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THE PREVALENCE OF SNORING AND SLEEP APNOEA SYMPTOMS REPORTED BY ADULT PATIENTS

IN JOHANNESBURG URBAN GENERAL PRACTICE

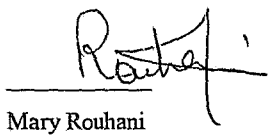
Mary M. Rouhani-N

A research report submitted to the Faculty of Health Sciences, University of the Witwatersrand, in fulfillment of the requirements for the degree of Master of Family Medicine.

November 2000

Declaration

I, Mary Rouhani, declare that this research report is my own work. It is being submitted in partial fulfillment of the requirements for the degree of Master of Family Medicine at the University of the Witwatersrand, Faculty of Health Sciences, Johannesburg. It has not been submitted before for any degree or examination at any other university.



Mary Rouhani

15th day of November, 2000.

Dedication

To: Behzad Robert
Nadia Lua and
Juliet Ella

and to my parents Ata and Aqdas Rouhani

Abstract

The prevalence of Obstructive sleep apnoea (OSA) has never to date been researched in South Africa. OSA is characterized by recurrent pharyngeal airway collapse during sleep, snoring and daytime sleepiness. It is diagnosed in 4% of men and 2% of women between the ages of 30 and 60 in the USA, and is associated with increased morbidity and mortality. This cross sectional descriptive study of adult patients was undertaken to examine the prevalence of OSA symptoms reported by patients in urban general practice in Johannesburg. A total of 304 self administered questionnaires were obtained for analysis. Snoring prevalence was 48.6% with increasing prevalence over a five year period (prevalence rates increased from 43.9% five years ago to a current prevalence of 48.6%). The prevalence of witnessed apnoeas, restless sleep and gasping were noted to be 12.4%, 44.6% and 11.8% respectively. 68% of men and 32.5 % of women snored and prevalence rates were found to increase with increasing age. A significant association was also found between OSA symptoms and comorbidities, e.g., hypertension and depression.

Based on the findings of this study OSA appears to be a significant problem in South Africa. Routine screening of patients for OSA seems warranted, and doctors need to be made aware of these statistics.

Key words
Obstructive sleep apnoea
Mortality
Snoring Prevalence

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Chapter 1

Introduction

1.1 Obstructive sleep apnoea

Obstructive sleep apnoea (OSA) is a medical disorder associated with abnormal respiration during sleep (sleep disordered breathing) and the occurrence of episodes of complete (apnoea) or partial pharyngeal obstruction[1] (hypopnoea), resulting in multiple arousals, snoring, daytime somnolence and nocturnal oxygen desaturation[2,3]. The constellation of symptoms and signs is called Obstructive Sleep Apnoea (OSA). It is a sleep disorder with potentially fatal consequences that mostly affects men in mid-life[4,5].

Accumulated knowledge appears to associate OSA with a range of cardiovascular[6-10], and neurobehavioural sequelae[11-13]. The OSA patients appear to have twice as much hypertension, three times as much ischeamic heart disease, and four times as much cerebrovascular disease as the general United States population[1,6-10]. Neurobehavioural sequelae include delayed reaction times, deficit in verbal and non-verbal memory and difficulty maintaining vigilence.[1,14] Unrecognized OSA is reported to be a cause of increased mortality from highway crashes and industrial accidents[15]. There is also a significant co-morbidity associated with OSA[1,14].

OSA also has socio-behavioural consequences as shown in the Swedish study in 1995[11], where the presence of OSA was shown to be associated with a greater

frequency of divorce, worse self-rated perceived health, impaired work performance and more sick-leave.

Despite the many complications there is considerable disparity in the availability of investigations and treatment for such patients in different countries. In the United States of America it has been estimated that there is more than one sleep laboratory per 250,000 people. In Australia there is at least one per 1,000,000 people but in the United Kingdom the ratio is much lower[14]. In South Africa there are only nine sleep laboratories nationally (Sleep Society of South Africa), thus making the ratio 1 sleep laboratory per 4 million people. The difference in the availability of facilities may reflect a divergence in the relative importance with which health planners and policy makers view disorders of breathing in sleep[14].

Most patients with OSA, despite the finding of associated high mortality and morbidity, go unrecognized by the medical community[1], and unfortunately in OSA the patient is often the last to realize the extent of the mental and physical effects of the disorder![14]

A 1991 survey of patients' charts from 10 internal medicine and family practice clinics in Northern California found no mention of sleep apnoea or sleep disorders (National Commission on Sleep Disorders Research, 1993)[1]. This observation suggests the absence of any recognition strategy amongst primary care givers. In one managed - care setting between 1991 and 1994 the rate of new patients undergoing sleep studies increased 30% per year (possibly due to an increased awareness of OSA), resulting in recognition of perhaps 10% - 30% of individuals in the health management organization estimated to meet minimal criteria of OSA[1]. Sleep apnoea and sleep disorders are not included in expert surveys that identify important diseases needing further outcomes research or practice guidelines[1].

There is then a mismatch between the community prevalence of OSA and medical appreciation of its impact or presence in patient populations.[16] Although this may not be unusual for chronic diseases, the magnitude of the mismatch is rather large and pervasive in primary providers and health system managers[1,16].

The prevalence of OSA in the USA is reported as being 4% of men in the general population, 2% of women in the general population[17], 4-8% of men 40-65 years of age and 25% in those more than 65 years old. There are pockets of higher prevalence; clinical and epidemiological surveys find high rates of 20-62% of OSA in the elderly[1,16]. Expert opinion suggests that prevalence rates are high in early childhood[1,18] related to changes in craniofacial characteristics or the size of pharyngeal and tonsillar tissue[1,18]. Prevalence rates also vary with gender, obesity and race[1] (this is discussed in detail in the literature review).

Therefore OSA is not a rare diagnosis, and illness associated with OSA can be considered a major health problem[1]. It appears from the literature that OSA occurs as frequently in the population as other common diseases such as asthma.[1,16] However to date, there have been no studies done in South Africa to look at the prevalence of OSA or the symptoms most indicative of OSA (such as snoring, day time sleepiness, witnessed apnoeas, restless sleep and waking up gasping during the night).

1.2 Aims and objectives

The aim of the research therefore was to explore:

The prevalence rate of snoring and sleep apnoea symptoms in adults ≥ 20 years in urban general practice in the greater Johannesburg area.

The seven objectives of this study were to examine:

1. The number of patients who had OSA symptoms and snoring.
2. The relationship of demographic variables (age, gender, ethnic group) and OSA symptoms.
3. The link between Body Mass Index (BMI) and OSA symptoms.
4. The familial associations of OSA symptoms.
5. The association of OSA symptoms with other co-morbidities such as hypertension, heartburn, depression, angina, ischaemic heart disease, stroke and cardiac failure.
6. The association of life style factors (alcohol and smoking) and OSA symptoms.
7. The association of socio-econonic and educational factors with OSA symptoms.

1.3 Importance of the Research - Motivation and Rationale

The motivation of this study, to examine the prevalence rate of snoring and sleep apnoea symptoms in urban general practice, in the greater Johannesburg area, was to be able, firstly, to view the magnitude of this often overlooked but serious diagnosis, with associated high mortality and morbidity rates, at a local level, as mentioned.

Secondly there would for the first time be a prevalence rate of the symptoms of OSA, which would allow for comparison with overseas prevalence rates, thereby, bringing the attention of caregivers to this often overlooked condition and provide insight into the necessity for implementing some change in our diagnosis and

management of similar patients. Patients could be suspected of having OSA on the basis of predictive symptoms at an initial level of screening or primary care level. Thus the patients requiring sleep studies could be found more easily and unnecessary sleep studies avoided.

This study may form the basis of more extensive studies into the actual prevalence of OSA itself in South Africa.

Chapter 2

Review of literature

2.1 Introduction

This chapter begins with an explanation of what obstructive sleep apnoea is and why it occurs. It then continues with the overseas experience of prevalence rates of OSA and its most common symptoms, the mortality and morbidity of OSA and the associated risk factors as well as the related neuropsychological deficits caused by OSA in order to paint a background of the severity of this often overlooked entity. It concludes with a review of the relevant articles on the recognition of sleep apnoea by clinicians, the importance and implications of OSA, based on its symptoms.

2.2 Obstructive sleep apnoea

The pathophysiology of OSA is explained by the fact that as sleep comes on, muscle tone in the patient with OSA declines slowly and there is relaxation of the muscles that hold open the upper airway, initially resulting in vibration (snoring) and then obstruction (witnessed apnoea)[18].

