the hyoid was significantly more anterior in low AHI/high BMI subjects compared with controls. A more anteriorly placed hyoid was reported by Battagel and L'Estrange (1996) however they used point B as the horizontal reference, and therefore have included a vertical component to their horizontal measure of distance.

The variability of the relationship of the hyoid to the cervical spine supports the knowledge that the hyoid is mobile and its position is influenced by the muscles and ligaments that maintain its position. The literature suggests that obese OSA subjects tend to have an

increased hyoid to cervical spine distance. This study found a positive correlation between BMI and the distance from the cervical spine to the hyoid. Using RDI > 20 as the definition of OSA there was no significant difference in this distance between snorers and OSA subjects which suggests the association between RDI and the distance from the cervical spine to the hyoid is of less importance as OSA severity increases.

**Table 12.4-5: Hyoid Bone Linear Measurements**

| | | | H – MP | Vertical | Horizontal | C-Spine |
|---|---|---|---|---|---|---|
| Andersson et al (1991) | A vs C | U | 0.05 | | | |
| Andersson et al (1991) | A vs S | U | 0.01 | | | |
| Battagel et al (1996) | A vs C | U | NS | NS[1] | 0.042[2] | |
| Battagel et al (1996) | A vs C | U | | | NS[3] | |
| deBerry-Borowiecki et al (1988) | A vs C | U | | 0.009[4] | NS[5] | |
| deBerry-Borowiecki et al (1988) | A vs C | U | | 0.001[6] | NS[7] | |
| deBerry-Borowiecki et al (1988) | A vs C | U | | 0.043[8] | | |
| Hochban et al (1994) | A vs C | U | 0.000[1] | 0.0001[4] | 0.0005[2] | |
| Hochban et al (1994) | A vs C | U | | 0.05[9] | 0.05[10] | |
| Hochban et al (1994) | A vs C | U | | | NS[7] | |
| Hochban et al (1994) | A vs C | U | | | 0.005[11] | |
| Johns et al (1998) | A vs S | U | 0.01 | 0.01[12] | | |
| Lowe et al (1996) I | A vs C | U | 0.003 | 0.005[13] | NS[14] | 0.031[15] |
| Lowe et al (1996) I | A vs C | S | 0.001 | 0.005[13] | NS[14] | NS[15] |
| Lowe et al (1996) II | A vs C | U | 0.031 | NS[13] | NS[14] | NS[15] |
| Lowe et al (1996) II | A vs C | S | NS | NS[13] | 0.032[14] | NS[15] |
| Lowe et al (1996) III | A vs C | U | 0.047 | NS[13] | NS[14] | 0.001[15] |
| Lowe et al (1996) III | A vs C | S | NS | NS[13] | NS[14] | NS[15] |
| Lyberg et al (1995a) | A vs C | U | 0.001 | NS[4] | | NS[15] |
| Lyberg et al (1995a) | A vs C | U | | 0.001[16] | | |
| Maltais et al (1991) | A vs C | U | 0.05 | | | |
| Mayer et al (1995) | A vs C | U | NS | | | |
| Mayer et al (1996) | A vs S | U | 0.01 | | | |
| Mochizuki et al (1996) | A vs S | U | 0.01 | | | |
| Pracharktam et al (1994) | A vs S | U | 0.001 | | 0.05[15] | |
| Pracharktam et al (1996) | A vs S | U | 0.000 | 0.01[17] | | |
| Sakakibara et al (1999) | A1 vs C | U | 0.001 | | | NS[19] |
| Sakakibara et al (1999) | A2 vs C | U | 0.001 | | | 0.001[19] |
| Sakakibara et al (1999) | A1vs A2 | U | NS | | | 0.001[19] |
| Strelzow et al (1988) | A vs C | U | | 0.05[6] | 0.05[18] | |
| Strelzow et al (1988) | A vs C | U | | 0.05[4] | NS[5] | |
| Strelzow et al (1988) | A vs C | U | | 0.05[8] | | |
| Tangugsorn et al (1995a) | A vs C | U | 0.001 | 0.001[4] | | NS[20] |
| Tangugsorn et al (1995a) | A vs C | U | | 0.001[16] | | 0.001[21] |
| Tsuchiya et al (1992) | A3 vs C | U | NS | NS[13] | NS[15] | NS[22] |
| Tsuchiya et al (1992) | A4 vs C | U | 0.01 | 0.01[13] | NS[15] | 0.01[22] |
| Tsuchiya et al (1992) | A3 vs A4 | U | 0.05 | 0.01[13] | 0.05[15] | NS[22] |
| Zucconi et al (1992) | A vs C | U | 0.001 | | | |
| **Sherring (2001) RDI > 10** | **A vs S** | **U** | **0.05** | **0.000[4]** | **NS[10]** | **0.000[22]** |
| **Sherring (2001) RDI > 10** | **A vs S** | **U** | | **0.02[6]** | | **0.000[23]** |
| **Sherring (2001) RDI > 15** | **A vs S** | **U** | **0.032** | **0.008[4]** | **NS[10]** | **0.006[22]** |
| **Sherring (2001) RDI > 15** | **A vs S** | **U** | | **NS[6]** | | **0.007[23]** |
| **Sherring (2001) RDI > 20** | **A vs S** | **U** | **NS** | **NS[4]** | **NS[10]** | **NS[22]** |
| **Sherring (2001) RDI > 20** | **A vs S** | **U** | | **NS[6]** | | **NS[23]** |

A = obstructive sleep apnoea subject; C = non-snoring, non-apnoeic subject; S = snoring, non-apnoeic subject; A1 = non-obese obstructive sleep apnoea subject; A2 = Obese obstructive sleep apnoea subject; High apnoea index/Low BMI; A4 Low apnoea index/High BMI.

1 H/ANS-PNS; 2 H – B; 3 ANS-H; 4 S – H; 5 Gn-H; 6 Ar-H; 7 H-PhW; 8 Go-H; 9 H-AA; 10 H–Me; 11 H-PhW (Me-H); 12 PNS-H; 13 H-H1; 14 H-RGn; 15 C3 – H; 16 H (FH; 17 H-Ver; 18 H-PhW (Go-H); 19 H-VL; 20 AH-C3 Hor; 21 AH-C3 Ver; 22 C3-H; 23 C4-H.

**Table 12.4-6: Hyoid Bone Angular Measurements**

| | | | Go-Gn-H | N-S/Ar-H | N-S-H | C3-RGn-H |
|---|---|---|---|---|---|---|
| deBerry-Borowiecki et al (1988) | A vs C | U | 0.000 | 0.017 | 0.042 | |
| Hochban et al (1994) | A vs C | U | 0.005 | 0.0001 | 0.0001 | |
| Pracharktam et al (1994) | A vs S | U | | | | 0.001 |
| Strelzow et al (1998) | A vs C | U | 0.05 | 0.05 | NS | |
| **Sherring (2001) RDI > 10** | **A vs S** | **U** | **NS** | **NS** | **NS** | **0.05** |
| **Sherring (2001) RDI > 15** | **A vs S** | **U** | **0.037** | **NS** | **NS** | **0.029** |
| **Sherring (2001) RDI > 20** | **A vs S** | **U** | **NS** | **NS** | **NS** | **NS** |

A = obstructive sleep apnoea subject; C = non-snoring, non-apnoeic subject; S = snoring, non-apnoeic subject.

## 12.4.6 Maxilla

The dimensions and the location of the maxilla may be important in the development of UAW obstruction at the nasopharyngeal level. The superior soft palate is attached to the posterior edge of the bony maxilla, and is therefore influenced by the anteroposterior position of the maxilla. A maxilla that is located in a protrusive position relative to the cranial base would be expected to have greater distance between its posterior border and the posterior nasopharyngeal wall. Conversely, a retrognathic maxilla would be expected to lie closer to the posterior nasopharyngeal wall. The tendency of the soft palate to cause nasopharyngeal airway obstruction, whilst possibly being influenced by the dimensions of the soft palate, might also be influenced by the anteroposterior position of the maxilla. The inclination of the maxillary plane can alter its effective length. A maxilla that is increased in anteroposterior length may not be measured cephalometrically as prognathic (relative to the cranial base as measured by angle S-N-A), nor might it cause narrowing of the nasopharynx simply because of rotation of the palatal plane relative to a horizontal reference plane.

The length of the maxilla may effect the tendency of the UAW to obstruct at the level of the nasopharynx. Posterior extension of the maxilla into the nasopharynx, decreasing the distance between the posterior nasopharyngeal wall and the maxilla might predispose to UAW obstruction at this level. The absolute length of the bony maxilla can be measured from the posterior nasal spine to the anterior nasal spine or to point A, the point of maximum concavity on the anterior maxilla below the anterior nasal spine. This study measured the distance ANS-PNS and found a significantly increased maxillary length in OSA subjects. This length was significantly increased in subjects diagnosed with OSA with an RDI > 10.

This finding is not reported elsewhere in the literature considering maxillary length in OSA subjects. Andersson and Brattstrom (1991); deBerry-Borowiecki et al (1988); Lowe et al (1996); Sakakibara et al (1999); Strelzow et al (1988); Tangugsorn et al (1995a), and Tsuchiya et al (1992) all found OSA subjects had a decreased anteroposterior length of the maxilla. In contrast Johns et al (1998); Lowe et al (1995); Lyberg et al (1989a), and Zucconi et al (1993) found no difference in maxillary length. These results are shown in Table 12.4-7.

The angulation of the palatal plane (ANS-PNS) to the cranial base (S-N) was not significantly different between OSA subjects or snorers irrespective of the RDI used to separate subjects in the study population (Table 12.4-8). This finding is supported by Bacon et al (1989), and Lowe et al (1996). Hochban and Brandenburg (1994) reported a dorsocaudal rotation of the palatal plane in OSA subjects, effectively decreasing the anteroposterior length of the maxilla. Despite increased maxillary length in the OSA subjects reported in our group separated by an RDI > 10 there is no rotation of the palatal plane to diminish the relative anteroposterior length of the maxilla. Thus in our group increased anteroposterior length of the maxilla may contribute to nasopharyngeal airway obstruction.

The dimensions of the bony skeleton should be stable following the completion of growth. Growth of the maxillofacial skeleton, as measured on longitudinal cephalometric radiographs, has been reported to continue into the fifth decade (Lewis and Roche, 1988). Comparison of maxillary dimensions with known cephalometric norms for a Caucasian population was performed in addition to comparison based upon RDI.

Anteroposterior position of the maxilla was measured in this study using the angle between the cranial base (S-N) and point A on the anterior maxilla. This angle will give an indication of the anteroposterior position of the maxilla but is influenced by the angulation of the cranial base. The steeper the cranial base the more acute this angle will tend to be. There was no significant difference in this angle between subjects diagnosed with OSA or as simple snorers irrespective of the diagnostic criteria. This is in agreement with the majority of the literature reviewed (Bacon et al, 1989; Johns et al, 1998; Lowe et al, 1995; Lyberg et al, 1989a; Maltais et al, 1991; Mayer and Meier-Ewert, 1995; Mochizuki et al, 1996; Ono et al, 1996; Pracharktam et al, 1996; Sakakibara et al, 1999; Tangugsorn et al, 1995a; Zucconi et al, 1992; and Zucconi et al, 1993). These results are shown in Table 12.4-8. There was however a tendency for this angle to become less acute as BMI increased. This would suggest that those subjects who snore or have OSA and have a low BMI are more likely to have a retrognathic maxilla with respect to the cranial base compared to those subjects with an elevated BMI. This suggestion is supported by Tsuchiya et al (1992) who reported a significantly retrognathic maxilla in a subgroup of OSA subjects with a high AHI/low BMI. Maxillary

retrognathia may be more important in OSA subjects with a low BMI than in those with an elevated BMI.

**Table 12.4-7: Maxilla Linear Measurements**

| | | | ANS-PNS | Dc - A | PNS – A |
|---|---|---|---|---|---|
| Andersson et al (1991) | A vs C | U | 0.01 | | |
| Bacon et al (1989) | A vs C | U | | 0.01 | |
| deBerry-Borowiecki et al (1988) | A vs C | U | 0.001 | | |
| Johns et al (1998) | A vs S | U | NS | | |
| Lowe et al (1995) | A vs C | U | NS | | |
| Lowe et al (1996) I | A vs C | U | 0.002 | | |
| Lowe et al (1996) I | A vs C | S | NS | | |
| Lowe et al (1996) II | A vs C | U | 0.031 | | |
| Lowe et al (1996) II | A vs C | S | NS | | |
| Lowe et al (1996) III | A vs C | U | NS | | |
| Lowe et al (1996) III | A vs C | S | NS | | |
| Lyberg et al (1995a) | A vs C | U | NS | | |
| Mochizuki et al (1996) | A vs S | U | | | NS |
| Pracharktam et al (1994) | A vs S | U | | | 0.05 |
| Pracharktam et al (1996) | A vs S | U | | | NS |
| Sakakibara et al (1999) | A1 vs C | U | 0.001 | | |
| Sakakibara et al (1999) | A2 vs C | U | NS | | |
| Sakakibara et al (1999) | A1vs A2 | U | NS | | |
| Strelzow et al (1988) | A vs C | U | 0.05 | | |
| Tangugsorn et al (1995a) | A vs C | U | 0.01 | | |
| Tsuchiya et al (1992) | A3 vs C | U | 0.05 | | |
| Tsuchiya et al (1992) | A4 vs C | U | NS | | |
| Tsuchiya et al (1992) | A3 vs A4 | U | NS | | |
| Zucconi et al (1992) | A vs C | U | NS | | NS |
| Zucconi et al (1992) | A vs S | U | NS | | 0.05 |
| **Sherring (2001) RDI > 10** | **A vs S** | **U** | **0.03** | | |
| **Sherring (2001) RDI > 15** | **A vs S** | **U** | **NS** | | |
| **Sherring (2001) RDI > 20** | **A vs S** | **U** | **NS** | | |

A = obstructive sleep apnoea subject; C = non-snoring, non-apnocic subject; S = snoring, non-apnoeic subject; A1 = non-obese obstructive sleep apnoea subject; A2 = Obese obstructive sleep apnoea subject; A3 High apnoea index / Low BMI; A4 Low apnoea index / High BMI.

**Table 12.4-8: Maxilla Angular Measurements**

| | | | SNA | A – P | SN/ANS-PNS | Inclination |
|---|---|---|---|---|---|---|
| Bacon et al (1989) | A vs C | U | NS | | NS | |
| deBerry-Borowiecki et al (1988) | A vs C | U | 0.018 | | | |
| Hochban et al (1994) | A vs C | U | 0.05 | NS[1] | 0.02 | NS[2] |
| Johns et al (1998) | A vs S | U | NS | | | |
| Lowe et al (1995) | A vs C | U | NS | | | |
| Lowe et al (1996) I | A vs C | U | | | NS | |
| Lowe et al (1996) I | A vs C | S | | | NS | |
| Lowe et al (1996) II | A vs C | U | | | NS | |
| Lowe et al (1996) II | A vs C | S | | | NS | |
| Lowe et al (1996) III | A vs C | U | | | NS | |
| Lowe et al (1996) III | A vs C | S | | | NS | |
| Lyberg et al (1995a) | A vs C | U | NS | | | |
| Maltais et al (1991) | A vs C | U | NS | | | |
| Mayer et al (1995) | A vs C | U | NS | | | |
| Mochizuki et al (1996) | A vs S | U | NS | | | |
| Ono et al (1996) | A vs C | U | NS | | | |
| Pracharktam et al (1994) | A vs S | U | | 0.05[3] | | 0.01[4] |
| Pracharktam et al (1996) | A vs S | U | NS | | | NS[4] |
| Sakakibara et al (1999) | A1 vs C | U | NS | | | |
| Sakakibara et al (1999) | A2 vs C | U | NS | | | |
| Sakakibara et al (1999) | A1vs A2 | U | NS | | | |
| Strelzow et al (1988) | A vs C | U | 0.05 | | | |
| Tangugsorn et al (1995a) | A vs C | U | NS | | | |
| Tsuchiya et al (1992) | A3 vs C | U | 0.05 | | | |
| Tsuchiya et al (1992) | A4 vs C | U | NS | | | |
| Tsuchiya et al (1992) | A3 vs A4 | U | NS | | | |
| Zucconi et al (1992) | A vs C | U | NS | | | |
| Zucconi et al (1993) | A vs S | U | NS | | | |
| **Sherring (2001) RDI > 10** | **A vs S** | **U** | **NS** | | **NS** | |
| **Sherring (2001) RDI > 15** | **A vs S** | **U** | **NS** | | **NS** | |
| **Sherring (2001) RDI > 20** | **A vs S** | **U** | **NS** | | **NS** | |

A = obstructive sleep apnoea subject; C = non-snoring, non-apnoeic subject; S = snoring, non-apnoeic subject; A1 = non-obese obstructive sleep apnoea subject; A2 = Obese obstructive sleep apnoea subject; A3 High apnoea index / Low BMI; A4 Low apnoea index / High BMI.

1 FH/NA; 2 ANS-PNS/FH; 3 ANS-PNS/PM; 4 Ar-N/PM

## 12.4.7 Mandible

The relationship of the mandible and tongue may play a role in oropharyngeal and hypopharyngeal airway obstruction just as the interplay between the maxilla and soft palate may contribute to nasopharyngeal airway obstruction. The tongue attaches in the midline anteriorly to the genial tubercles on the lingual side of the mandibular symphysis. Greco et

al (1989); Ching (1995) and Hochban et al (1996) have reported that mandibular setback decreased the posterior airway space as measured between the dorsal surface of the tongue and the posterior pharyngeal wall. This space is increased by mandibular advancement (Hochban et al, 1994; Isono, 1995) and is the mechanism by which mandibular repositioning appliances and orthognathic surgery are thought to treat upper airway obstruction (Kuo et al, 1979; Riley et al, 1989; Riley et al, 1993; Riley et al, 1994; Clark et al, 1996 and Thornton and Roberts, 1996). Thus, there appears to be a relationship between mandibular position and the UAW space between the dorsum of the tongue and the posterior pharyngeal wall.

Changes in the length of the mandible, particularly retrognathia, might be expected to contribute to UAW obstruction due to a retropositioned tongue. The current study did not find any significant difference in mandibular length measured from the angle of the mandible to the symphysis (Gn-Go). The findings of this study with respect to mandibular length are in agreement with deBerry-Borowiecki et al (1988); Lowe et al (1995) and Tsuchiya et al (1992). These results are shown in Table 12.4-9.

A mandible that is deficient in the anteroposterior dimension, but that is not retrognathic when measured by the angle formed between the cranial base and point B (S-N-B), might not be expected to contribute to UAW obstruction. A mandible fitting this criteria will not adversely affect tongue position because the genial tubercles are anteriorly positioned. In this situation there is likely to be an increased in angle S-N-B and an anti-clockwise rotation of the mandible. A mandible of normal length that has an increased mandibular plane angle relative to the cranial base or Frankfort Horizontal may however contribute to UAW obstruction. In this situation the genial tubercles may be positioned further posterior, thus allowing the tongue to occupy a more posterior position in the oropharynx or hypopharynx.

The current study did not find a significant difference in the angle formed between the cranial base and supramentale (S-N-B). This is in agreement with Battagel and L'Estrange (1996); deBerry-Borowiecki et al (1988); Johns et al (1998); Lyberg et al (1989a); Maltais et al (1991); Mayer and Meier-Ewert (1995); Mochizuki et al (1996); Pracharktam et al (1996); Strelzow et al (1988); Zucconi et al (1992) and Zucconi et al (1993). These results are shown in Table 12.4-10. There was however a relationship between this angle and BMI. The angle tended to be more acute in patients with a low BMI compared to those subjects with an elevated BMI. This indicates that those subjects in this study who snore, or who have OSA and a low BMI, are more likely to have a retrognathic mandible than those patients exhibiting the same symptoms of UAW obstruction and an elevated BMI. This supports the literature that suggests there is a subgroup of OSA subjects who primarily have a skeletal discrepancy as the cause of their sleep-disordered breathing as opposed to excessive soft tissue.

The angulation of the mandibular plane may modify the impact mandibular length has on the aetiology of UAW obstruction. Similar to the influence palatal plane angle has on the relationship of maxillary length to the nasopharyngeal airway width, so too angulation of the mandibular plane can exaggerate or reduce the impact of mandibular length on posterior airway space. Measurement of the mandibular plane angle in this study was done in reference to the cranial base. There was no significant difference in this angle between OSA subjects and snoring subjects irrespective of the RDI used to define OSA (Table 12.4-10). This finding is supported by deBerry-Borowiecki et al (1988); Hochban and Brandenburg (1994) and Ono et al (1996).

**Table 12.4-9: Mandibular Linear Measurements**

| | | | Total Length | AP Length | Vertical - Anterior | Ramus Height | Proportion |
|---|---|---|---|---|---|---|---|
| Andersson et al (1991) | A vs C | U | 0.01[1] | 0.001[2] | | | |
| Andersson et al (1991) | A vs S | U | | | 0.05[3] | | |
| Battagel et al (1996) | A vs C | U | | 0.002[4] | | | |
| Battagel et al (1996) | A vs C | U | | 0.002[5] | | | |
| deBerry-Borowiecki et al (1988) | A vs C | U | | NS[2] | | 0.020[6] | |
| deBerry-Borowiecki et al (1988) | A vs C | U | | NS[5] | | | |
| Johns et al (1998) | A vs S | U | NS[1] | | | | |
| Lowe et al (1995) | A vs C | U | | NS[2] | | NS | |
| Lowe et al (1996) I | A vs C | U | | 0.004[2] | | | |
| Lowe et al (1996) I | A vs C | S | | NS[2] | | | |
| Lowe et al (1996) II | A vs C | U | | NS[2] | | | |
| Lowe et al (1996) II | A vs C | S | | NS[2] | | | |
| Lowe et al (1996) III | A vs C | U | | NS[2] | | | |
| Lowe et al (1996) III | A vs C | S | | NS[2] | | | |
| Lyberg et al (1995a) | A vs C | U | NS[7] | NS[8] | | | |
| Pracharktam et al (1994) | A vs C | U | | | | | 0.01[9] |
| Pracharktam et al (1996) | A vs C | U | | | | | NS[9] |
| Sakakibara et al (1999) | A1 vs C | U | | NS[4] | | NS[6] | |
| Sakakibara et al (1999) | A2 vs C | U | | NS[4] | | NS[6] | |
| Sakakibara et al (1999) | A1 vs A2 | U | | NS[4] | | NS[6] | |
| Sakakibara et al (1999) | A1 vs C | U | | 0.001[10] | | | |
| Sakakibara et al (1999) | A2 vs C | U | | NS[10] | | | |
| Sakakibara et al (1999) | A1 vs A2 | U | | 0.001[10] | | | |
| Strelzow et al (1988) | A vs C | U | | 0.05[2] | | 0.05[6] | |
| Strelzow et al (1988) | A vs C | U | | 0.05[5] | | | |
| Tangugsorn et al (1995a) | A vs C | U | NS[7] | NS[8] | | | |
| Tangugsorn et al (1995a) | A vs C | U | | 0.05[11] | | | |
| Tsuchiya et al (1992) | A3 vs C | U | | NS[2] | | | |
| Tsuchiya et al (1992) | A4 vs C | U | | NS[2] | | | |
| Tsuchiya et al (1992) | A3 vs A4 | U | | NS[2] | | | |
| Zucconi et al (1992) | A vs C | U | | 0.05[2] | | | |
| Zucconi et al (1992) | A vs C | U | | 0.05[4] | | | |
| Zucconi et al (1993) | A vs S | U | | 0.01[2] | | | |
| Zucconi et al (1992) | A vs C | U | | 0.05[4] | | | |
| **Sherring (2001) RDI > 10** | **A vs S** | **U** | | **NS[4]** | | | |
| **Sherring (2001) RDI > 15** | **A vs S** | **U** | | **NS[4]** | | | |
| **Sherring (2001) RDI > 20** | **A vs S** | **U** | | **NS[4]** | | | |

A = obstructive sleep apnoea subject; C = non-snoring, non-apnoeic subject; S = snoring, non-apnoeic subject; A1 = non-obese obstructive sleep apnoea subject; A2 = Obese obstructive sleep apnoea subject; A3 High apnoea index / Low BMI; A4 Low apnoea index / High BMI.