At a certain critical point, the tissues in the pharynx close together when the patient begins to take in a breath. The suction force of the air being drawn into the lungs through the upper airway is too high and the airway is sucked closed. This

blocks the normal flow of air into the lung completely. This is called 'obstructive' sleep apnoea (OSA) and is experienced as an apnoea or breathholding, as witnessed by a sleep partner. Each additional chest movement becomes stronger and stronger in an effort to overcome the obstruction. Unfortunately the increased effort only makes the problem worse by creating greater suction pressure in the already obstructed airway. Due to the obstruction, air is not breathed in and there is a fall in arterial oxygen saturation (SaO_2) of at least 4%. By this time the struggle to breathe and the lack of fresh air flowing in and out of the lungs begins to disrupt sleep due to the high carbon dioxide (CO_2) level.[18] Arousal from sleep is produced as the high CO_2 level stimulates the respiratory centre to initiate breathing[18]. This is experienced as gasping and restless sleep. Sometimes the patients are aware of this part of the cycle but are usually unaware of the actual apnoeas.

2.2.1 Symptoms of Obstructive Sleep Apnoea

The symptoms used most commonly to characterise, and also to screen for OSA at certain times, may be categorised into several broad groups; breathing disturbances occurring during sleep (snoring frequency and intensity, choking or gasping, apnoeas or pauses); difficulties maintaining sleep (frequent awakenings, restless sleep); daytime sleepiness; fatigue; mood effects (depression and irritability); and general impairment of daily function and quality of life (eg, problems with performance of daily tasks, social interactions, sexual function).[19,20]

Clinical features of OSA are summarised as follows:[3,20]

1. Regular loud snoring.
2. Excessive daytime sleepiness.
3. Nocturnal choking, coughing or gasping.

4. Witnessed apnoeas (breath holding).
5. Morning headaches (related to the nocturnal oxygen desaturation).
6. Dry Mouth (arousals from sleep result in mouth breathing due to the blockage in nasal airway).
7. Waking up with bodily aches and pains.
8. Intellectual deterioration.
9. Personality changes.
10. Impotence (neuroendocrine changes related to OSA).
11. Obesity.
12. Social and marital problems.

It is important to note that:

1. The key symptoms are loud snoring and excessive daytime somnolence. According to Barnes[20], indeed any patient with this combination should be regarded as having OSA until proven otherwise.
2. Many patients sleep alone due to the noise of their snoring; therefore the absence of a history of witnessed apnoeas in no way excludes the diagnosis.[20]
3. Many patients are unaware of their apnoeas – they may wake but later may become unaware of such episodes.
4. A number of patients describe a fragmented sleep pattern with multiple arousals occurring for no obvious reason.
5. The condition is more common in males than in females – until the latter reach the menopause when they tend to catch up with their male counterparts.[20]
6. Obesity and heavy alcohol use are common aggravating factors.[20]

Sleep apnoea may be associated and/or worsened by the presence of several factors:

1. A narrow floppy throat i.e., reduction in pharyngeal diameter.

2. Fatty tissue in obesity, or increased soft tissue enlargement causes a narrow posterior airway space.[17]
3. A blocked nose which increases the suction pressure required to inhale, e.g., polyps, tumors, septal deviation.
4. Alcohol consumption that relaxes the upper airway dilator muscles causing upper airways resistance and snoring.
5. Sleeping on the back that results in movement of the mandible posteriorly and therefore further obstruction by the tongue or if the patient has retrognathia, i.e. a posteriorly positioned mandible.
6. A large tongue (hypothyroidism, Down's Syndrome).
7. Endocrine and metabolic causes - Hypothyroidism
   - Acromegaly
   - Cushing's Syndrome
   - Marfan's Syndrome
8. Familial and other predisposing factors.
9. Functional factors have also been implicated in apnoea generation. Pharyngeal temperature sensitivity is decreased in apnoeic patients, presumably through sensory nerves or receptor damage caused by snoring induced trauma.[17] Pharyngeal mechanoreceptors and the nerves important in airways reflex arcs which maintain airway muscle tone may be affected by the same mechanism. Muscle lesions are also seen in apnoeic patients indicating damage which may be important in increasing airway colapsability.[3,17] In addition mechanisms such as ventilatory instability and incoordination of muscle activity have been proposed which may show a tendency to worsen with time.[17]

Kingman et al elaborate that the most sensitive and specific method for diagnosing OSA, is to perform an overnight polysomnogram.[1] This approach provides comprehensive data, enabling quantification of sleep fragmentation and

assessment of interrelationships among breathing and oxygen saturation, sleep and movement variables. Sleep architecture and arousals are assessed by EEG (electro encephalogram, from central and sometimes occipital leads), right and left electro-oculograms (EOG), and electro-myogram (EMG). Breathing is assessed qualitatively, with airflow measured with thermistors or nasal pressure and oximetry measures continuous haemoglobin oxygen saturation. Other measures often used include electrocardiogram, body position, snoring (detected by microphones), assessment of bodily movements and chest and abdominal effort.[19]

Johns argues that the severity of chronic daytime sleepiness in OSA patients is an important aspect of each patient's assessment and thus a simple standardized test for measuring a patients general level of sleepiness, which is independent of short-term variations in sleepiness, as mentioned by Kingman et al, would be invaluable. Such a test is the Epworth Sleepiness Scale (ESS), developed and validated by Johns.[21] This simple self administered questionnaire provides a measurement of the subject's general level of daytime sleepiness and shows a significant difference between normal subjects and patients in various diagnostic groups including OSA, narcolepsy and idiopathic hypersomnia. In patients with OSA, the ESS scores significantly correlated with the AHI and the minimum SaO_2 recorded overnight in polysomnography.[21] It is important to note that this questionnaire based scale as brief and simple as the ESS is, can give valid measurements of sleep propensity in adults and is sensitive enough to distinguish patients who simply snore from those with even mild OSA.[21]

2.3 Prevalence of Obstructive Sleep Apnoea and Symptoms

2.3.1 Prevalence of Obstructive Sleep Apnoea Symptoms

The distribution of the prevalence of self reported symptoms of OSA obtained from a modified Specialised Centres of Research in Cardiopulmonary Disorders Sleep Questionnaire conducted in USA, according to gender, is described below.[19] The symptoms asked included snoring, gasping, apnoeas and excessive daytime sleepiness.

For community sample (USA) studied reports of 'snoring ever disturbing others' was reported by 68.0% of males and 50.0% of females whilst frequent or greater 'loud snoring' was reported by 34% of males and 22% of females. The number of subjects waking up at night gasping for breath either frequently or always was 17.0% in males and 6.0% in females. It was noted that the prevalence of observed apnoeas occurring frequently or always was 4.0% for males and 2.0% for females. Frequent or greater excessive daytime sleepiness was reported by 20.0% males and 17.0% females.[19] One may conclude that symptoms of OSA are common in the population with males having the higher prevalence.

It has been reported in the Swedish Obese Subjects study that frequent snoring ("often" or "very often") occurred in 51.6% of the men and 33.0% of the women. Frequent witnessed apnoeas were reported by 33.4% of the men and 12.6% of the women.[11]

Other epidemiological studies found prevalence rates of 19.0% for snoring every or almost every night in an unselected population with 25.0% snoring prevalence

in males and 15.0% snoring prevalence in females.[22] It was further found that for men 41 to 64 years of age 60.0% snored while 40.0% of women in the same age group snored.[22] Koskenmo et al found prevalence rates of 30.0% for people 40 to 69 years for habitual snoring.[23] For snoring in 30 to 69 years old age group, for both sexes, prevalence rates of 15.5% were reported.[24] For married couples, it was reported that 86.0% of husbands and 57.0% of wives were habitual snorers in the epidemiological survey by Norton et al.[25]

The variation in reported snoring prevalence from 15.0% to 86.0% may be explained by the ways in which the history was elicited for snoring. There are straightforward questions regarding the occurrence of 'any snoring' or 'loud or disruptive snoring', those formulated to scale the overall intensity or quality of snoring (e.g. from soft to 'can be heard through a closed door'), and questions asking about associated gasping in an attempt to distinguish subjects with 'resuscitative snoring' from those with 'simple snoring'. Use of graded responses regarding frequency and intensity of snoring can improve discrimination.

2.3.2 Prevalence of Obstructive Sleep Apnoea

Sleep apnoea (OSA) has been shown to be common in adults and is the most common disorder that is diagnosed after a full night of polysomnography (PSG) in most sleep labs[26].

However due to the variability in measuring, defining and qualifying OSA parameters, comparison and interpretation of data from various centres and locales is difficult.[16] Most researchers use a working definition of five apnoeas per hour to define 'sleep disordered breathing' (an apnoea is agreed upon as a cessation of breathing for 10 or more seconds)[3]. OSA is defined as a condition

where at least 15 witnessed apnoeas/hypopnoeas, per hour of sleep (AHI > 15) occur in combination with two or more clinical features, namely, excessive daytime sleepiness, loud snoring, disturbed sleep and secondary polycythaemia or ankle swelling without other known causes[3]. Alternatively, according to Pillar et al, a definite diagnosis of OSA is based on polysomnographic findings of more than 10 witnessed apnoeas/hypopnoeas per hour of sleep (AHI > 10), associated with a decrease of at least 4 % in oxygen saturation[27]. However, according to Mc Namara et al, recent studies have shown that some forms of OSA may occur without the presence of snoring or witnessed apnoeas, but with obvious clinical effects.[14] Similarly, daytime sleepiness may not occur, but instead the clinical picture may mimic an anxiety state, especially in women[6].