1 Cd-Gn; 2Go-Gn; 3 Id-Gn; 4 Go-Me; 5 Go-B; 6 Ar-Go; 7 Ar-Pgn; 8 Go-Pgn; 9 Ram/MCF; 10 G-VL; 11 Pg

**Table 12.4-10: Mandibular Angular Measurements**

| | | | Mandibular Plane Angle | S-N-B | S-N-Pg | Gonial Angle |
|---|---|---|---|---|---|---|
| Andersson et al (1991) | A vs C | U | 0.05[1] | | | |
| Bacon W et al (1989) | A vs C | U | 0.05[1] | | | |
| Battagel et al (1996) | A vs C | U | | NS | | NS[2] |
| deBerry-Borowiecki et al (1988) | A vs C | U | NS[1] | NS | | NS[3] |
| deBerry-Borowiecki et al (1988) | A vs C | U | NS[4] | | | |
| Hochban et al (1994) | A vs C | U | NS[1] | 0.02 | | NS[2] |
| Hochban et al (1994) | A vs C | U | NS[4] | | | |
| Johns et al (1998) | A vs S | U | 0.01[4] | NS | | |
| Lowe et al (1995) | A vs C | U | 0.02[1] | 0.01 | | |
| Lowe et al (1996) I | A vs C | U | 0.033[1] | 0.003 | | |
| Lowe et al (1996) I | A vs C | S | NS[1] | 0.006 | | |
| Lowe et al (1996) II | A vs C | U | NS[1] | NS | | |
| Lowe et al (1996) II | A vs C | S | NS[1] | NS | | |
| Lowe et al (1996) III | A vs C | U | NS[1] | NS | | |
| Lowe et al (1996) III | A vs C | S | NS[1] | NS | | |
| Lyberg et al (1995a) | A vs C | U | | NS | NS | NS[5] |
| Maltais et al (1991) | A vs C | U | NS[6] | NS | | |
| Mayer et al (1995) | A vs C | U | | NS | | |
| Mochizuki et al (1996) | A vs S | U | | NS | | |
| Ono et al (1996) | A vs C | U | NS[1] | | | |
| Pracharktam et al (1996) | A vs C | U | | NS | | |
| Sakakibara et al (1999) | A1 vs C | U | | | NS | |
| Sakakibara et al (1999) | A2 vs C | U | | | NS | |
| Sakakibara et al (1999) | A1 vs A2 | U | | | NS | |
| Strelzow et al (1988) | A vs C | U | 0.05[1] | NS | | NS[3] |
| Strelzow et al (1988) | A vs C | U | NS[4] | | | |
| Tangugsorn et al (1995a) | A vs C | U | 0.001[1] | 0.01 | 0.01 | 0.01[5] |
| Tsuchiya et al (1992) | A3 vs C | U | 0.01[1] | 0.01 | | NS[7] |
| Tsuchiya et al (1992) | A4 vs C | U | NS[1] | 0.05 | | NS[7] |
| Tsuchiya et al (1992) | A3 vs A4 | U | 0.05[1] | 0.05 | | NS[7] |
| Zucconi et al (1992) | A vs C | U | | NS | | |
| Zucconi et al (1993) | A vs S | U | | NS | | NS[3] |
| **Sherring (2001) RDI > 10** | **A vs S** | **U** | **NS[8]** | **NS** | | |
| **Sherring (2001) RDI > 15** | **A vs S** | **U** | **NS[8]** | **NS** | | |
| **Sherring (2001) RDI > 20** | **A vs S** | **U** | **NS[8]** | **NS** | | |

A = obstructive sleep apnoea subject; C = non-snoring, non-apnoeic subject; S = snoring, non-apnoeic subject; A1 = non-obese obstructive sleep apnoea subject; A2 = Obese obstructive sleep apnoea subject; A3 High apnoea index / Low BMI; A4 Low apnoea index / High BMI.

1 SN/Go-Gn; 2 Ar-Go-Me; 3 Ar-Go-Gn; 4 FH/Go-Gn; 5 RL/ML; 6 SN/Go-B; 7 Not defined; 8 SN/Go-Me

### 12.4.8 Maxilla and Mandible Inter-Relationship

The relationship in the anteroposterior direction between the maxilla and mandible may be important in the prediction of site of obstruction from lateral cephalometric radiographs. Most authors have concentrated on measuring this difference using the angle formed between subspinale (point A), nasion (N) and supramentale (point B). This allows the relationship of the maxilla and mandible to be considered independent of the cranial base. Using the angles S-N-A or S-N-B to determine the AP position of the maxilla and mandible will allow the angle of the cranial base to influence our measurement of their relationship.

The angle A-N-B will allow classification of the skeletal relationship of the maxilla and mandible into three broad categories. These categories are analogous to the dental classification as proposed by Angle (1899) which related to the inter-relationship of the maxillary and mandibular third molar teeth. A "normal" relationship of the maxilla and mandible sees a positive angle A-N-B with a range of normal $0^0$ to $5^0$, usually corresponding to a dental class I relationship. A retrognathic maxilla relative to the mandible would see the angle A-N-B measure less than $0^0$, corresponding to an Angle class III dental relationship. A retrognathic mandible relative to the mandible would have the angle A-N-B measure greater than $5^0$, corresponding to a dental class II relationship.

One of the problems with relying on angle A-N-B to determine the intermaxillary relationship is the influence cranial base length has upon this angle. A long cranial base will reduce angle A-N-B whilst a short cranial base will increase angle A-N-B (although the relation of the maxilla to the mandible is unchanged). Thus, there may be some misclassification of subjects due to the influence of cranial base length. Likewise clockwise rotation of the jaws produces an increase in angle A-N-B, thus possibly overstating the number of subjects with a class II skeletal pattern. Dolichofacial (long face) subjects can have a larger anteroposterior discrepancy of the maxilla and mandible than is suggested by angle A-N-B, again possibly resulting in the misclassification of subjects.

If obstruction of the UAW is to occur due to malposition of the jaws, it follows that retrognathia of one or both jaws is more likely to contribute to this obstruction than prognathism. Interestingly this study found a greater proportion of subjects with an RDI > 20 and maxillary and/or mandibular prognathia than retrognathia. Two subjects had bimaxillary retrusion greater than three standard deviations below the mean and an RDI > 20. One of these subjects had a BMI > 30 $kg/m^2$, with the other in the range 25 < BMI < 30. Seven subjects had bimaxillary protrusion greater than three standard deviations above the mean and an RDI > 20. All seven of these subjects had a BMI > 30 $kg/m^2$. This finding suggests that

obesity may have a greater influence on the severity of obstructive sleep apnoea than jaw position.

The interrelationship of the maxilla and mandible in sleep apnoeic patients cannot be considered in isolation because the angle A-N-B may fall within the range $0^0 < \text{A-N-B} < 5^0$, yet both jaws may be prognathic or retrognathic. Considering only those subjects with a class I skeletal relationship, and angle SNA and angle SNB greater than three standard deviations from the mean, one subject had bimaxillary retrusion and ten had bimaxillary protrusion. In these subjects, the malposition of the jaws may not be obvious clinically, however radiographic measurement would allow these anomalies to become clearer.

One subject with a skeletal class II relationship had bimaxillary retrognathia, with both maxilla and mandible greater than three standard deviations below the population mean. This subject was had an RDI in the range $15 < \text{RDI} < 20$. Bimaxillary protrusion occurring in the presence of a class II skeletal relationship occurred in two subjects. One of these subjects had an RDI in the range $10 < \text{RDI} < 15$ ($25 < \text{BMI} < 30$ kg/m$^2$) and the other an RDI $> 20$ (BMI $> 30$ kg/m$^2$). On clinical assessment subjects with a skeletal class II relationship are generally presumed to have a retrognathic mandible, and may be treated accordingly with surgical or non-surgical means to advance the tongue and/or mandible. Two subjects with a skeletal class II relationship had a greatly prognathic maxilla and mandible, yet still suffered obstructive sleep apnoea. One of these subjects was also obese, which may have played a greater role in the severity of obstruction, however the second subject was borderline overweight (BMI 25.4 kg/m$^2$). This subjects jaw position may play a greater role in the aetiology of obstructive sleep apnoea than BMI. This study did not look at treatment of these patients, however future investigation is needed to determine whether further advancement of an already prognathic mandible is necessary, or effective. Another aspect to be considered in future studies is whether treatment of patients with skeletal discrepancy should be influenced by BMI. Are surgical and non-surgical treatments to advance a retrognathic mandible as effective in subjects with elevated BMI when compared to subjects with the same jaw discrepancy but no increase in BMI?

Two subjects with a skeletal class III relationship had bimaxillary retrognathia greater than three standard deviations below the population mean. Both these subjects had an RDI $> 20$. Three subjects had bimaxillary protrusion in the presence of a class III skeletal relationship. One of these subjects was a snorer (RDI $< 10$) with the other two having an RDI in the range $15 < \text{RDI} < 20$.

The numbers of subjects in all groups with significantly prognathic or retrognathic jaws are too small to allow statistical comparison. Future studies with larger numbers of subjects are

needed to clarify whether these findings have any significance as predictors of OSA incidence or severity.

The literature suggests that if the maxilla or mandible, or both are retrognathic then a person is more likely to suffer OSA. The numbers in this study are small, however there is a tendency for subjects with bimaxillary retrusion to have UAW obstruction. Three of the four subjects with bimaxillary retrognathia greater than three standard deviations from the mean had an RDI > 20, irrespective of BMI. This suggests that extreme retrognathia may be a risk factor for severe OSA irrespective of BMI.

Forty-one subjects had bimaxillary protrusion with the maxilla and mandible at least one standard deviation above the Caucasian norm. Twelve of these subjects were snorers (RDI < 10), nine had mild OSA (10 < RDI < 20) and 20 were diagnosed with moderate to severe OSA (RDI > 20). Twenty-eight subjects had bimaxillary protrusion greater than two standard deviations above the population mean, with seven snorers (RDI < 10), nine mild OSA (10 < RDI < 20) and twelve moderate or severe OSA (RDI > 20). Subjects with protrusive maxilla and mandible at least three standard deviations from normal numbered 14. Two of them were snorers (RDI < 10), 12 had OSA (RDI > 10), with nine of the OSA subjects having an RDI > 20). At least 86% of these subjects were overweight with a BMI > 25 kg/m$^2$, and 57% were obese with a BMI > 30 kg/m$^2$.

These findings may also influence treatment. A finding of bimaxillary retrusion and obstructive sleep apnoea (RDI > 20) may suggest orthognathic surgery to correct the jaw malposition should be given a higher priority. Conversely, bimaxillary protrusion may mean advancement of the jaws, surgically or with a mandibular repositioning appliance, is not as likely to help the patient. These hypotheses require further studies to determine their clinical validity.

This study measured the angle A-N-B and found no difference between subjects irrespective of the RDI used to define OSA. This finding is consistent with Andersson and Brattstrom (1991); Battagel and L'Estrange (1996); Bacon et al (1989); deBerry-Borowiecki et al (1988); Hochban and Brandenburg (1994); Lyberg et al (1989a); Maltais et al (1991); Ono et al (1996); Sakakibara et al (1999); Tangugsorn et al (1995a) and Tsuchiya et al (1992). These results are shown in Table 12.4-11. These findings indicate there was no tendency for obstructive sleep apnoea to occur in patients with a particular maxillo-mandibular relationship (skeletal class) compared to others with-in the study group.

There was a greater proportion of subjects with a discrepancy in maxillary or mandibular position as measured by angle A-N-B when compared to the normal population. An epidemiological study of Australian Aborigines and Caucasian subjects reported 87.0% of

Caucasians with a Class I malocclusion, 11.1% with a Class II malocclusion and 1.9% with a class III malocclusion (Clinch, 1951). In this study thirty-nine subjects (41.5%) of subjects were classified as skeletal class I ($0^0$ < A-N-B < $5^0$); thirteen subjects (13.8%) of subjects were classified as skeletal class II (A-N-B > $5^0$) and forty-two subjects (44.7%) were classified as skeletal class III (A-N-B < $0^0$).

There is a significantly greater number of people with a Class III malocclusion compared with the Australian population. This skeletal relationship may be produced by a combination of a retrognathic maxilla (S-N-A < $80^0$) and a normally positioned ($78^0$ < S-N-B < $82^0$) or prognathic mandible (S-N-B > $82^0$). Alternatively the maxilla may be normally positioned ($80^0$ < S-N-A < $84^0$) and the mandible prognathic (S-N-B > $82^0$). The increase in incidence of this skeletal subtype may be due to alteration of the ethnic mix of the Australian population since 1951, with post war migration to Australia of European and more recently South-east Asian people. There may be an anatomic reason why people with this pattern of relationship between the maxilla and the mandible have tendency to snore or suffer OSA. Lowe et al (1996) reported subjects with a class III skeletal relationship had an increased tongue height and a more inferiorly and anteriorly placed hyoid than the control subjects. Tongue height was not measured in this study. We did find the hyoid located more anteriorly and inferiorly in OSA subjects, but did not analyze our data according to skeletal class due to insufficient numbers in all groups to make valid statistical comparison.

Four subjects with a class III malocclusion (A-N-B < $0^0$) had a retrognathic maxilla (S-N-A < $76^0$) which is greater than three standard deviations below the population mean. One of these subjects had an RDI < 10 and are simple snorers, with the remaining three having an RDI > 20. The numbers are small however this would support the hypothesis that a retrognathic maxilla may be associated with an increased incidence and severity of obstructive events during sleep. Thirteen subjects with a class III malocclusion (A-N-B < $0^0$) had a prognathic mandible (S-N-B > $86^0$), which is greater than three standard deviations above the population mean (Steiner, 1959). Three of these subjects had an RDI < 10 and are simple snorers; four were in the range 10 < RDI < 20 whilst the remaining six had an RDI > 20. The numbers are too small to do a statistical analysis, however a markedly prognathic mandible appears to be related to an increased severity of obstructive events during sleep.

The findings from this study suggest that bimaxillary retrusion is likely to be related to OSA in the nonobese subject whilst the subject exhibiting bimaxillary protrusion and OSA is likely to be obese. This suggests the non-obese subject is likely to have primarily a skeletal malposition and no major abnormality of the soft tissue dimensions. The obese subject is likely to have obstruction of their upper airway due to increased tissue bulk associated with

obesity. This proposal was first raised by Tsuchiya et al, (1992) and is supported by the findings of this study.

The effect of mandibular or maxillary retrognathia on the severity of OSA was striking. In all patients who exhibited a retrognathic maxilla or mandible (as measured by angle SNA and SNB respectively), whether one, two or three standard deviations from the mean and who had OSA, the OSA was moderate ($15 < RDI < 20$) or severe ($RDI > 20$). Not all patients with mandibular or maxillary retrognathia had an $RDI > 10$ and although subjects with retrognathia tended to have a higher RDI this cephalometric criteria could not be used as a diagnostic tool. Clinicians should have a higher index of suspicion if a patient who is being investigated for possible OSA does have retrognathia. More subjects exhibited prognathism of the maxilla or the mandible however there was not such a strong correlation with the incidence of UAW obstruction during sleep. When subjects with a normal or slightly elevated BMI were considered very few subjects who exhibited signs of OSA by measurement of RDI also had markedly malpositioned maxilla or mandible. The small number of patients affected suggests there is either little relationship between the position of the jaws and the incidence of OSA or this method of determining the anteroposterior position of the jaws lacks sensitivity.

Obesity was a more important contributor to severity of OSA in our population for subjects with a Class I or a Class III skeletal pattern of malocclusion, as measured by angle ANB. Subects with a Class II skeletal pattern of malocclusion were found to have a greater contribution from the skeletal position than from obesity, as measured by BMI.

Mandibular advancement alone for the management of obstructive sleep apnoea has not been reported often in the surgical literature. Most authors who undertake surgery to correct skeletal anomalies perform bimaxillary procedures, where both the mandible and the maxilla are advanced. This is somewhat surprising given approximately 16% of the Caucasian population are estimated to have at least a 6mm overjet (Proffit, 1986). Thus a significant proportion of the general population has a retrognathic and or short mandible anteroposteriorly and this has been suggested as a cause of oropharyngeal and hypopharyngeal airway narrowing (Lowe et al, 1996; Andersson and Brattstrom, 1991; Battagel and L'Estrange, 1996; Lowe et al, 1996; Sakakibara et al, 1999; Strelzow et al, 1988; Tangugsorn et al, 1995a; Zucconi et al, 1992; and Zucconi et al, 1993).

One of the goals of this thesis is to characterize the cephalometric characteristics of a population of snoring and obstructive sleep apnoeic patients. If there is a subgroup demonstrating obstructive sleep apnoea and a retrognathic mandible a question will arise as to whether mandibular advancement alone may be appropriate treatment. A second issue with mandibular advancement alone as opposed to bimaxillary advancement is the change in

occlusion that will occur from mandibular surgery alone. Orthognathic surgery is complicated in dentate individuals by the requirement to maintain (or possibly improve) occlusion. Mandibular advancement alone may be indicated from a diagnostic work-up but be impractical because of the requirement for complex (and often expensive) orthodontic intervention in an age group where tooth movement is neither routine nor simple.

**Table 12.4-11: Maxilla and Mandible Angular Relationship**

| | | | ANS-PNS/Go-Gn | SN/OP | A-N-B |
|---|---|---|---|---|---|
| Andersson et al (1991) | A vs C | U | 0.05 | | NS |
| Bacon et al (1989) | A vs C | U | | 0.05 | NS |
| Battagel et al (1996) | A vs C | U | | | NS |
| deBerry-Borowiecki et al (1988) | A vs C | U | NS | | NS |
| Hochban et al (1994) | A vs C | U | NS | | NS |
| Lowe et al (1995) | A vs C | U | | | 0.02 |
| Lowe et al (1996) I | A vs C | U | | 0.044 | 0.027 |
| Lowe et al (1996) I | A vs C | S | | NS | 0.011 |
| Lowe et al (1996) II | A vs C | U | | NS | NS |
| Lowe et al (1996) II | A vs C | S | | NS | NS |
| Lowe et al (1996) III | A vs C | U | | NS | NS |
| Lowe et al (1996) III | A vs C | S | | NS | NS |
| Lyberg et al (1995a) | A vs C | U | | NS | NS |
| Maltais et al (1991) | A vs C | U | | | NS |
| Ono et al (1996) | A vs C | U | | | NS |
| Sakakibara et al (1999) | A1 vs C | U | | | NS |
| Sakakibara et al (1999) | A2 vs C | U | | | NS |
| Sakakibara et al (1999) | A1 vs A2 | U | | | NS |
| Strelzow et al (1988) | A vs C | U | 0.05 | | |
| Tangugsorn et al (1995b) | A vs C | U | | | NS |
| Tsuchiya et al (1992) | A3 vs C | U | | | NS |
| Tsuchiya et al (1992) | A4 vs C | U | | | NS |
| Tsuchiya et al (1992) | A3 vs A4 | U | | | NS |
| **Sherring (2001) RDI > 10** | **A vs S** | **U** | | | **NS** |
| **Sherring (2001) RDI > 15** | **A vs S** | **U** | | | **NS** |
| **Sherring (2001) RDI > 20** | **A vs S** | **U** | | | **NS** |

A = obstructive sleep apnoea subject; C = non-snoring, non-apnoeic subject; S = snoring, non-apnoeic subject; A1 = non-obese obstructive sleep apnoea subject; A2 = Obese obstructive sleep apnoea subject; A3 High apnoea index / Low BMI; A4 Low apnoea index / High BMI.

## 12.4.9 Cranial measurements

The length of the cranial base (sella – nasion) was not significantly different between snoring or OSA subjects irrespective of the RDI used to define OSA. These findings are in

agreement with deBerry-Borowiecki et al (1988) and Strelzow et al (1998). Sakakibara et al (1999) reported the length of the cranial base to be significantly shorter in non-obese obstructive sleep apnoeic subjects compared with controls and with obese obstructive sleep apnoeic subjects. They reported no significant difference between obese obstructive sleep apnoeic subjects and controls with respect to this measurement. Zucconi et al (1993) reported no difference in cranial base length between obstructive sleep apnoeic subjects and controls, but a significantly shorter cranial base when compared with snoring subjects. Other authors also report a significantly shorter cranial base in subjects with obstructive sleep apnoea (Andersson et al, 1991; Bacon et al, 1989 and Battagel et al, 1996) and suggested this finding may indicate a more retruded facial skeletion.

Subjects with a Class II skeletal malocclusion did have a significantly shorter cranial base than subjects with a Class I skeletal malocclusion or a Class III skeletal malocclusion. These results are shown in Table 12.4-12. This suggests a more retruded face in subjects with a Class II skeletal malocclusion, as suggested by Bacon et al (1989). This group was the smallest of all subgroups and it may be that with a larger group a correlation with RDI is found.

**Table 12.4-12: Cranial Base Linear Measurements**

| | | | S-N | S-Ba | N-SC | Ar-S | N-Ba |
|---|---|---|---|---|---|---|---|
| Andersson et al (1991) | A vs C | U | 0.001 | 0.01 | | | |
| Bacon et al (1989) | A vs C | U | 0.001 | | | | |
| Battagel et al (1996) | A vs C | U | 0.01 | | | | |
| deBerry-Borowiecki et al (1988) | A vs C | U | NS | | NS | NS | |
| Sakakibara et al (1999) | A1 vs C | U | 0.001 | NS | | NS | NS |
| Sakakibara et al (1999) | A2 vs C | U | NS | NS | | NS | NS |
| Sakakibara et al (1999) | A1 vs A2 | U | 0.001 | NS | | NS | 0.001 |
| Strelzow et al (1988) | A vs C | U | NS | | | | |
| Tangugsorn et al (1995a) | A vs C | U | 0.001 | 0.001 | | | 0.001 |
| Zucconi et al (1993) | A vs C | U | NS | | | | 0.01 |
| Zucconi et al (1993) | A vs S | U | 0.05 | | | | 0.01 |
| **Sherring (2001) RDI >10** | **A vs S** | **U** | **NS** | | | | |
| **Sherring (2001) RDI >15** | **A vs S** | **U** | **NS** | | | | |
| **Sherring (2001) RDI >20** | **A vs S** | **U** | **NS** | | | | |
| **Sherring (2001)** | **Cl I vs Cl II** | **U** | **0.04** | | | | |
| **Sherring (2001)** | **Cl II vs Cl III** | **U** | **NS** | | | | |
| **Sherring (2001)** | **Cl II vs Cl III** | **U** | **0.03** | | | | |

A = obstructive sleep apnoea subject; C = non-snoring, non-apnoeic subject; S = snoring, non-apnoeic subject; A1 = non-obese obstructive sleep apnoea subject; A2 = Obese obstructive sleep apnoea subject; A3 High apnoea index / Low BMI; A4 Low apnoea index / High BMI.

This study found no significant difference in the divergence of the cranial base as measured by the angle between Frankfort horizontal and the sella – nasion line. This is in agreement with Mayer and Meier-Ewert (1995) and Strelzow et al (1988) and is shown in Table 12.4-13. The implication from this finding is that there is not an increase in upper facial height as measured by this parameter. This was reported by deBerry-Borowiecki et al (1988) and Lyberg et al (1995a). These groups suggested that the divergence of the cranial base indicated clockwise rotation of the facial skeleton and therefore may contribute to narrowing of the UAW. The increase in the angle of the sella – nasion line, as discussed previously may increase angle ANB, although the true anteroposterior discrepancy of the maxilla and mandible is less than that suggested by this angle.

Measurement of angle Ba-S-N showed no significant difference between any of the groups based upon RDI or skeletal classification. This is in agreement with Hochban et al (1994); Johns et al (1998); Lyberg et al (1995a); Pracharktam et al (1994); Pracharktam et al (1996); Sakakibara et al (1999); Tangugsorn et al (1995a) and Zucconi et al (1993). A few authors have reported contrary results (Andersson et al, 1991; Battagel et al, 1996 and Steinberg et al, 1995). The pattern of skeletal malocclusions seen in this study would seem not to be a result of abnormalities of cranial base angulation, and nor would cranial base angulation appear to have an influence on the incidence of obstructive sleep apnoea syndrome.

**Table 12.4-13: Cranial Base Angular Measurements**

| | | | Ba-S-N | SN/FH | N-S-Ar | MCF | FH/Hor | SN/Ver |
|---|---|---|---|---|---|---|---|---|
| Andersson et al (1991) | A vs C | U | 0.01 | | | | | |
| Battagel et al (1996) | A vs C | U | 0.01 | | | | | |
| deBerry-Borowiecki et al (1988) | A vs C | U | | 0.045 | NS | | | |
| Hochban et al (1994) | A vs C | U | NS | | | | | |
| Johns et al (1998) | A vs S | U | NS | | | | | |
| Lyberg et al (1995a) | A vs C | U | NS | 0.05 | | | | |
| Mayer et al (1995) | A vs C | U | | NS | | | | |
| Pracharktam et al (1994) | A vs S | U | NS | | | 0.01 | | |
| Pracharktam et al (1996) | A vs S | U | NS | | | NS | NS | |
| Sakakibara et al (1999) | A1 vs C | U | NS | | | | | NS |
| Sakakibara et al (1999) | A2 vs C | U | NS | | | | | NS |
| Sakakibara et al (1999) | A1vs A2 | U | NS | | | | | NS |
| Steinberg et al (1995) | A vs C | U | 0.0001 | | | | | |
| Strelzow et al (1988) | A vs C | U | | NS | | | | |
| Tangugsorn et al (1995a) | A vs C | U | NS | | | | | 0.01 |
| Zucconi et al (1993) | A vs C | U | NS | | | | | |
| Zucconi et al (1993) | A vs S | U | NS | | | | | |
| **Sherring (2001) RDI >10** | **A vs S** | **U** | **NS** | **NS** | | | | |
| **Sherring (2001) RDI >15** | **A vs S** | **U** | **NS** | **NS** | | | | |
| **Sherring (2001) RDI >20** | **A vs S** | **U** | **NS** | **NS** | | | | |
| **Sherring (2001)** | **Cl I vs Cl II** | **U** | **NS** | | | | | |
| **Sherring (2001)** | **Cl I vs Cl III** | **U** | **NS** | | | | | |
| **Sherring (2001)** | **Cl II vs Cl III** | **U** | **NS** | | | | | |

A = obstructive sleep apnoea subject; C = non-snoring, non-apnoeic subject; S = snoring, non-apnoeic subject; A1 = non-obese obstructive sleep apnoea subject; A2 = Obese obstructive sleep apnoea subject; A3 High apnoea index / Low BMI; A4 Low apnoea index / High BMI.