The prevalence estimates that have been calculated for data from large samples have been based predominantly on estimates using the AHI (Apnoea / Hypopnoea Index) without consideration of other clinical data, and have used different cutoff values.[16] Values of 10,15,20,15 and 30 for AHI have been used in different settings to distinguish pathological from normal degrees of sleep related breathing disturbances.[19] It is because of those factors that there is a 'range' of prevalence estimates, with consideration given to the methods used to derive such estimates, and the sampling assumptions made[16,19].

The original screening questions for estimates of OSA prevalence were derived from clinical case series, and center reporting of signs and symptoms associated with severe OSA. The prevalence of OSA in middle aged adults was approximated at 0,3-1%[1,4]. Subsequent larger and more general samples have suggested at least a twofold higher prevalence of OSA in the USA.[1,16] It was also shown that about 1% of an unselected inpatient population admitted to a general hospital in Italy during a one year period had OSA[28]. Generally however, there appears to be a wide range in the reported prevalence of OSA of 1-9%[14,16]. The

higher prevalence rate may be explained by the fact that different questions or different populations were used in the studies.

There are pockets of higher prevalence; clinical and epidemiological surveys find high rates of 20-81% of OSA in the elderly[1,16,19]. When considering an AHI>5 and the presence of self-reported sleepiness as OSA, the prevalence estimates were changed in that population to 2% in women and 4% in men respectively.[1,16]

A survey of Wisconsin State employees less than 65 years of age suggested that prevalence of OSA (defined as an AHI>5 and 4% O_2 desaturations) was 9% in women and 24% in men[16].

Expert opinion suggests that prevalence rates also increase in early childhood[1,18], due to changes in craniofacial characteristics and/or the size of pharyngeal and tonsillar tissue[1,18]. In the Cleveland Family Study, 2% of children less than 18 years had an AHI>10.[16] Among children related to a patient with laboratory diagnosed OSA, the prevalence of sleep disordered breathing was 8%[19].

Prevalence rates were increased with obesity, age, male gender and non-Caucasian races[1] (in the Cleveland family study in the USA). It also appears from the literature that OSA occurs as frequently in the general adult population as other common diseases such as asthma;[1,19] OSA is therefore probably common (just like asthma) and may be specially prevalent in certain subgroups (just like asthma).

2.4 Risk factors of OSA

According to Kingman et al, the strong well documented risk factors for OSA are obesity and age and some data suggests that male gender and family history of OSA also carry significant risk. Additional conditions that may predispose patients to OSA are race (African American), alcohol and tobacco use, low vital capacity, snoring, excessive daytime sleepiness, and medications (sedatives and sleep promoting agents)[1].

2.4.1 Obesity and body fat distribution

Obesity has been considered to play a significant role in the pathogenesis of OSA, because the majority of patients with OSA are obese and weight loss is beneficial in the treatment of OSA.[29]

Obesity, as defined by a Body Mass Index (BMI) greater than 28 kg/m^2 is present in 60-90% of OSA patients evaluated in sleep clinics in the USA[1]. This is corroborated by Shinohara et al where 60% of obese subjects in Japan exhibited OSA.[29] However, obesity and OSA are less tightly linked in more general population samples[1]. In a study by Lavie, looking at the incidence of sleep apnoea in a working population, none of the workers with AI≥10 (sleep apnoea) were obese.[4] Apnoeic and non-apnoeic workers had no significant difference in how much their body weight varied from ideal body weight.[4] Nonetheless, most data supports the previous reports, showing that obesity is a major contributor in the pathogenesis of OSA.[29] Fat accumulation around the neck, reduction of lung volume, and the structural alteration of pharyngeal muscle fibers have been

considered contributory factors to the narrowing of the upper airway, thus promoting collapse of the airway in apnoea.[29]

There are two types of obesity, namely 'visceral fat obesity', characterized by a marked accumulation of visceral fat in the abdominal cavity, and 'subcutaneous fat obesity', characterized by fat accumulation mainly in the subcutaneous region[29]. Measures of central obesity i.e., visceral fat obesity, e.g., waist to hip ratio or neck circumference, have a relatively better correlation with OSA than does BMI[1]. For instance, among subjects with a BMI<28, an increased neck circumference is described as an identifying characteristic for those with greatest OSA risk, according to Kingman et al. This is corroborated by Pillar et al who report that obesity itself, was not an independent risk factor for OSA unless it was connected with an increased neck circumference.[27]

Another recent study by Shinohara et al investigated the connection between abdominal visceral fat accumulation and the presence of OSA in obese subjects.[29] He found that visceral adipose tissue (AT) area as determined by computed tomography (CT) at the level of the umbilicus, and its ratio to total AT area, were significantly greater in OSA patients, compared with patients without OSA. A multiple regression analysis revealed that the visceral AT area significantly correlated with witnessed apnoea index when age, AT mass and lean body mass were taken into account. In contrast, no significant differences were observed in body height, body weight, BMI, abdominal AT area, lean body mass and body fat ratio between the OSA and non OSA groups.[29] This corroborates the study by Pillar et al on the predictive value of risk factors for OSA in which BMI did not meet any critical significance to enter the model.[27]

Experimental data cited by Kingman et al suggest that it is associations between OSA and metabolic processes that influence fat accumulation and deposition (e.g.

insulin resistance common to both conditions). Hypoxia or sleep interruption in OSA contributes to changes in body composition over time by increasing stress hormones or altering glucose metabolism.[1] Thus OSA may promote development of central obesity. Patients with OSA may also overeat in order to stay awake and overcome their daytime sleepiness[1]. These patients may therefore enter a cycle where the fatigue results in increased eating which causes increased central obesity which results in deterioration of the OSA.

Shinohara et al study further demonstrated that the relationship of OSA and obesity can be partly ascribed to changes of the upper airway, since the cephalometric measurements demonstrated that in obese subjects with a large visceral adipose tissue area, the soft palate was positioned backward and downward, and the base of the tongue fell back against the posterior pharyngeal wall with inspiratory efforts[29].

Such abnormalities of the upper airway in 'visceral fat obesity' could be considered to be predisposing factors completing pharyngeal occlusion during sleep[29]. A further hypothesis is that the increase in visceral fat, rather than subcutaneous fat and chest fat, may have a predominant effect on reducing lung volume and thoracic compliance, by elevating the diaphragm and restricting its motion. It is well documented that even relatively small weight reduction is associated with a significant improvement in OSA[29].

2.4.2 Age

Data from the Cleveland Family study is representative of the increasing prevalence of OSA and snoring with age.[1] OSA as an illness is commonly recognized in the 5th through 7th decades; however OSA is more prevalent in the

older population (age>65 years) than is reflected by the demographic distribution of speciality practice or clinical referrals.[1] For example, more than 70% of one nursing home population had AHI>5, and more than 40% had AHI>15.[29] Clinical and epidemiological surveys find high rates (20%-62%) of OSA in the elderly.[1,16] One community study demonstrated that 81% of elderly residents had an AHI > 5 and 44% an AHI > 20.[19] A study considering evolution of moderate OSA has found that there is a deterioration in AHI over a mean of 17 months irrespective of the change in BMI and upper airway anatomy.[17]

However, clinical recognition of OSA may not occur in the elderly because snoring is more common in that group, because other morbid conditions overshadow OSA presentation or because there are different consequences of OSA in the older age group.[16]

Expert opinion suggests that prevalence rates also increase in early childhood related to craniofacial characteristics or the size of pharyngeal and tonsillar tissue[1,18] as previously mentioned. This is further corroborated by Shinohara et al, who cite that current clinic and hospital based literature emphasizes tonsillar hypertrophy and genetic diseases as risk factors for OSA in children.[29] However there are no population studies in this age group and recognition strategies could be differently constructed for children than for adults.[29]

In summary then, increasing age is shown to be a significant risk factor associated with OSA in the studies cited above.