## 12.4.10 Dental Measurements

There was no significant difference in the angulation of the lower incisors relative to the mandibular plane. This was also reported by Lowe et al (1995). Upper incisor angulation relative to the cranial base (U1 – SN) was not significantly different if an AHI > 10 or an AHI > 15 was used to separate the subjects. However, using an AHI > 20 as the diagnostic criterion for obstructive sleep apnoea, upper incisor angulation was significantly increased in this group. Bacon et al (1989); Battagel and L'Estrange (1996); Ono et al (1996) and Tsuchiya et al (1992) reported no alteration in upper incisor angulation. Lowe et al (1996) reported the upper incisors were more upright in OSA subjects with a class I dental malocclusion. They also reported angle SNB to be significantly decreased in OSA subjects with a class I dental malocclusion compared to the control group. The upright maxillary

central incisors could be dental compensation for the discrepancy in anteroposterior lengths between the maxilla and mandible to maintain a normal overjet relationship.

The lower incisors were proclined in OSA subjects with a class I dental malocclusion according to Lowe et al (1996), although other authors are in agreement with the findings in this study (Battagel and L'Estrange, 1996). Tsuchiya et al (1992) reported an increase in lower incisor angulation only in subjects with a high apnoea index and a low BMI. This group of subjects was identified as having a higher incidence of malposition of the maxilla and mandible in the anteroposterior direction. The resultant lower incisor angulation may be compensation by the dental structures to an underlying skeletal discrepancy.

The current study did find a significant correlation between upper incisor angulation and BMI such that as BMI increased so too did upper incisor proclination. This could be due to increased pressure being placed upon the palatal surfaces of the upper incisors from the tongue, although in this situation lower incisor angulation would also be expected to increase. A more likely explanation is related to the discrepancy in anteroposterior position of the maxilla and mandible. The population had a large number of subjects who exhibited a class III skeletal relationship. The proclination of the upper incisors is likely to be dental compensation for a relatively short maxilla relative to the mandible. The net result of upper incisor proclination is to maintain an overjet that allows the teeth to incise food effectively. This proposal is supported as upper incisor proclination is greatest in subjects with a class III skeletal relationship ($107.6^0$), least in those with a class II skeletal relationship ($102.0^0$) and those with a class I skeletal relationship have their upper incisors proclined $106.6^0$.

**Table 12.4-14: Dentition Angular Measurements**

| | | | U1/L1 | U1/ANS-PNS | U1/S-N | U1/N-A | L1 to MP | L1/Go-Gn | L1/N-B |
|---|---|---|---|---|---|---|---|---|---|
| Bacon et al (1989) | A vs C | U | NS | | | | | | |
| Battagel et al (1996) | A vs C | U | | NS | | | | NS | |
| Lowe et al (1995) | A vs C | U | | | NS | | NS | NS | |
| Lowe et al (1996) I | A vs C | U | | | 0.037 | | | 0.002 | |
| Lowe et al (1996) I | A vs C | S | | | 0.035 | | | NS | |
| Lowe et al (1996) II | A vs C | U | | | NS | | | NS | |
| Lowe et al (1996) II | A vs C | S | | | NS | | | NS | |
| Lowe et al (1996) III | A vs C | U | | | NS | | | NS | |
| Lowe et al (1996) III | A vs C | S | | | NS | | | NS | |
| Ono et al (1996) | A vs C | U | | | NS | | | | |
| Tsuchiya et al (1992) | A3 vs C | U | | | | NS | | | 0.05 |
| Tsuchiya et al (1992) | A4 vs C | U | | | | NS | | | NS |
| Tsuchiya et al (1992) | A3 vs A4 | U | | | | NS | | | NS |
| **Sherring (2001) RDI > 10** | **A vs S** | **U** | | | **NS** | | **NS** | | |
| **Sherring (2001) RDI > 15** | **A vs S** | **U** | | | **NS** | | **NS** | | |
| **Sherring (2001) RDI > 20** | **A vs S** | **U** | | | **0.03** | | **NS** | | |

A = obstructive sleep apnoea subject; C = non-snoring, non-apnoeic subject; S = snoring, non-apnoeic subject; A1 = non-obese obstructive sleep apnoea subject; A2 = Obese obstructive sleep apnoea subject; A3 High apnoea index / Low BMI; A4 Low apnoea index / High BMI.

## 12.4.11 Conclusion

The lateral cephalometric linear and angular variables that were measured do show particular trends with respect to the position of important anatomic structures when related to the RDI. There is an increased soft palate thickness (UW1 – UW2) irrespective of the RDI used to separate subjects into snoring or obstructive sleep apnoeic groups. Similarly, the length of the upper airway measured from the posterior nasal spine to the vallecula was significantly increased in all subjects classified as obstructive sleep apnoeic irrespective of the RDI used to separate subjects. These results suggest that irrespective of the severity of obstructive sleep apnoea the thickness of the soft palate and the length of the upper airway may be used as indicators of the presence of obstructive sleep apnoea when looking at a lateral cephalometric radiograph.

Separation of the subjects at an AHI > 10 or AHI > 15 to diagnose obstructive sleep apnoeic subjects yielded similar results. Using these criteria the hyoid position tended to be more anteriorly and inferiorly placed. The significant measurements indicating an anteriorly placed hyoid in obstructive sleep apnoeic subjects in these groupings were the distances H –

$C_3$ and H – $C_4$. In the obstructive sleep apnoeic group separated at an AHI > 10 the distance Go – H was also increased. Interestingly when an AHI > 15 was used, the distance Go – H was not increased but the angular measure of the antero-posterior position of the hyoid was increased (<Go-Gn-H). This is a measurement of the height of the hyoid, and an increased angle indicates an inferiorly place hyoid bone. The hyoid was also inferiorly positioned as measured by the distances S – H and MP – H and angle H – $H_1$ in obstructive sleep apnoeic subjects separated at an AHI > 10 or and AHI > 15. In contrast when AHI > 20 was used as the criterion for diagnosis of obstructive sleep apnoea there was not a significant difference measured in hyoid position between the snoring group (AHI < 20) and the obstructive sleep apnoeic group. This suggests that hyoid position may be more important in mild obstructive sleep apnoeic subjects and become a less important discriminant of obstructive sleep apnoea as severity increases.

The length of the maxilla was significantly longer in the obstructive sleep apnoeic group only if the sample population was defined as obstructive sleep apnoeic by an AHI > 10. The maxilla was not found to be significantly longer in obstructive sleep apnoeic subjects if the threshold for defining obstructive sleep apnoea was increased to an AHI > 15 or and AHI > 20. Increased maxillary length, with no concomitant increase in mandibular length would be expected to result in either:

1. an increased incidence of a skeletal class II relationship in these subjects, or
2. a decreased posterior airway space behind the soft palate at the level of the nasopharynx, or
3. a rotation of the maxillary plane (ANS – PNS) relative to a fixed horizontal reference plane such as Frankfort Horizontal or the line Sella – Nasion (cranial base).

None of these measurements was significantly different between the groups. The obstructive sleep apnoeic group, on average, had an increased mandibular length of 1.71 +/- 0.99 mm, whilst the average increase in maxillary length was 1.75 +/- 0.54 mm compared to the snoring group. Thus there was an increase in anteroposterior length of the maxilla and mandible in obstructive sleep apnoeic subjects who had an AHI > 10 events per hour, which would account for the fact there was no significant increase in the incidence of a skeletal class II relationship.

The grouping where obstructive sleep apnoea was defined as an AHI > 20 had proclined upper incisors relative to the cranial base (U1 – SN). The significantly increased proclination of the upper central incisors in the obstructive sleep apnoeic group may indicate a compensatory mechanism by the teeth to obtain a positive overjet. Dental compensation arises when there is a discrepancy in the maxillary and mandibular lengths relative to each

other. Proclined upper incisors and/or retroclined lower incisors are usually found in subjects with a skeletal class III jaw relationship, representing a compensatory mechanism for a relatively short maxilla compared to the mandibular length. The teeth, under the influence of the tongue and lips will tend to adapt in order to obtain a satisfactory incisal relationship. Thus, the proclination of the upper incisors in this group of subjects either may be masking a relative lack of maxillary length compared to the mandibular length, or the maxilla may indeed be short. Alternatively the maxilla may be of normal length but the mandible is increased in its anteroposterior dimensions. Nineteen subjects with an RDI > 20 (46.34%) had a skeletal class III relationship, indicating a relatively short maxilla compared with the mandible. There were not a significantly increased number of subjects with obstructive sleep apnoea (RDI > 20) and a skeletal class III relationship. Overall 42/94 (44.7%) of subjects had a skeletal class III relationship and 41/94 (43.6%) of subjects had obstructive sleep apnoea with an RDI > 20. It must be remembered, however, that there was a much greater incidence of subjects in this study with a skeletal class III relationship than is found in the normal population.

Tongue length was significantly greater in those subjects classified as obstructive sleep apnoeic when an AHI > 15 or and AHI > 20 was used as the threshold for diagnosis. Tongue length measured from the tongue tip to the epiglottic tip and base was significantly increased when AHI > 15, and the length measured from the tongue tip to the epiglottic base was significantly increased when AHI > 20. These differences were not seen when subjects were separated at an AHI > 10 as being diagnostic for obstructive sleep apnoea. These findings suggest that if the length of the tongue, as measured from a lateral cephalometric radiograph, is increased then that person is more likely to have obstructive sleep apnoea.

The characterization of the appearance of an obstructive sleep apnoeic subject on a lateral cephalometric radiograph might be a person with an elongated upper airway and a thick soft palate. They are likely to have an increased tongue length and may have a short maxilla or proclined upper incisors. The position of the hyoid is probably anterior and inferior when compared to a snoring subject. From our study it is also more likely these subjects will have a skeletal class III relationship.

## 12.5 Cephalometry as an Imaging Modality for OSA

The advantages and limitations of lateral cephalometric radiographs have been reviewed in Chapter 3.7. A standardized protocol was followed for all patients having lateral cephalometric radiographs in this study, thus minimizing problems with systematic errors as defined by Brown et al (1970). Although the radiographs were not always exposed by an

experienced radiographer, the patient positioning and exposure of the radiograph, if performed by a student, were done so under the direction and supervision of one of two experienced dental radiographers to ensure the patients were correctly positioned.

The Adelaide Dental Hospital is the only teaching hospital for dental undergraduates in South Australia. During their final year of study all dental students are attached to the Dental Radiography Unit to gain experience in the technique of intra-oral and extra-oral radiography pertinent to the practice of dentistry. This is a supervised teaching period however several lateral cephalometric radiographs taken of study participants were incomplete or of a standard that made interpretation of some cephalometric landmarks difficult. The hyoid bone was not included at all on one radiograph and on three others was only partially visible. In each case the patient was unwilling to have a second radiograph taken. These patients were included in the study however where ambiguity or lack of definition of a cephalometric landmark occurred the measurements using that landmark were excluded.

The radiographs utilised for statistical purposes in this study were those taken with the dental prosthesis removed. This is the standard method of obtaining a lateral cephalometric radiograph of edentulous patients used in the Adelaide Dental Hospital by the Oral and Maxillofacial Surgery Unit. There is no literature comparing lateral cephalometric radiographs on OSA subjects with and without dental prostheses in position. Tallgren et al (1983) did find an increase in the distance from the hyoid to cervical spine in edentulous subjects. This may be an important factor if the patient sleeps without the dental prosthesis in position as Lowe et al (1996) reported a decreased hyoid to cervical spine distance in subjects with OSA. Obese patients with OSA have also been reported to have a decreased distance between the hyoid and cervical spine compared with patients without an increased BMI (Sakakibara et al, 1999 and Tsuchiya et al, 1992). Thus the loss of teeth may result in alteration of the muscular attachment of the extrinsic tongue muscles to the mandible resulting in anterior repositioning of the hyoid. There are no reports in the literature of differences in oropharyngeal or hypopharyngeal airway space between edentulous and dentate subjects. Further investigation could be made to determine if the loss of teeth does influence the position of the muscular attachments of the tongue or the posture of the mandible and what, if any, effect a dental prosthesis has on these dimensions. Subjects were not questioned as to whether they routinely slept with their dentures in position.

The adherence to correct patient positioning and exposure ensured that errors associated with projection should not be significant in this study, and is supported by Houston et al (1986); Midtgard et al (1974); and Solow (1966).

Errors of landmark identification were minimized by having only one investigator identify the landmarks, which standardized the errors with respect to landmark measurement and ensured no ambiguity about landmark identification. These have been cited as significant sources of errors in studies using lateral cephalometric radiographs (Baumrind and Frantz, 1971a; Broch et al, 1981; Brown et al, 1970; Chate, 1987; Houston, 1983; Midtgard et al, 1974; van der Linden, 1971; and Vincent and West, 1987). One investigator tracing, identifying and digitizing the cephalometric landmarks has also been reported to be more accurate than the use of multiple investigators as inter-observer variability is eliminated (Savage et al, 1987; Solow, 1966; and Stabrun and Danielsen, 1982). All cephalometric points were digitized and a double determination of 20% of the radiographs confirmed the error of measurement for each point by the single investigator. Double determination and digitizing points has been reported as the most accurate method of locating landmarks and minimizing measurement error (Baumrind and Frantz, 1971a and 1971b). Errors are associated with the use of lateral cephalometric radiographs, and their use requires an awareness by clinicians of the possible sources of error and methods to minimize these errors when interpreting the radiograph. The principal source of error associated with the measurement of landmarks is their identification (Houston et al, 1986 and Sandler, 1988). Oral and Maxillofacial Surgeons and Orthodontists are the most reliant upon lateral cephalometric radiographs of all clinicians. The interpretation of these radiographs and landmark identification is therefore likely to be most accurately performed by them.

This study has found a number of significant correlations between the severity of UAW obstruction and measurements from lateral cephalometric radiographs, as discussed in the previous section. Additionally significant differences have been found between cephalometric measures when comparing subjects with UAW obstruction diagnosed as snorers and those diagnosed with OSA. Whilst universal screening of all people being investigated for snoring by means of a lateral cephalometric radiograph cannot be justified on the basis of the findings of our study, we believe lateral cephalometric radiographic assessment does have a role to play. This position is also supported by other authors in the literature (Hochban et al, 1994; Powell and Riley, 1995; and Riley et al, 1993). Before any irreversible treatment is undertaken, either soft tissue surgery or maxillofacial surgery, a lateral cephalometric radiograph should be obtained. Given soft tissue position is influenced by the position of the underlying bony structures it would seem wise to correct any underlying skeletal abnormalities prior to modifying soft tissue position. This would allay some of the potential problems, particularly velopharyngeal incompetence that may occur if, for example, a UPPP is performed prior to bimaxillary advancement. This recommendation is also supported by the literature (Conradt et al, 1997).

A question raised by Nelson and Hans (1997) is how soon may people be identified as being at risk of developing obstructive sleep apnoea? Do the dimensions and orientation of the middle cranial fossa in children predict future apnoeic activity? A longitudinal study may answer this question, but to date has not been done.

Difficulties also arise when attempting to compare findings between studies. A myriad of parameters is measured from lateral cephalometric radiographs, yet no two authors standardize their studies to consider the identical landmarks, linear, angular and area measurements. This study has attempted to use cephalometric landmarks that have allowed comparison between a large number of studies so that meaningful comparison between our sample population and other reported populations may be possible. At times this has resulted in landmarks being identified in very close proximity to each other, such as in the upper airway. Clinically it would not be necessary to utilise all the landmarks in this study when analysing a radiograph of a patient.

Targeting treatment to the site of obstruction is essential for most surgical and non-surgical treatments for obstructive sleep apnoea. The obvious exceptions are tracheostomy, which bypasses the upper airway completely and non-surgical techniques such as nCPAP, weight loss and pharmacological treatment. There has been no single diagnostic procedure or investigation reported that would determine the site of upper airway obstruction. All reported evaluations of the upper airway rely on a combination of acquired information from history, clinical examination and imaging to determine the most likely site of obstruction for each individual patient. Lateral cephalometric radiographs do not, by themselves, dictate treatment for individual patients. They are the best method of assessing the anteroposterior relationship of the maxilla and mandible and can be relied upon to assess and plan surgical or non-surgical intervention involving alteration of jaw position. They are of limited value in assessing soft tissue area and the dimensions of the upper airway and further information on these sites must be obtained through other means. This may involve visual assessment and grading of the soft palate size, as suggested by Mallampati et al (1982) or nasendoscopy as reported by Crumley et al (1987); Skatvedt (1993) and Woodson and Wooten (1994). Other imaging modalities have also been suggested as being useful in assessing the upper airway. Somnofluoroscopy (Katsantonis and Walsh, 1986), CT (Kuna et al, 1988; Lowe et al, 1995; Schwab, Gefter, Hoffman et al, 1983; Schwab, Gefter, Pack et al, 1983; Shephard et al, 1990;) or MRI (Schwab et al, 1995). The advantages and limitations of each of these modalities have been discussed in Chapter 4. None has been shown conclusively to predict with more accuracy the site of upper airway obstruction when compared to lateral cephalometric radiographs. Nasendoscopy, CT and MRI have shown most promise, however these three investigations all deliver the information at a higher cost, either economically or in terms of

risk to the patient. In terms of planning surgery to the facial skeleton in the treatment of obstructive sleep apnoea, and reproducibility none would seem an adequate replacement for the lateral cephalometric radiograph. Thus it seems at this time there is a role for both lateral cephalometry and another imaging modality for the assessment of patient anatomy in those suspected of suffering obstructive sleep apnoea.

The value of screening all patients with a lateral cephalometric radiograph who are suspected of obstructive sleep apnoea has not been clearly addressed in the literature. In this study we found 55 subjects (58.51%) had a skeletal class II or class III relationship. From the work done by Clinch (1951) we should expect to find only 12 subjects (13%) in our sample with a skeletal class II or class III relationship. The implication of this finding is there may be a correlation between "abnormal" jaw position and obstructive sleep apnoea syndrome in our population. If so, the lateral cephalometric radiograph may be very useful as a rapid, inexpensive screening tool for detecting jaw malposition in patients being screened for obstructive sleep apnoea. The obvious question raised is will surgical correction of the maxilla and/or mandible in these subjects adequately treat their obstructive sleep apnoea? Further study must be done to answer this question with regards to our population.

## 12.6 Implications For Treatment of OSA

The non-surgical treatment of obstructive sleep apnoea syndrome has been reviewed in Chapter 8 and the surgical treatment in Chapter 9. Treatment may be directed at temporarily inhibiting obstruction, such as nCPAP or mandibular repositioning appliances, or at permanently bypassing the obstructed upper airway, such as by tracheostomy or correcting the cause of the obstruction, such as bimaxillary advancement or UPPP. Since the reported effectiveness of nCPAP in treating obstructive sleep apnoea syndrome by Sullivan et al (1981) all subsequent treatments have been measured against this "gold standard".

Lateral cephalometric radiographs may be useful, not only in the diagnostic work-up of patients with obstructive sleep apnoea, but also in monitoring the success of failure of treatment. Non-surgical treatment with a mandibular repositioning appliance, as reported by many authors including Clark et al (1993); Clark et al (1996); Eveloff et al (1994); Ferguson et al (1996); Kloss et al (1986); Meurice et al (1996); O'Sullivan et al (1995); Schmidt-Nowara et al (1991); and Thornton and Roberts (1996); and would profit from information gained from a lateral cephalometric radiograph. The direct effect on the hard and soft tissues adjacent to the upper airway and alterations in position and shape of the upper airway can all be determined in the lateral dimension.

Compliance is a problem with all forms of non-surgical intervention for the management of obstructive sleep apnoea (Clark et al, 1993; Foreyt and Goodrick, 1993; Kribbs et al, 1993; Nadazawa et al, 1992; Schmidt-Nowara et al, 1991; and Strollo et al, 1995). Patients who are successfully managed with a mandibular repositioning appliance (as determined by overnight polysomnographic sleep study) should be assessed by a lateral cephalometric radiograph with and without the appliance in-situ. Those patients that respond to a mandibular repositioning appliance may be offered surgery that mimics the movement obtained by the device. This allows accurate assessment and presurgical planning such that patient compliance is no longer an issue in the control of symptoms.

A trial of a mandibular repositioning appliance prior to surgery to correct a retrognathic mandible should also be considered. A lateral cephalometric radiograph is used to confirm the presence of mandibular retrognathia and if hypopharyngeal upper airway obstruction is suspected such an appliance reversibly advances the mandible in a similar manner to that of surgical advancement. Confirmation that such advancement has successfully managed the obstructive events then allows utilization of a lateral cephalometric radiograph in the surgical planning phase of treatment.

Lateral cephalometric radiographs have little role to play in the assessment of the nose prior to nasal surgery, although hypoplasia of the midface can certainly be detected. Increased nasal airway resistance has been reported by a number of authors in subjects with obstructive sleep apnoea (Blakley and Mahowald, 1987; Dayal and Phillipson, 1985; Hester et al, 1995; Olsen, 1991; and Miljeteig et al, 1992). Soft palate size has been implicated in the aetiology of snoring (Ikematsu, 1964) and obstructive sleep apnoea (Fujita et al, 1981; Katsantonis and Walsh, 1986; and Riley et al, 1985).

Information obtained from a lateral cephalometric radiograph may by helpful in determining those patients unlikely to benefit from corrective surgery of the structures surrounding the upper airway. The decision not to surgically operate on a patient based upon the lateral cephalometric radiograph findings alone would be flawed. This is so because, as this thesis contends, lateral cephalometric radiographs alone seem unable to determine the presence or absence of obstructive sleep apnoea, and more particularly the site of upper airway obstruction. This modality provides additional information for clinicians diagnosing and treating patients with signs and/or symptoms of obstructive sleep apnoea syndrome. A corollary to this idea is phasing treatment, as proposed by Powell and Riley (1993). Following diagnostic evaluation patients are offered surgery that seeks to correct the site(s) most likely to be the cause of upper airway obstruction. Following post-operative polysomnographic study further surgery, usually a bimaxillary advancement, is performed. They claim no significant difference in efficacy between this staged surgical approach and the

use of nCPAP. Thus orthognathic surgery is the final treatment offered in those patients who were initially not diagnosed with a skeletal malposition from a lateral cephalometric radiograph.

## 12.7 Public Health Issues Related to OSA

Obstructive sleep apnoea is a significant health issue affecting a relatively small percentage of the population (Lavie, 1983 and Young et al, 1993) but with potentially serious or fatal consequences if not treated (Benaim et al, 1992 and He et al, 1988). The incidence of undiagnosed obstructive sleep apnoea is extremely high (Young et al, 1997). Males are more commonly affected than females (Ancoli-Israel, 1987; Guilleminault and Dement, 1978; Kales et al, 1985 and Young et al, 1993). Obstructive sleep apnoea is reported to occur more commonly in older populations (Ancoli-Israel, 1989; Bixler et al, 1998; and Jennum and Wildschiodtz, 1987), however the significant morbidity and mortality occurs in younger people with the condition (Bixler et al, 1998). A disturbing number of people with moderate to severe obstructive sleep apnoea may not be aware they are suffering this condition (Young et al, 1997).

Daytime hypersomnolence associated with obstructive sleep apnoea syndrome is a commonly reported symptom of patients with obstructive sleep apnoea (Bixler et al, 1979; Klink and Quan, 1987; Lavie, 1983; and Moldofsky, 1992). Hypersomnolence has been shown to adversely impact on the ability to control a motor vehicle (Findley et al, 1986 and Findley et al, 1989). There is also a reported increased incidence of motor vehicle accidents and near misses in this population (Gonzalez-Rothi et al, 1988 and Jennum et al, 1993). Drivers of other vehicles and pedestrians are also placed at risk of death or injury due to involvement in a motor vehicle accident.

One of the unresolved difficulties when considering the cost of obstructive sleep apnoea to the community is the problem of determining exactly what adverse medical conditions are caused by this sleep disorder. As discussed in Chapter 2 a number of medical conditions have been reportedly associated with obstructive sleep apnoea, however there is some disagreement in the literature as to whether obstructive sleep apnoea is to blame. Obesity (Browman et al, 1984; Harman et al, 1982; Smith et al, 1985; and Young et al, 1983) and increased age are common features of most subjects diagnosed with this condition. Medical conditions associated with obstructive sleep apnoea are often similar to those experienced by elderly overweight patients (Davies et al, 1992). It is most likely that age, obesity and obstructive sleep apnoea syndrome contribute to the increased morbidity experienced by these patients (Bradley, 1992). A second consideration when considering weight loss is the fact some non-

obese patients present with obstructive sleep apnoea. This was first identified by Tsuchiya et al (1992). In this smaller group of patients there obviously is a mechanism other than obesity causing airway obstruction.

Overnight polysomnographic sleep studies are costly in terms of manpower, both to conduct the study and also to interpret the results. There are a limited number of beds available for these studies to be undertaken and a large number of the population with obstructive sleep apnoea are not effectively screened (Young et al, 1997). Education of general medical and dental practitioners regarding the symptoms of obstructive sleep apnoea and the potential increased morbidity and mortality would seem wise. Screening tools may be helpful to these practitioners in determining which patients should undergo such studies. These screening tools include questionnaires, history, physical examination and, as this study shows, lateral cephalometric radiography. Unfortunately, as with most medical conditions, no one screening tool is 100% specific or 100% sensitive for detecting obstructive sleep apnoea. In combination with an inquisitive mind the screening tools mentioned may allow early, judicious referral of obstructive sleep apnoeic patients to a sleep physician for diagnosis, thus potentially saving the community and patient much in terms of future health care.