2.4.3 Gender

OSA affects 4% of men and 2% of women between the ages of 30 and 60.[17] In general population samples, the male:female ratio is approximately 2-3:1, compared to 10:1 to 90:1 ratio from clinical based studies.[1] Pendlebury et al found a significant difference between women and men in the incidence of OSA with women having a much lower incidence, and a slower progression of OSA in women than in men.[17]

It should be noted that clinical under-recognition of OSA in females may be partly based on underreporting and/or nonspecificity of snoring and witnessed apnoea symptoms in women as compared to men.[1] This is corroborated by Grunstein et al, in the Swedish study,[11] where it was found that the symptoms of snoring and witnessed apnoea were reported less frequently by women than men despite objective evidence of a similar degree of witnessed apnoea severity.[11] There are several possible explanations. Symptoms of OSA may be more socially acceptable for men; for example, in a woman, loud snoring and witnessed apnoea may attract more criticism than in a man,[11] and sleepiness at work may have variable acceptance depending on the sufferer's gender.[17]

2.4.4 Familial and genetic risk

Familial and genetic risk is reported to have a significant association with OSA.[1,3,14] The risk of OSA, independent of age, obesity and gender is increased two to four fold with increasing numbers of affected family members.[1] Kingman et al found that not only OSA itself but also the symptoms attributed to OSA show a significant familial pattern e.g. snoring appears to be more likely if there is a

family history of snoring. The risk increases as the affected number of family members increases.[1] Such familial risk may be the result of similarities in facial structure affecting upper airway dynamics in sleep.[14] Estimates are that 40-50% of the variance in apnoeic activity can be attributed to familial factors.[1]

There are rare genetic syndromes recognized as having an inherent risk of OSA e.g. Marfan's Syndrome and Down's Syndrome, the former because of the laxity in connective tissue, and the latter due to pharyngeal crowding, brachycephalic head form, and obesity.[1] Pierre-Robin Syndrome, evident by a shortened mandible, is also associated with snoring and sleep apnoea in children and adults.[3]

While significant, the overall magnitude of the relationship between familial or genetic factors and OSA does not appear to be sufficiently strong to justify screening of asymptomatic family members.[3]

2.4.5 Race

Data from the Cleveland Family study indicate a higher prevalence of age-adjusted OSA in Blacks compared to Whites.[1] Racial differences were not accounted for by differences in BMI, alcohol exposure, or tobacco use; the effect was most evident in individuals <25 years old and therefore African Americans could be a group at particularly high risk for OSA-related effects and complications. According to Kingman et al, other populations in which a high prevalence of OSA are suspected are Mexican Americans and Pacific Islanders.[1] In these populations, comorbid factors of obesity, hypertension and non-insulin dependent diabetes are more prevalent than in Caucasians. Formal studies with comparison groups, however have not been performed, so the prevalence of OSA relative to comorbidity and to other races is not known.

2.4.6 Tobacco and Alcohol Use

According to Kingman et al smoking has been suggested to increase the risk of snoring, possibly by producing pharyngeal inflammation. Data from the Wisconsin-Cohort Study also suggest that a smoking history may be a dose-dependent risk factor for OSA.[1]

Alcohol also can be an important precipitant of apnoea during sleep.[3,14] Alcohol preferentially suppresses the activity of upper airway dilator muscles in comparison to the diaphragm and therefore has been shown to increase upper airway resistance.[3] Alcohol increases the number of apnoeas in patients with the disease and induces apnoea in asymptomatic male snorers.[3] This is corroborated by Kingman et al who cite that alcohol use has been implicated as a significant risk in some but not in all studies. The differences in these studies are not obvious, but they could relate to the level of alcohol use in the different cohorts or to variability in the accuracy of classifying alcohol exposure.[1] According to Mc Namara et al, although some studies have suggested that lifetime alcohol consumption may be a risk factor for the development of OSA, other studies have failed to find a link between life time alcohol consumption and OSA.

In summary, it appears that acute alcohol ingestion promotes the development of witnessed apnoea during sleep,[14] but that OSA is not linked to mild long term alcohol consumption.

2.4.7 Vital capacity

A low vital capacity has been identified as a co-risk factor for OSA in the elderly and in the Cleveland family study.[1] Reduction in vital capacity are associated with cardiovascular morbidity and mortality.[1] The relationship between vital capacity and vascular morbidity may be partly based on an association with OSA.[1] It should be noted that a low vital capacity may be a marker for central obesity which as previously cited is a risk factor for OSA.

2.4.8 Snoring

Snoring is often reported for years to decades before the onset of OSA - related illness, as determined by symptoms of excessive sleepiness or cardiopulmonary failure.[1] Although snoring may be a symptom of OSA, it may also be a risk factor. As a risk factor, snoring could contribute to the progression of OSA either indirectly, as a marker of nasal or nasopharyngeal disease, such as allergy, or directly by inducing pharyngeal narrowing. Such narrowing would be caused by vibration injury to the upper airway mucosa or surrounding structures.[3] There are no studies directly addressing the progression if any, from 'simple' snoring to fully expressed OSA,[1] however snoring progression to OSA is generally considered to be a continuum. Snoring is not a risk factor that can be taken in isolation and most people would now consider asymptomatic snoring as a mild form of OSA possibly expressed as sleep related breathing disorder (SRBD) (Dr. AJ Bentley, personal communication).

2.4.9 Sleepiness

Sleepiness may be a risk factor in the amplification of apnoeic activity over time.[1] It may also be a significant symptom in diagnosing OSA as cited by Lavie.[4] Studies reported by Kingman et al demonstrate that sleep restriction or total sleep deprivation increases the number of witnessed apnoeas both in smokers and in patients with modest degrees of apnoeic activity (AHI 10-15).[1] It is interesting to note that spouses of patients with OSA have twice the prevalence of apnoeic activity compared to spouses of control subjects, a finding that could relate to chronic sleep disruption experienced by the former group.[1] These reports suggest that prior sleep can affect the expression and consequences of OSA. Patients working in occupations with high potential for sleep restriction and obesity are observed to have high prevalence of OSA.[30]

2.4.10 Medications

The use of medications in chronic diseases can affect sleep quality, sleep architecture, and daytime symptoms, according to Kingman et al.[1] Sedative medications, some antihistamines as well as sleep-promoting agents, can directly affect witnessed apnoea expression, while some antihypertensive agents can increase or decrease apnoeic activity by as much as 20%.[6]

Increasing numbers of medicines are associated with sleep complaints in the elderly perhaps due to the fact that medications alter sleep-wake patterns or promote arousals.[1]

2.5 Mortality associated with OSA

Due to the biological significance of sleep interruption and hypoxaemia on behaviour and on cardiopulmonary physiology, the impact of OSA on mortality in the general population could be significant.[1]

Unrecognized OSA is reported to be a cause of increased mortality from motor vehicle and industrial accidents[1,14]. It is also strongly suspected to produce adverse events and even death with preoperative anaesthesia and during recovery from operations[1].

Data from a number of centres show a higher actual accident rate in patients with OSA compared with controls.[14] Patients with OSA also perform worse on driving simulator tasks. Mc Namara et al reported that 15 patients with OSA ran off the road 101 times in a 60 - 90 minute simulated highway drive compared with only twice in 10 controls.[14] However self-reported accident rates may not differ between patients with OSA and controls, which questions the reliability of patient history in this area.[14] Patients may be reluctant to admit problems in a questionnaire, due to the fear of losing driving privileges which can be economically and socially disastrous.

Several studies have examined mortality in OSA with observations derived from patient clusters attending speciality clinics[1]. Two of the early studies demonstrated increased mortality ratios for patients with severe OSA treated with conservative therapy or surgery (UPPP – Uvulo Palato Pharyngo Plasty – therapeutic efficacy of 50.0%), compared with patients treated with tracheostomy or continuous airway pressure (CPAP). Both those studies demonstrated mortality rates of approximately 6.0% per 5 to 8 years. Deaths appear to be largely attributable to vascular diseases.[1,19] Increased mortality was most apparent in

subjects younger than 50 years old (i.e. eight year mortality rate: 10.0% for those with more and 2.0% for those with less OSA).[19]

Another study found that all-cause mortality rates were greater in patients who presented to a sleep laboratory than in the general population.[19] After adjusting for BMI, hypertension and underlying cardiopulmonary disease, the apnoea index predicted all cause mortality in a dose dependant fashion. Two of the three community based studies of mortality focused on elderly subjects. Among subjects greater than 65 years of age recruited from the community, no significant relationships were demonstrated between AHI and mortality.[3,19] It is possible that older subjects with OSA may represent 'survivors' who are less susceptible to the endorgan effects of the disorder.[19] In a broader age range, a long term prospective community study demonstrated that a history of sleepiness and sleeping greater than 8 hours increased mortality by 30%.[19] These analyses were adjusted for multiple potential confounders and were not likely biased by survival or selection biases. The limitations of the aforementioned studies – retrospective data collection, with the possibility of survival biases; potential selection biases related to mortality patterns in patient groups with high comorbidities, relatively small sample sizes or few mortal events; and lack of appropriate control groups – therefore make it difficult to assess the overall impact of OSA and different degrees of OSA on mortality in all-comers. The aggregate available data do suggest, however, that increased mortality rates are likely in individuals with severe levels of apnoea, specially those in whom OSA presents in middle life.[3,19]

2.6 OSA association with vascular diseases

OSA patients appear to have twice as much hypertension, three times as much ischemic heart disease, and four times more cerebrovascular disease than the general United Stated population.[1,3]

2.6.1 Hypertension

Systemic blood pressure, pulmonary arterial pressure or both increase during sleep with sleep-related witnessed apnoeas.[31] This rise in systemic and pulmonary arterial pressure may persist during the daytime.[31]

Prevalence of daytime hypertension in patients with OSA has been reported to exceed 40%;[6] conversely, the prevalence of OSA in hypertensives has been estimated to be 20-30% and thus to be at least threefold greater than in subjects without hypertension.[6] When the effects of obesity were carefully assessed and analyses stratified by BMI, the relationship between AHI and blood pressure remained significant.[1,6]

More direct proof of the association would be demonstrated by the fact that treatment of sleep apnoea alone eliminates hypertension.[3] Acute treatment of OSA with nasal CPAP reduces markedly the variability of blood pressure during sleep that results from the cyclical oscillations in pressure in relation to witnessed apnoea. Patients with OSA and hypertension have been described who normalize their pressure following tracheostomy treatment. The largest series of sleep apnoeics with hypertension who were treated with tracheostomy show a subset of patients whose hypertension was essentially cured by treatment of their obstructive

apnoea.[3] There are some patients with nasal CPAP, in whom reductions of blood pressure are large and of clinical consequence.[3] This is corroborated by Kingman et al, who cite reductions of 5-10 mm Hg in mean blood pressure in OSA patients post treatment of OSA.[1]

There is a stronger relationship observed between hypertension and OSA in younger subjects <50 years than in subjects older than 50 years.[1] One report shows that in the elderly, sleep disordered breathing is associated with a fall rather than an increase in blood pressure.[1] OSA may have different consequences in cardiovascular regulation in older and younger people; alternatively these observations may be due to a survival bias. Snorers suffer twice as much hypertension as non-snorers, a difference that appears to be driven by some degree of undiagnosed OSA.[3]

2.6.2 Ischaemic Heart Disease

The cyclical changes in blood pressure associated with falls in 0_2 saturation (SaO_2) occurring in relationship with apnoeic events, might conceivably increase the risk for other acute cardiovascular events e.g. myocardial infarction.[3] One of the first studies in this area was done in Hispanic Americans and reported a higher prevalence of myocardial infarction in snorers (7,4%) as compared to non-snorers (2,2%).[3] A case-control study of snoring in 50 patients with documented myocardial infarction and 100 age-and sex-matched hospital and community control subjects, demonstrated an odds ratio of 4,4 (confidence interval: 1.1-17,9) for the symptoms of snoring every night.[9] A prospective study of incident events in male Finnish twins demonstrated an age-adjusted relative risk of 1.91, which was reduced only to 1.71 with adjustment for BMI, hypertension, smoking and

alcohol.[1] An association of OSA and 'nocturnal' angina and or asthma has been shown in two case-control studies.[10] These observations may contribute to the known circadian expression of ischaemic events and sudden death.[1]

In another study which examined overnight polysomnography results in unselected male survivors of acute myocardial infarction and in controls, the increased relative risk of myocardial infarction in those patients with OSA versus non OSA patients was extremely high at 23,3.[3] Thus the data suggest that sleep apnoea is an important risk factor for acute vascular events. This is corroborated by a recent paper studying ECG changes in OSA. It was noted that OSA has acute and chronic effects on the cardiovascular system and both right and left sides of the heart are affected.[31]

There were characteristic ECG changes in OSA which were not associated with any established cardiac problem.[31] The most common patterns were deep S waves in leads I, AVF, V6 with left axis (27%), deep S waves in leads II, III, AVF and V6 with left axis (12%) and deep S waves in leads I, II, III, AVF and V6 with left axis (11%). There was a statistically significant difference in polysomnographic data and daytime PaO_2 between OSA patients with and without ECG findings. The latter had the mildest evidence of OSA ($P<0.01$). This allows one to speculate that the ECG findings were probably the result of OSA.[31]

This corroborates the study cited by Kingman et al where right ventricular (RV) and left ventricular (LV) hypertrophy have been described in 71% and 31% respectively of OSA patients. Furthermore Kingman et al state that cardiac arrhythmias are reported to occur commonly in OSA patients. However, it was noted that abnormalities of interventricular septal movement and LV filling that occur during OSA were reversed with treatment.[1]

2.6.3 Cerebrovascular Disease

The studies cited by Pack show that habitual snoring is associated with an increased risk of cerebral infarction and that an increased risk is still found after adjusting for age and BMI.[3]

Data obtained from the Finnish Twin Registry, demonstrated that the combined adjusted relative risk for both ischaemic heart disease and stroke was 2,08, as compared to 1,71 for ischemic heart disease alone.[1] A study of some 400 stroke victims and age and gender- matched control subjects demonstrated an odds ratio of 3.2 for the symptom of snoring.[1] According to Mc Namara et al, snoring is a strong risk factor for sleep related strokes while sleep apnoea symptoms (snoring plus reported witnessed apnoeas or excessive daytime sleepiness) increase the risk of cerebral infarction with an odds ratio of 8,0.[14]

There is no information currently available on an increased thrombotic tendency or accelerated atherosclerosis in OSA.[1,14]

2.7 Neuropsychological and social consequences of OSA

The literature on neurocognitive impairment associated with sleep apnoea has addressed intellectual ability, memory, attention and concentration, complex problem solving, visual and psychomotor performance. In most studies, the performance of those patients with large numbers of measured apnoeas was compared with that of age-matched healthy subjects.

Neuropsychologic measures of overall performance were moderately impaired in approximately 50% of the subjects with >30 apnoeas per hour of sleep.[1] Some but not all exhibited markedly delayed reaction times, deficits in verbal and nonverbal memory, and difficulty maintaining vigilance.[1] Several studies have found that patients with OSA who perform poorly on psychometric tests compared with controls have a variable degree of improvement after nasal CPAP therapy.[14]

OSA may lead to both impairment of work performance and driving.[14] One problem is that patients themselves may not be aware of their degree of sleepiness. Excessive daytime sleepiness is characteristic but not pathognomonic of OSA and is predominantly related to repetitive arousal and sleep fragmentation but may also be a direct effect of hypoxaemia. According to Mc Namara et al, patients with OSA perform worse on driving simulator tests than those without OSA;[14] fifteen patients with OSA ran off the road 101 times in a 60-90 minute simulated highway drive compared with only twice in twenty controls.[14] This is corroborated by George et al[5] who report data from a number of centers showing higher actual accident rates in patients with OSA compared to controls. Interestingly, self-reported accident rates may not differ between patients with OSA and controls, due to the fact that loss of driving privileges can be economically and socially disastrous to patients and they may therefore be reluctant to admit problems.[14] As Mc Namara et al point out evidence exists that treatment with nasal CPAP dramatically improves daytime sleepiness and even driving simulator performance.[14]

The Swedish Obese Subjects Study, looking at the psychosocial factors in sleep apnoea, revealed interesting results. The study demonstrated that OSA (measured by symptoms such as snoring, frequent witnessed apnoea and daytime somnolence) had a major impact on psychosocial function in obesity and a high

likelihood of OSA was associated with a greater frequency of divorce, worse self-rated perceived health, impaired work performance and more sick leave.[11]

Although estimated visceral fat mass was strongly related to a number of these psychosocial and economic parameters, OSA or symptoms strongly related to OSA, such as frequent daytime sleepiness, were important independent contributors as revealed by multivariate analysis.[11] It is well recognized anecdotally that loud snoring may promote marital discord and many of the reported symptoms in subjects with OSA, including personality changes, disturbed cognition and vigilance, reduced motivation and excessive sleepiness are not conducive to harmonious relationships, as expounded by Grunstein et al. In men it was found that sleep apnoea was a risk factor for divorce only when associated with frequent sleepiness.[11] In contrast OSA had a greater impact on divorce rate in women; women with OSA reported more anxiety-like symptoms compared to men and this may possibly influence relationships. Male partners of women with OSA may be less tolerant of loud snoring or daytime sleepiness than female partners of men with OSA.[11] These data emphasize the propensity of sleep apnoea to affect personal relationships.

Impaired work performance was found to be related to excessive daytime sleepiness, as was the amount of sick leave, which averaged 5 weeks more sick leave in the previous twelve months for those subjects with OSA vs. those subjects without OSA.[11] As can be seen, if these findings are extrapolated to the entire population, then OSA is likely to lead to a major reduction in productivity and increased health care costs.[1,11]

2.8 Recognition of sleep apnoea by Clinicians

Despite increasing numbers of epidemiological studies reporting OSA in the general population, the majority of individuals with apnoeas or hypopnoeas, snoring and sleepiness are unrecognized by the medical community.[19] Review of the literature demonstrates the clinicians' confusion as to the nature of OSA.[1,3,14,16] This is corroborated by Redline et al who mention that despite considerable clinical experience diagnosing and treating patients with OSA (often producing clinical successes that are among the most remarkable experienced by internal medicine specialists), there is substantial controversy regarding how to best define and identify the syndrome and disagreement regarding which patients have OSA.[19]

Kingman et al propose that delayed recognition of OSA may be due to ignorance of the illness, lack of general knowledge of the illness in the medical community or one specialist's lack of recognition of symptoms in the domain of another specialist.[1] However, Redline et al elaborate that although a disparity between community prevalence of chronic disease and clinical recognition is not uncommon, the large disparity between the community prevalence of OSA and its recognition in patient groups is. The reasons relate to deficiencies in recognition strategies among primary care providers and among specialists.[19] Patients may also present with different complaints; for instance patients with witnessed apnoeas who consulted psychiatrists had a common complaint of sleepiness; those who went to neurologists more often complained of neurocognitive difficulties, memory difficulties and behavioural problems; and those who went to pulmonary physicians had cardiopulmonary complaints.[1]

Another problem with diagnosing OSA based on history of symptoms alone is that many of these symptoms may be common in the general community and may have more than one aetiology.[1] For instance, Kingman et al emphasize how 'sleepiness'

may be a nonspecific symptom resulting from many common and acute conditions. Some 30-50% of community residents without sleep apnoea will have symptoms of moderate to severe sleepiness or report them on direct questioning. The causes for sleepiness in the community are domestic and occupational demands e.g. the number of children, number of jobs, shift work, etc., all factors that cause sleep restriction or sleep interruptions.[1,19] Reduced light-dark exposure and medications are prominent factors implicated in sleepiness in the elderly.[1,19] Hence, sleepiness as discussed by Kingman et al, unless qualified by chronicity or severity, is not significantly discriminatory enough to diagnose OSA.