# Chapter 13

# Conclusions

1. The results of this study showed that there are a number of significant differences in the craniomorphologic structure of obstructive sleep apnoeic subjects when compared to snoring subjects using lateral cephalometric radiographs.

2. Increasing age, obesity, neck circumference and being male were all correlated with an increasing incidence of obstructive sleep apnoea.

3. Soft palate thickness and pharyngeal length are significantly increased in obstructive sleep apnoeic subjects and snorers independent of the RDI used to define obstructive sleep apnoea.

4. Hyoid position on lateral cephalometric radiographs was significantly altered in mild obstructive sleep apnoeic subjects. In these subjects the hyoid was located inferior and anterior.

5. The hyoid was located inferiorly and posteriorly in obese obstructive sleep apnoeic subjects.

6. Tongue length was significantly increased in obstructive sleep apnoeic subjects with an RDI >15 but not in those with an RDI < 15.

7. Maxillary length was increased in mild obstructive sleep apnoeic subjects.

8. The relationship of the maxilla to the mandible was significantly different for all subjects compared to the normal population with the majority having a maxilla placed posteriorly compared to the mandible.

9. All subjects with a retrognathic maxilla as measured by angle SNA were obstructive sleep apnoeic.

10. All subjects with a retrognathic mandible as measured by SNB were obstructive sleep apnoeic.

11. Mandibular retrognathia was a more important predictor of obstructive sleep apnoea than elevated BMI.

12. Maxillary and/or mandibular retrognathia and obesity results in a greater severity of OSA.

13. Bimaxillary retrusion was associated with obstructive sleep apnoea in the non-obese subject.

14. Bimaxillary protrusion was associated with obstructive sleep apnoea in the obese subject.

15. Obesity was an uncommon finding in obstructive sleep apnoeic subjects with a significant malposition of the maxilla and/or mandible.

# Chapter 14

# Glossary

**ADH (Lowe A et al, 1995)**

Anterior dental height: Maxillary incisor length.

**AH (Lyberg et al, 1989b;Tangugsorn et al, 1995b)**

The most anterior and superior point on the body of the hyoid bone, representing the inferior part of the tongue.

**AH-S Ver (Tangugsorn et al, 1995a)**

The vertical distance measured perpendicular to Frankfort horizontal plane (FH) from the hyoid bone (AH) to sella (S). (Recorded under S – H)

**ANS-PNS/PM (Pracharktam et al, 1994)**

The angle between the palatal plane and posterior wall of the maxilla.

**Ar (deBerry-Borowiecki et al, 1988; Strelzow et al, 1988)**

Articulare. The intersection of a line along the posterior border of the mandible and the inferior border of the basilar occipital bone.

**Ara (Pracharktam et al, 1994)**

Anterior Ramus plane: a line from the intersection of the functional occlusal plane (FOP) and the anterior border of the ramus drawn parallel to PRa up to the reference line.

**Ar – A/ Ar – B (Pracharktam et al, 1996)**

Relative comparison of cumulative maxillary length with cumulative mandibular length measured along the reference line proposed by Enlow.

**Ar – H (deBerry-Borowiecki et al, 1988)**

Distance between articulare and the hyoid.

**Ar – N/PM (Pracharktam et al, 1994)**

The angle between the posterior wall of the maxilla relative to the constructed cranial base (Ar – N).

**Ar-Pgn (Lyberg et al, 1989a)**

The length of the mandible.

**Area Intermaxillary Space (Battagel and L'Estrange)**

The space defined by a line drawn through the maxillary and mandibular planes, the posterior pharyngeal wall and the lingual aspect of the lower incisor.

**Area Oropharynx (Lowe et al, 1996)**

Area outlined by an extension of the line ANS-PNS to pharyngeal wall, posterior surface of soft palate, line parallel to palatal plane from point P to dorsal surface of tongue, posterior inferior surface of tongue, line parallel to palatal plane through point Et, and posterior pharyngeal wall.

**Area Hypopharynx (Lowe et al, 1996)**

Area outlined by inferior border of oropharynx, posterior surface of epiglottis, line parallel to palatal plane through point C4, and posterior pharyngeal wall.

**ATA (deBerry-Borowiecki et al, 1988; Strelzow et al, 1988)**

Anterior tubercle of atlas.

**B (Battagel and L'Estrange)**

Point B. The point of maximum concavity of the mandibular alveolus.

**Ba (Bacon et al, 1989; Lyberg et al, 1989a; Tangugsorn et al, 1995b)**

Basion: The most posterior inferior point on the clivus.

**Ba – PNS (Bacon et al, 1989)**

The AP dimension of the bony pharynx.

**C2 (Battagel and L'Estrange, 1996)**

Most posterior superior point on the second cervical vertebra.

**C3 (Tsuchiya et al, 1992)**

The inferior anterior position on the third cervical vertebrae.

**C3 – H (Tsuchiya et al, 1992)**

The linear distance between C3 and H.

**Cataplexy**

A sudden loss of muscle tone, such as jaw drop, head drop, weakness of the knees or paralysis of all skeletal muscles with collapse.

**Cd (Andersson and Brattstrom , 1991;)**

Condylion

**Cd-Gn (Andersson and Brattstrom , 1991)**

Distance between the most posterior superior point on the mandibular condyle and the most anterior inferior point on the symphysis of the mandible.

**CL (Lyberg et al, 1989)**

Contact length between the dorsal contour of the tongue and the soft palate.

**CVT (Ozbek et al, 1998)**

Cervical Vertebrae Tangent: Tangent to the posterior superior point on the body of the second cervical vertebra and the inferior posterior point on the body of the fourth cervical vertebra.

**Dc (Bacon et al, 1989)**

Centre of the condylar neck on Ba-N line.

**Dc – A (Bacon et al, 1989)**

Sagittal dimension of the upper face from Dc to A.

**EA (deBerry-Borowiecki et al, 1988)**

Apex of the epiglottis.

**Eb (Pae et al, 1994;Pracharktam et al, 1994; Lowe et al, 1996)**

Base of epiglottis: The deepest point of the epiglottis.

**Et (Pae et al, 1994; Lowe et al, 1996)**

Tip of epiglottis: The most superior point of the epiglottis.

**FOP (Pracharktam et al, 1994)**

Functional occlusal plane: A line from the most posterior occlusal contact point to the last fully erupted maxillary and mandibular molars to the most anterior maxillary-mandibular first premolar occlusal contact.

**G (deBerry-Borowiecki et al, 1988)**

Genial tubercle: the most posterior point on the symphysis of the mandible.

**G (Sakakibara et al.,1999)**

Most posterior point on the symphysis of the mandible. (Retrognathion)

**G – VL (Sakakibara et al, 1999)**

Linear distance along the line from G to VL.

**GE (Lyberg et al, 1989b; Tangugsorn et al, 1995b)**

Genial Tubercle: Representing the most posterior point of the mandibular symphysis and the antero-inferior part of the tongue.

**Go (Andersson and Brattstrom , 1991; Battagel and L'Estrange, 1996)**

Gonion. The point where the bisector of the angle between the posterior and lower mandibular border tangents meets the mandibular angle.

**Go-B (Battagel and L'Estrange, 1996)**

The length of the mandible in the horizontal plane.

**Go-Gn (Andersson and Brattstrom , 1991)**

The length of the mandible.

**Go – H (deBerry-Borowiecki et al, 1988)**

The distance between gonion and the most anterior superior point on the body of the hyoid.

**Go-Me (Battagel and L'Estrange, 1996)**

Mandibular body length.

**Go-Pgn (Lyberg et al, 1989a)**

The length of the horizontal part of the mandible (corpus).

**Gn (Andersson and Brattstrom , 1991)**

Gnathion.

**H (deBerry-Borowiecki et al, 1988; Andersson and Brattstrom, 1991; Pae et al, 1994; Lowe et al, 1996)**

Hyoid bone. The most anterosuperior point on the body of the hyoid bone.

**H (Lyberg et al, 1989b; Tangugsorn et al, 1995b)**

The most superior point of the tongue in relation to the line from V to T.

**HH1 (Lowe et al, 1996)**

Linear distance between H and perpendicular to C3 to retrognathion.

**H-Ph (Andersson and Brattstrom , 1991)**

Perpendicular distance between the point H and line Ph. (Recorded under H-MP)

**H-Ver (Pracharktam et al, 1996)**

Vertical position of the hyoid relative to a line which is perpendicular to pterygomaxillary vertical line (PM) and passes sphenoethmoidal junction (SE).

**H-VL (Sakakibara et al, 1999)**

The linear distance along the line from H to VL.

**Hypnagogic hallucination**

False sesory perception occurring while falling asleep.

**Hypnopompic hallucination**

False perception occurring while awakening from sleep.

**IAS (Lowe et al, 1996)**

Width of the airway between the posterior surface of the tongue and the pharyngeal wall along the line Go-B.

**Id (Andersson and Brattstrom, 1991)**

Infradentale. The point of intersection of the lower central incisor tooth and the mandibular alveolus.

**Id-Gn (Andersson and Brattstrom, 1991)**

The anterior height of the bony mandible.

**Inclination Spinal (Battagel and L'Estrange, 1996)**

The angle between a line drawn between the sixth and second cervical vertebrae and a vertical through sella.

**In Mx Area (Pracharktam et al, 1994)**

Intermaxillary Area: Calculated by the average of anterior and posterior intermaxillary space height multiplied by intermaxillary length.

**L1 to MP (Lowe et al, 1995)**

Lower incisor length.

**Length Intermaxillary Space (Battagel and L'Estrange, 1996)**

The distance between the posterior pharyngeal wall and the lingual aspect lower incisor at the level of the occlusal plane.

**Length Intermaxillary Space (Pracharktam et al, 1994)**

Length of the intermaxillary space: Linear distance measured along the FOP from the point where it intersects Tt anteriorly to where it intersects PhW posteriorly.

**LFH (Bacon et al, 1989)**

The distance between ANS and Me on a line parallel to the occlusal plane.

**LFH/MFH (Pracharktam et al, 1994)**

Ratio of lower anterior facial height (a line from the functional occlusal plane to the mandibular plane which is parallel to the PM) to middle anterior facial height (a line from the functional occlusal plane to the palatal plane).

**LPW (Tangugsorn et al, 1995b)**

Lower pharyngeal wall: Intersection of a perpendicular line from V with the posterior pharyngeal wall.

**MAS (Johns et al, 1998)**

Distance between midpoint of soft palate and posterior pharyngeal wall on a line parallel to Frankfort horizontal.

**MAS (Lowe et al, 1996)**

Distance from the tip of the soft palate to the pharyngeal wall along a line parallel to Go-B. (Recorded under UT-PhW)

**MCF (Pracharktam et al, 1994)**

Middle Cranial Fossa: Represented by a line from Bjork's articulare (Ar) to SE.

**MCF Cranial Base (Pracharktam et al, 1994)**

Middle cranial fossa and posterior maxillary relative alignment (MCF/PM). A neutral MCF/PM is an internal angle of 40.3°. When a subject's PM is forward the neutral PM, a clockwise (+) rotation results. This forward (+) alignment of the MCF results in a mandibular retrusive effect. When a subject's PM is behind the neutral PM, a counterclockwise (-) rotation results. The backward (-) alignment of the MF results in a mandibular protrusive effect. If the neutral MCF plane coincides with the individual's own MCF, a neutral effect (0) is present.

**MdMH (Lowe et al, 1995)**

Mandibular molar height.

**Me (Battagel and L'Estrange, 1996)**

Menton. The point of intersection of the lower mandibular body and the symphyseal outline.

**ML (Lyberg et al, 1989a)**

Mandibular Line: The tangent to the lower border of the mandible through Gn.

**MxH (Lowe et al, 1995)**

Maxillary molar height.

**N (Andersson and Brattstrom, 1991)**

Nasion

**Nasion Perpendicular (McNamara, 1984)**

The dropping line from nasion (n) perpendicular to Frankfort horizontal plane (FH)

**N-Gn (Andersson and Brattstrom , 1991)**

Facial height. The distance between nasion and gnathion.

**NP (Lyberg et al, 1989a; Tangugsorn et al, 1995)**

**NREM**

Non-rapid eye movement.

**OA (Tangugsorn et al, 1995b)**

Oral Area: Included tongue area (TA) and extended superiorly to the outline of the soft and hard palate.

**Ocl – Pal 6 (Johns et al, 1998)**

Occlusal to palatal plane perpendicular at maxillary first molar (palatal vault height).