It is crucial to develop recognition strategies that are appropriate and that optimize the available resources for diagnosis and treatment. This case finding may take place by clinicians at a primary health level where patients are questioned about the most significant OSA related symptoms on history (i.e. an amalgamation of symptoms rather than just one or two symptoms).[26] Of those symptoms, those attributable to disrupted breathing (snoring, apnoeas, nocturnal choking) best predict level of AHI.[11,19] In general reported snoring has relatively high sensitivity and reported apnoeas have high specificity.[11,19] The probability that a patient has OSA can be calculated according to a prediction formula and the estimates can be used to decide on subsequent diagnostic testing with portable monitors or a polysomnogram.[26] The inclusion of data on body habitus (body mass index (BMI) or neck circumference), age, gender, and blood pressure has been shown by several studies to improve predication formulae. Tables have been developed to translate clinical scores, based on neck circumference, hypertension, snoring and bed partner-reported nocturnal choking, to likelihood ratios. Knowing the overall prevalence rate of OSA in the patient population, one can calculate the probability that a patient with any given clinical score will demonstrate an elevated AHI. The problem however faced by most clinicians attempting to use these rules is that they involve complicated mathematical formulae that limit their usefulness.[26]

Physical examination has also not proved to be very helpful in identifying patients with OSA.[19] A recent study demonstrated that experienced sleep physicians had limited ability to predict OSA based on the patient's history and physical examination. In fact, their clinical impression had only 50% - 60% sensitivity and 63-73% specificity.[19]

Redline et al report that the most sensitive and specific method for diagnosing OSA, is to perform an overnight polysomnogram. The disadvantages of this approach are high cost, limited access and the possibility that monitoring in an unfamiliar environment may adversely influence the patient's sleep quality or otherwise produce data that are unrepresentative of a patient's usual sleep pattern.[19]

Pillar et al justly reason that since whole night polysomnographic recordings are expensive and time consuming, less expensive and easier ways of diagnosing OSA are required.[27]

Excessive daytime sleepiness measured by the ESS may be used as one of the significant symptoms in diagnosing OSA by Clinicians.[21] This was corroborated by Lavie in a study looking for the first time at the relationship between OSA and excessive daytime sleepiness.[4] A significant association was found between the complaint of excessive daytime sleepiness and the incidence of OSA. Other variables for which there were statistically significant differences between apnoeic and non-apnoeic workers, were heavy and disturbing snoring, excessive movement in sleep or restless sleep, frequent headaches, and comorbidity of hypertension.[4]

Since previous studies had demonstrated that using questionnaires alone to diagnose OSA had unsatisfactory sensitivity and specifity,[32] Pack citing two

studies, suggested a predictive model of Apnoea Index (AI) based on questionnaires and pharyngeal examinations.[3] The maximal AI of that model could not exceed 50, which limited the model to mild to moderate cases of OSA. Thus a study by Pillar et al was undertaken which evaluated the specificity and sensitivity of several risk factors, signs and symptoms in predicting OSA.[27] This revealed that FOUR variables were significant as positive predictors of OSA. These four variables were:

1. Self reporting of apnoeas
2. Neck circumference index
3. Age
4. Tendency to fall asleep unintentionally

Redline et al, state that the symptoms attributable to OSA, namely snoring, witnessed apnoeas and nocturnal choking or gasping best predict levels of OSA.[19] OSA should be considered by clinicians on the basis of symptoms of disrupted breathing during sleep (snoring and witnessed apnoeas), daytime sleepiness unexplained by other factors, and response to trials of specific treatment.[19] Thus on the basis of the several variables mentioned by the various authors above, clinicians, whilst maintaining a high index of suspicion, will be able to better recognize and identify patients with or at risk of OSA at a primary health care level. These patients may then be referred to a sleep specialist centre to obtain further important confirmatory evidence of the diagnosis and other underlying conditions.

In summary then, the most frequent symptoms associated with OSA are snoring, witnessed apnoeas during sleep, excessive daytime sleepiness and restless and non restorative sleep.[11,19,27] Other symptoms include frequent arousals during sleep, morning headaches, dry mouth, tiredness and body aches and pains.[1] Studies

outlined by Kingman et al and Shinohara et al indicate that not only the above symptoms but also body measures of central obesity, and to a lesser extent blood pressure, gender and age are good predictors of OSA in the community.[29]

This present study focussed on the most significant symptoms of OSA cited from the literature namely: snoring[1,3], witnessed apnoeas,[19,29] restless sleep,[1,4,19] waking up gasping[19,29] and excessive daytime sleepiness.[4,19,21,29] Although these five main symptoms are the ones considered for the purposes of this study, as far as possible the other four symptoms were also considered; these are waking up with headaches, dry mouth, tiredness and body aches and pains.[29] As far as possible all nine symptoms have been considered and studied.

If the prevalence of OSA symptoms is known at a local level, the extent of the public health impact in proportion to the risk such individuals pose while driving, and the extent to which they engage in potentially hazardous activities may be realised.

Recognition of OSA by clinicians and appropriate treatment will lead to relief of sleepiness, with attendant improvement in quality of life and thus be of clear benefit to the individual patient.[19] Individuals with underlining cardiovascular and pulmonary disorders may be especially vulnerable to the effects of OSA and would benefit from recognition and treatment. It behooves physicians who are involved with diagnosis to maintain a high index of suspicion and to assure that all individuals are screened with respect to OSA symptoms.

Chapter 3

Research Methodology

3.1 Definitions

OSA (Obstructive Sleep Apnoea) For the purposes of the study, the term OSA refers to a medical disorder associated with abnormal respiration during sleep (sleep disordered breathing), with the occurrence of episodes of complete or partial pharyngeal obstruction, resulting in multiple arousals, snoring, daytime somnolence and nocturnal oxygen desaturation.

OSA symptoms - refers to the symptoms most likely to be associated with OSA. In this study it referred to snoring, witnessed apnoeas, restless sleep (frequent awakenings), waking up gasping, excessive daytime sleepiness, waking up in the morning with headaches, dry mouth, body aches and pains.

3.2 Study Design

The study was a Cross Sectional Descriptive study.

3.3 Sample Selection

The study was conducted at two general/primary care practices in Randburg (North of Johannesburg), two general/primary care practices in Eldorado Park (South of Johannesburg) and one general/primary care practice in Lenasia South (South-West of Johannesburg). These practices were chosen specifically to be representative of the different race groups in order to assess the contribution of race to the prevalence rate. The practices were chosen based on the first willing doctor in each geographic area that allowed the study to be conducted at his/her practice. The Eldorado Park doctor operated two practices and therefore both practices took part in the study. The second Randburg practice was needed due to the fact that there was a change of receptionist in the first practice, which caused a lack of commitment to complete the study.

The Lenasia South practice consisted predominantly of Indian patients, the Randburg practices were predominantly White patients, and the Eldorado Park practices were predominantly Coloured and Black patients.

The study sample consisted of adult patients ≥ 20 years at these practices. A cutoff age of 20 years and above was chosen for adulthood for the purposes of this study due to the fact that the study looked into the relationship between occupation and OSA symptoms and it was felt that ages below 20 may not have had the opportunity to enter the labour market, while ages much greater than 20 may have excluded a large number of adults. Every patient over the age of 20, not having completed the questionnaire before, coming to the consulting room was given a questionnaire by the receptionist, prior to consultation with the doctor and was asked to fill in the questionnaire before seeing the doctor.

Sample size was determined by statistical calculations performed by Dr. Esther Viljoen, a statistician from the Medical Research Council. The primary objective of this study was to estimate the prevalence of snoring and sleep apnoea symptoms reported by adults in urban general practices in Johannesburg. For the purposes of sample selection for this study mean prevalence of 20% was used, which was based on the lower limits of snoring prevalence found in several previous studies.[11,19,22-25] Assuming an expected prevalence of 20%, a sample of at least 287 subjects was required to estimate the prevalence to an accuracy of within 5%. The sample size calculations were made by employing the programme StatCalc in the Epi Info V6.04 software developed at the Center for Disease Control and Prevention (CDC), USA which is shareware (freeware) provided by the World Health Organization (WHO) in Geneva, Switzerland.

It was hoped that a total of 100 questionnaires would be obtained in each of the three geographical areas to fulfill the required sample size. A sample size of 304 was reached in this study thus meeting the criteria of adequacy by the statistician.

3.4 Pilot Study

A pilot study was conducted at a separate practice in Randburg, in order not to use the study population. Permission for such a study was granted and no difficulties were encountered in the pilot study.

Ten questionnaires were completed, these were inspected together with Dr. Alison Bentley to ensure that they were fully completed and that no misunderstandings or ambiguities had arisen in the administration, format, wording or completion of the questionnaires. No problems were encountered in these inspections and therefore it was agreed to proceed with the main study.

3.5 Measuring instrument - questionnaire

The measuring instrument was a questionnaire which was self administered. A covering letter served the purpose of explaining the procedure and obtaining informed consent to fill in the questionnaire (see appendix 1 for covering letter and questionnaire).

The questionnaire consisted of 24 questions. The questionnaire was based on the questionnaires used at SleepWake Laboratories (Dr. Alison Bentley, Sleep Specialist). The original questionnaire used at the above mentioned laboratories, is given to patients who have been admitted for an overnight polysomnogram for an accurate diagnosis of their particular sleep disorder.

The questionnaire commences with questions pertaining to demography (Questions 1,2 & 6). As the issue of "racial grouping" is a sensitive one in South Africa, a general question about ethnic groups has been asked.

Questions 3,4,5 try to ascertain educational level.

Question 6 ascertains ethnic group.

Question 7 and 8 about height and weight are asked in order to determine the Body Mass Index (BMI).[2]

Question 9 and 10 attempt to see the association between symptoms of sleep apnoea and patient awareness.

Question 11 to 14 attempt to examine patient's sleep practices (number of wakings and reasons for waking).

Question 15 and 18 examines the symptoms of OSA.

Question 16 examines the symptoms of OSA five years ago.

Question 17 seeks to investigate the hereditary aspects of sleep apnoea symptoms.

Question 19 is the Epworth Sleepiness Scale (ESS), which is a predetermined scale used in sleep laboratory questionnaires, to score the extent of daytime somnolence in an individual.

Question 20 seeks to investigate the comorbidity associated with sleep apnoea symptoms namely high blood pressure, heartburn and etc.

Question 21 asks about medication usage thus checking for comorbidities which the patients may not know they suffer from. The dosage will also give the authors an idea of severity of the conditions in question.

Question 22 and 23 seek to look at lifestyle factors associated with sleep apnoea symptoms.

Question 24 is to see if there is patient interest in this condition.

3.6 Administration

The study commenced after consent had already been obtained from the practices mentioned and a briefing session was held with the receptionist concerned.

Each geographic area was supplied with 150 questionnaires in a box. It was explained to the receptionist that consecutive patients (who had not already participated in this study) coming to the waiting rooms to see the doctor (whether walk-ins or by appointment) should be asked, after the usual greetings by the receptionist: 'Are you 20 years old or above?', if so, the receptionist would hand over the questionnaire with the attached patient information covering letter and ask "Could you please read this before you see the doctor and see whether you wish to participate in this research study." If the patient refused the questionnaire, the receptionist was asked to note the refusal. At the end of every day completed questionnaires would be taken and put away for later analysis while the uncompleted (i.e., refused) questionnaires were returned to the original pile for later handout and the number of refusals were noted.

In this way a total of 304 completed questionnaires were obtained and the number of refusals were noted as 33. The receptionists were each given a present as recompense for their efforts. Thus the response rate was 90.2%.

3.7 Ethical and legal considerations

The protocol was approved by the Human Ethics Committee of the University of the Witwatersrand. Permission had already been obtained for the conduction of such a study at the practices in question from the doctors and the receptionists concerned. The covering letter to the subject (see appendix 1) explained the anonymity and confidentiality of the questionnaires and ensured that there was informed consent.

On completion of the questionnaire the covering letter explained that the questionnaire needed to be placed in a box labeled for such purpose. Information

leaflets about snoring and sleep apnoea symptoms were made available at the reception counter (as explained in the covering letter) and patients were told that they were at liberty to ask their doctor for further information should they wish to do so.

3.8 Resources

Estimates of personnel, equipment and financial resources were minimal and will not be mentioned.

3.9 Limitations of Study

This study looked at the prevalence of snoring and sleep apnoea 'symptoms' in several general practice populations in Johannesburg. It did not attempt to determine the prevalence of obstructive sleep apnoea (OSA) per se. Any attempt to determine the prevalence of OSA in the general population by questionnaire is complicated by the fact that there is no definition of the syndrome upon which there is general agreement and thus to determine the prevalence of OSA itself,[19] we would have to perform an overnight polysomnogram on each member of our sample population.

The questionnaire is thus designed to ask questions pertaining to symptoms most commonly associated with OSA e.g., snoring, daytime sleepiness, restless sleep, waking up gasping and witnessed apnoeas, the presence of which have been shown to have a strong correlation with the co-existence of OSA as shown in several studies.[4,14,19,33]

This study does not tell us about the prevalence of obstructive sleep apnoea (OSA) in South Africa at large, but as the results obtained are from several different ethnic groups, it is hoped that there will be some light shed on the presence or absence of racial differences with respect to sleep apnoea symptoms, and that these results will be representative of urban areas of a similar magnitude.

The population under study may not have been representative of the population at large. This may be due to the fact that people who visit a general practitioner may among other illnesses have been preselected due to the fact that they have a sleep disorder. This would then result in an overestimation of the prevalence of OSA symptoms studied.

By enrolling patients for this study over a short period of time, the possible effects of environmental factors affecting the presentation of patients to their general practitioners may have been enhanced. Further an outbreak of flu at the time of this study may have caused a higher prevalence of certain OSA symptoms, namely, restless sleep, headache, fatigue and body aches. It would have been preferable to carry this study out over a much longer period of time.

Other limitations in data gathering were as follows:

1) Logistics involved in looking at such a large population group forced limiting the sample to selected practices in 3 areas within the greater Johannesburg area thus sampling was not as random as may have been desired. A more random method of selection of practices would have involved compiling a list of all general practices in each ethnic area and then using a random selection method to select one general practice in each area. However, it may be

possible that the doctor or the receptionist may not want to take part and assist in the study and therefore the basis of random sampling would be lost.

2) It was hoped that we would collect 100 completed questionnaires per practice, however we collected 100 from Lenasia South, 68 from Eldorado Park and 136 from Randburg,

3) The possibility exists that a large number of the patients in the practices sampled belonged to medical aids (this is due to the fact that people not belonging to medical aids and of the lower income group, generally cannot afford to be attended to by private general practitioners in South Africa), therefore they may be of the higher income group. The effect of this on the representativeness of the sample can not be determined.

3.10 Analysis techniques

The questionnaires were analyzed at the Medical Research Council by statisticians, Dr. Esther Viljoen and Dr. Piet Becker, using the SASS program. Analysis techniques included the - Students T test, Chi-Squared analysis and Anova (analysis of variance).

Chapter 4

Results

4.1 Introduction

In this chapter the results of the survey will be tabulated. The findings from the analysis will be discussed and expounded in Chapter 5. Difficulties and obstacles in gathering the information were stated under section 3.9 (Limitations).

4.2 General observations and demographic features

The general response rate was 90.2% (304 completed questionnaires and 33 refusals). Apart from the questions 5 and 6 described below, the response rate for each question was more than 80%. Not all questions were answered by all respondents, the number that answered each question has been indicated on the title of the summary table and figures relating to that question in the form of 'n ='.

Question 5 asked about employment; the response rate was 68.5%.

Question 6 pertained to ethnicity and asked a general question about which ethnic group the subject belonged to. The response rate was 77.0%. Considering the possible sensitivity of this type of question, the response rate is not unreasonable.

EDUCATION

The data obtained revealed that the subjects in this study were a relatively educated group of young, employed and skilled adults with a relatively equal gender distribution.

Table 1 – Frequency and break down of school education

n = 299	Frequency	%
Grade 1-11	78	26.1
Grade 12 and above	221	73.9
Tertiary	142	47.4
Total	299	100.0

As can be seen in table 1, it was found that 26.1% (78) had less than grade 12 education, while 73,9% (221) had achieved at least grade 12 education. Of the number who had obtained grade 12 education 64.2% had tertiary education (142 / 221).

EMPLOYMENT

Table 2 - Frequency and breakdown of occupation

n = 300	Frequency	%
Housewife	30	10.0
Student	19	6.3
Pensioner	21	7.0
Unemployed	22	7.3
Employed	208	69.3
Total	300	100.0

Table 2 indicates that 69.3% (208) of the respondents were employed.

Table 3 - Frequency and breakdown of employment

n = 208	Frequency	%
Pension	1	0.5
Unskilled	18	8.7
Skilled	115	55.3
Professional	72	34.6
Self Employed	2	1.0
Total	208	100.0

Referring to tables 2 and 3, it was found that 69,3% (208) were employed. Out of the employed group, 89,9% (187) were in the category 'skilled' and professional.