**OP (Bacon et al, 1989)**

The occlusal plane represented by a line passing through the middle of first molars and first premolars intercuspation.

**OPT (Ozbek et al, 1998)**

Odontoid Process Tangent: Tangent passing through the superior posterior point of second cervical vertebra and the inferior posterior point of second cervical vertebra.

**Oral Cavity Area (Sakakibara et al, 1999)**

The area of the oral cavity defined between PNS, A, Me, Go and PNS.

**Oral Cavity Space (Strelzow et al, 1988)**

The area of the oral cavity defined between PNS, ANS, Gn, Go and PNS.

**Oropharynx (Lowe et al, 1996)**

Area outlined by inferior border of nasopharynx, posterior surface of soft palate, line parallel to palatal plane from point P to dorsal surface of tongue, posterior inferior surface of tongue, line parallel to palatal plane through point Et, and posterior pharyngeal wall.

**OSA**

Obstructive sleep apnoea.

**P (Pae et al, 1994; Lowe et al, 1996)**

Palate point: The most inferior tip of the soft palate.

**PAS (ML) (Hochban and Brandenburg, 1994)**

Posterior airway space measured between the tongue base and posterior pharyngeal wall along an extension of the line Me-Go.

**PAS (OP) (Hochban and Brandenburg, 1994)**

Posterior airway space measured from the tongue base to the pharyngeal wall on the occlusal plane.

**PAS (NL) (Hochban and Brandenburg, 1994)**

Posterior airway space measured from PNS to the pharyngeal wall along an extension of the nasal line (ANS-PNS).

**Pg (Lyberg et al, 1989a; Tangugsorn et al, 1995)**

Pogonion: The most anterior point on the mandibular symphysis.

**Pg - NP (Lyberg et al, 1989a; Tangugsorn et al, 1995)**

Horizontal distance between pogonion and nasion perpendicular.

**Pgn (Lyberg et al, 1989a)**

Prognathion: the point on the mandibular symphysis farthest from Ar.

**Ph (Andersson and Brattstrom, 1991)**

The perpendicular from h to the mandibular plane (line Go-Gn).

**Ph-AS (deBerry-Borowiecki et al, 1988)**

Total area of the oropharynx and hypopharynx measured between the line ATA-PNS and a line through the hyoid bone (H) parallel to Frankfurt horizontal plane.

**PhT (deBerry-Borowiecki et al, 1988)**

Apex of the pharyngeal tubercle.

**PhW**

Pharyngeal wall.

**PhW1-PNS (Hochban and Brandenburg, 1994)**

Posterior airway space measured from PNS to the pharyngeal wall along the line Ba-PNS bisecting the posterior pharyngeal wall.

**Phw1-Psp (Pracharktam et al, 1994)**

Distance between the most posterior and superior point on the soft palate and the pharyngeal wall along a line parallel to line B-Go. (Recorded under UP-PhW).

**pm (Lyberg et al, 1989a; Tangugsorn et al, 1995b)**

Pterygomaxillare: The intersection between the nasal floor and the posterior contour of the maxilla.

**PM (Andersson and Brattstrom, 1991)**

Posterior maxilla. Corresponds to posterior nasal spine.

**PM (Pracharktam et al, 1994)**

Vertical PM: A line from SE through the averaged inferior-most points of the left and right pterygomaxillary fissure.

**PNS (deBerry-Borowiecki et al, 1988; Strelzow et al, 1988)**

Posterior nasal spine: Tip of the spine of the palatine bone of the hard palate.

**PNS (Riolo et al, 1974)**

The most posterior point at the sagittal plane on the bony hard palate.

**Post. In. Mx. Ht. (Pracharktam et al, 1994)**

Posterior intermaxillary space height: length of a perpendicular from the maxillary plane to the mandibular plane that passes through the point where the FOP intersects the PhW.

**PRa (Pracharktam et al, 1994)**

Posterior Ramus: A line from Ar to the intersection of the posterior border of the ramus with FOP.

**Proportion Tongue (Battagel and L'Estrange, 1996)**

The tongue area as a percentage of the intermaxillary space area.

**Prl (Battagel and L'Estrange, 1996)**

Width of oropharyngeal airway along the line that connects the most posterior point on the contour of the tongue and the pharyngeal wall.

**Prp1 (Battagel and L'Estrange, 1996)**

Width of nasopharyngeal airway along line connecting the most poster-superior point on the soft palate (determined by the eye) and the point on the pharyngeal wall at the same horizontal level.

**Prp2 (Battagel and L'Estrange, 1996)**

Width of the nasopharyngeal airway along the line of the lower incisor tip between the posterior aspect of the soft palate and the posterior pharyngeal wall.

**Psp (Lowe et al, 1996)**

Tip of uvula.

**R (Pae et al, 1994; Lowe et al, 1996)**

Roof of the pharynx: The point on the posterior pharyngeal wall constructed by a line PNS to the cross-sectional point of the cranial base and the lateral pterygoid plate.

**Ram/MCF (Pracharktam et al, 1994)**

Ramus width relative to middle cranial fossa horizontal dimension. The measure of ramus width is made at the level of REF by comparing to the horizontal dimension of the MCF along this line. The distances from Ar to Ara and Ar to neutral PM are measured along the reference line. A (+) effect occurs when the ramus width is less than Ar to PM neutral, and (-) effect when greater. When the measurements are the same, a neutral (0) effect results.

**REF (Pracharktam et al, 1994)**

Reference line: A line parallel to FOP from Ar anteriorly.

**REM**

Rapid eye movement.

**RGN (Pae et al, 1994; Lowe et al, 1996)**

Retrognathion: The most posterior point of the mandibular symphysis along a line perpendicular to the FH (Frankfort Horizontal) plane.

**RL (Lyberg et al, 1989a)**

Ramus Line: The tangent to the posterior border of the mandible

**RL/ML (Lyberg et al, 1989a)**

The gonial angle.

**SC (deBerry-Borowiecki et al, 1988)**

Sphenoidal crest.

**SE (Pracharktam et al, 1994)**

Sphenoethmoidal junction: The point at which the floor of the anterior cranial fossa intersects the averaged image left and right of the great wings of the sphenoid bone.

**S – H (deBerry-Borowiecki et al, 1988)**

Distance between sella and the most anterior superior point on the body of the hyoid.

**Sn (Andersson and Brattstrom, 1991)**

Subnasale. The most anterior point on the maxilla, corresponding with ANS.

**SN/Go-Gn (Andersson and Brattstrom, 1991)**

Angle between the mandibular plane and the cranial base.

**SN/OP (Bacon et al, 1989)**

The angulation of the occlusal plane with the SN line.

**SN-PM (Andersson and Brattstrom, 1991)**

Maxillary length. (Recorded under ANS-PNS)

**SPAS (Lowe et al, 1996)**

Narrowest distance between the posterior surface of the soft palate and the pharyngeal wall along a line parallel to Go-B. (Recorded under UP-PhW)

**SR (deBerry-Borowiecki et al, 1988; Strelzow et al, 1988)**

Sphenoidal rostrum: Superior extent to the pterygomaxillary fissure.

**T (Lyberg et al, 1989b; Tangugsorn et al, 1995b)**

The tip of the tongue.

**TB (deBerry-Borowiecki et al, 1988)**

Tongue base.

**TB-PhW (deBerry-Borowiecki et al, 1988)**

Linear distance from the tongue base to the posterior pharyngeal wall measured along a line parallel to Frankfurt horizontal.

**TA (Tangugsorn et al, 1995b)**

Tongue Area: The upper outline was defined by the dorsal contour of the tongue from V through H to T. The lower outline was reduced to a geometric polygon, of which the boundary was defined by line segments connecting the following points V, AH, GE and T.

**Tt (Pracharktam et al, 1994)**

Tongue tip: The most anterior point of the tongue that touches the lingual surface of the mandibular incisor.

**TT (deBerry-Borowiecki et al, 1988)**

Apex of the tongue.

**TT (Pae et al, 1994; Lowe et al, 1996)**

Tongue tip: The centre of the lead disk attached to the border between the ventral and dorsal surfaces of the tongue tip.

**TT-EA (deBerry-Borowiecki et al, 1988)**

Distance from the apex of the tongue to the tip of the epiglottis.

**TT-TB (deBerry-Borowiecki et al, 1988)**

Distance from the apex of the tongue to the tongue base.

**TV (deBerry-Borowiecki et al, 1988)**

Total area of the tongue measured at its superior limits and the line contained between TT, G, H and a line parallel to FH up to EA.

**U (Lyberg et al, 1989b; Tangugsorn et al, 1995b)**

Tip of the uvula: the most posteroinferior point of the uvula.

**UP (deBerry-Borowiecki et al, 1988)**

Soft palate protrusion: greatest posterior convexity of soft palate.

**UPW (Tangugsorn et al, 1995b)**

Upper pharyngeal wall: Intersection of the pm – ba line and the posterior pharyngeal wall.

**UP-PhW (deBerry-Borowiecki et al, 1988)**

Linear distance between the greatest convexity of the soft palate and the pharyngeal wall.

**UT-PhW (Battagel and L'Estrange, 1996)**

The point on the posterior pharyngeal wall at the same horizontal level as the tip of the soft palate.

**UT-PhW (deBerry-Borowiecki et al, 1988)**

Linear distance from the tip of the soft palate to the pharyngeal wall measured along a line parallel to Frankfurt Horizontal.

**UT-PhW (Hochban and Brandenburg, 1994)**

Linear distance from the tip of the soft palate to the pharyngeal wall measured along a line parallel to the occlusal plane.

**V (Sakakibara et al, 1999)**

Vallecula: The most antero-inferior point of the epiglottic fold.

**V (Lyberg et al, 1989b; Tangugsorn et al, 1995b)**

Vallecula: The intersection of the epiglottis and the base of the tongue.

**VL (Sakakibara et al, 1999)**

A line across C3 and C4.

# Appendix

**Patient Information Sheet – Cephalometry for Obstructive Sleep Apnoea**

Thank you for participating in this study designed to enable a better understanding of the causes of obstructive sleep apnoea and suitability of proposed treatment for patients suffering this condition.

Sleep apnoea and the related conditions of snoring can have serious effects on your health. Part of the problem appears to arise from the shape of your facial bones or the shape of your tongue, lips and throat.

Both the bones and the soft tissues of your face can be very accurately measured by a particular type of radiograph, known as a lateral head cephalogram. This type of x-ray is taken very commonly in young children to determine how their face and jaws are growing. Once the x-rays have been taken, quite simple but detailed methods of measurement are applied so that one can precisely determine the site, if any, of the problem that your are having with breathing.

The purpose of this particular study is to determine the characteristic face shapes of people with obstructive sleep apnoea. Overseas studies in the United States of America have shown that people from different communities have different face shapes associated with their sleep apnoea.

The shape of your face has impacted on your treatment. Thus, for example, if the x-ray shows that your lower jaw is small, there are some non-surgical and surgical treatments which can be done to improve the way that you breathe. Also, if your have a large tongue or a small throat, or a large palate, these will be shown on the x-ray and will help in planning of your treatment.

The x-ray exposure used is very small and represents less than 1% of the recommended annual exposure.

Thus this study may be of direct benefit to you in your treatment, as well as being of benefit to future patients being treated at the Royal Adelaide Hospital with this problem.

This study is being undertaken by Prof AN Goss DDS FRACDS (OMS) FICD; Dr DJ Sherring BDS and Dr R Antic MB BS FRACP.

If you wish to discuss aspects of the study you may also contact the Chairman, Research Ethics Committee, Royal Adelaide Hospital.

**THE UNIVERSITY OF ADELAIDE**

**CONSENT FORM**

**See also Information Sheet on reverse.**

1. I _____ (please print) hereby consent to take part in the research project entitled:

   "Cephalometry for Sleep Apnoea".

2. I acknowledge that I have read the information sheet entitled:

   "Cephalometry for Sleep Apnoea".

3. I have had the research project, so far as it affects me, fully explained to my satisfaction by the research worker. My consent is freely given.

4. Although I understand that the purpose of this research project is to improve the quality of medical care, it has been explained to me that my involvement may not be of any benefit to me.

5. I have been given the opportunity to have a member of my family or a friend present while the project was explained to me.

6. I have been informed that, while information gained during the study may be published, I will not be identified and my personal results will not be divulged.

7. I understand that I am free to withdraw from the project at any time and that this will bot affect medical advice in the management of my health, now or in the future.

8. I am aware that I should retain a copy of this Consent Form, when completed, and the relevant Information Sheet.

**SIGNED**_____ **DATE**_____

**NAME OF WITNESS**_____ **DATE**_____

I, _____ have described to _____

the nature of the procedures to be carried out. In my opinion he/she understood the explanation.

**SIGNED**_____ **DATE**_____

**STATUS IN PROJECT**_____

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