AGE

Table 4 - Age Distribution

n = 294	Frequency	%
20-30	115	39.1
31-40	89	30.3
41-50	52	17.7
51-60	25	8.5
61-70	8	2.7
71-80	5	1.7
Total	294	100.0

As can be seen in table 4, 87.1% (256) of the subjects were under fifty years of age, and 39.1% (115) were under 31 years of age.

GENDER

There were 44% (132) male vs. 56% (168) female subjects.

ETHNIC GROUP

The table below indicates the breakdown obtained.

Table 5 – Ethnic Group Distribution

n = 234	Frequency	%
White	108	46.2
Black	28	12.0
Coloured	25	10.7
Indian	73	31.2
Total	234	100.0

As can be observed from table 5, 46.2% (108) of the respondents belonged to 'White' ethnic group and 31.2% (73) belonged to 'Indian' ethnic group with an almost equal distribution of 'Black' and 'Coloured' ethnic groups, with a distribution of 12.0% (28) and 10.7% (25) respectively. 23% (70) failed to comment on ethnic group and may have been insulted by this question.

4.3 Analysis of data to meet objectives

General Features Related to Sleeping Problems

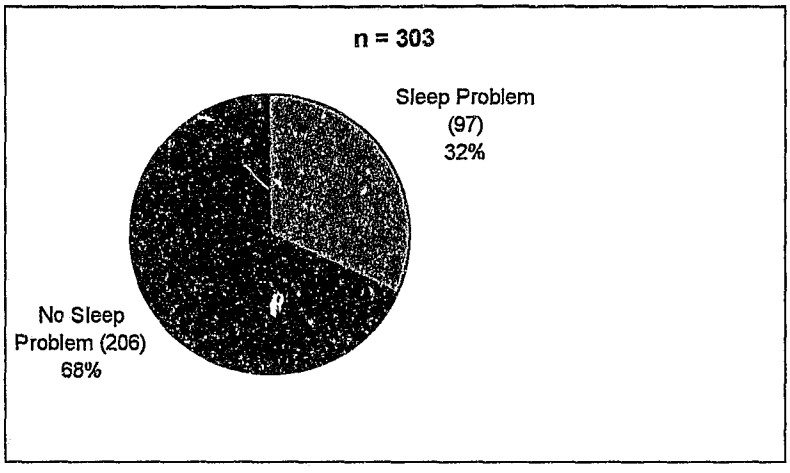


Figure 1 – Frequency of Sleep Problems

According to figure 1, as far as being aware of sleeping problems was concerned, 32.0% (97) of the subjects thought they actually had sleeping problems while 68.0% (206) felt they didn't. However, only 11,8% (35) had previously consulted a doctor about a sleeping problem.

Of the subjects who had sleep problems, the most common sleeping problems specified were insomnia (27.8% or 27 subjects), snoring (24.7% or 24 subjects), restlessness (11.3% or 11 subjects), broken sleep (6.2% or 6 subjects) and witnessed apnoeas (2.1% or 2 subjects). Other sleep problems mentioned were 'blocked throat', 'oversleeping and waking up tired', 'sweating and hot flushes', 'tension' and 'neck problems'.

WAKING UP AT NIGHT

Table 6 – Frequency of Waking Up at Night

n = 297	Frequency	%
Yes	191	64.3
No	106	35.7
Total	297	100.0

Table 7 – Number of Times Waking Up at Night

n = 172	Frequency	%
1	57	33.1
2	50	29.1
3	38	22.1
4 or greater	27	15.7
Total	172	100.0

As can be seen from table 6, 64.3% (191) of patients reported waking at night with 15.7% (27) waking four or more times at night (table 7). The most common reasons for waking up were to go to the toilet or drink water or to eat (53.2% or 104 subjects), for no reason (11.0% or 21 subjects), to tend to children (10.0% or 19 subjects) or getting disturbed (6.8% or 13 subjects).

LIFE STYLE HABITS

With regards to life style habits, smoking and alcohol consumption were the two variables considered in the study. Tables below indicate the results obtained.

Table 8 - Frequency of Smoking

n = 265	Frequency	%
None	170	64.2
1-5	31	11.7
6-10	27	10.2
11-20	27	10.2
21-30	8	3.0
31-40	2	0.8
Total	265	100.0

Table 9 - Frequency of Alcohol Consumption

n = 286	Frequency	%
Yes	148	51.7
No	138	48.3
Total	286	100.0

As can be seen from table 9, 48.3% (138) did not consume alcohol while 51.7% (148) did. The majority of the subjects were non-smokers, as shown in table 8, at 64.2% (170) and only 3.8% (10) smoked more than 20 cigarettes per day. It should be noted that 39 subjects did not respond to question 22, regarding number of cigarettes smoked per day, this is most likely due to the subject being a non smoker and therefore having skipped this question. Although a slot was provided for marking 'none', it is possible that this was not noticed by the subject.

With respect to interest in further knowledge on CSA, 53.6% (178) of the subjects were interested to receive more information (n = 280).

The results of the analysis as they related to the objectives are presented here.

4.3.1 Objective 1: The number of patients with OSA symptoms and snoring.

Excessive daytime sleepiness (EDS) was categorised into normal to mild, moderate, and severe EDS, as defined by an Epworth Sleepiness Scale (ESS) score of 0-9 (normal to mild), 10-11 (Moderate) and 12-16 (severe) respectively.[21] This categorization allowed for the main comparisons of EDS with other variables in this study. In this study EDS was used as one of the main symptoms of OSA. However separate graphs of the comparisons will be shown for EDS to simplify visual presentation. Table 10 shows the distribution of excessive daytime sleepiness in the population studied. The frequency of subjects with ESS score greater than 16 was four (1.6%). As the chi-squared testing requires a minimum cell count of 5 it was decided to exclude the greater than 16 category from the analysis of the main comparisons. The ESS score category of 16 to 24 relates more to narcolepsy and ideopathic hypersomnia.[21]

Table 10 – Summary of Frequency of OSA Symptoms in Respondents

Symptoms of OSA	Percentage
Snoring	48.6% (134), n = 276
Witnessed apnoeas	12.4% (31), n = 250
Restless Sleep	44.6% (120), n = 269
Waking up Gasping	11.8% (30), n = 254
Excessive Day Sleepiness	Normal/mild 77.1% (189), Mod. 9.8% (24), Severe 13.1% (32), n = 245
Morning Headache	35.1% (93), n = 265
Dry Mouth on Waking	45.1% (123), n = 273
Tiredness on Waking	63.3% (176), n = 278
Body Aches and Pains	42.5% (114), n = 268

As can be noted from table 10, 77.1% (189) had normal to mild excessive daytime sleepiness, 9.8% (24) had moderate excessive daytime sleepiness and 13.1% (32) had severe excessive daytime sleepiness. The breakdown furthermore revealed that 3.4% (9) were likely or very likely to fall asleep in a car while stopped for a few minutes in the traffic as shown in table 11. This category was specifically noted due to its public health and safety implications, pointing further to the importance of diagnosis of OSA.

Table 11 - Likelihood of Falling Asleep in a Car While Stopped for a Few Minutes in the Traffic

n = 266	Frequency	%
Never	232	87.2
Seldom	25	9.4
Likely	5	1.9
Very Likely	4	1.5
Total	266	100.0

Table 12 – Increase in Frequency of Symptoms of OSA Over a 5 Year Period in Individual Patients.

Symptoms	Frequency five years ago	Current prevalence	% Change
Snoring	43.9% (119), n = 271	48.6% (134), n =276	10.7%
Witnessed apnoeas	9.6% (24), n = 251	12.4% (31), n = 250	29.2%
Restless Sleep	31.4% (81) n = 258	44.6% (120), 269	42.0%
Waking up Gasping	9.6% (24), n = 249	11.8% (30), n = 254	22.9%

As can be seen from table 12, the four significant symptoms of OSA considered in this study were all found to increase in frequency over a five year period with restless sleep having the highest percentage change of 42.0%.

4.3.2 Objective 2: The relationship of demographic variables and OSA symptoms.

The demographic variables considered were age, sex and ethnic group. A presentation of OSA symptoms versus age is shown in table 13. Certain age categories were combined (51 to 60, 61 to 70, 71 to 80) into a greater than 50 age category due to cell counts being less than 5 which would have rendered chi-squared analysis invalid.

Table 13 – Age vs. OSA Symptoms

	n	20-30	31-40	41-50	> 50	P, *=significant
Snoring	268	34.3% (36/105)	46.3% (38/82)	61.7% (29/47)	79.4% (27/34)	0.000 *
Apnoeas	243	5.2% (5/97)	13.5% (10/74)	14.0% (6/43)	31.0% (9/29)	0.003 *
Restless sleep	260	36.2% (38/105)	44.9% (35/78)	48.9% (22/45)	59.4% (19/32)	0.105
Wakingup gasping	248	10.1% (10/99)	10.4% (8/77)	13.6% (6/44)	21.4% (6/28)	0.396
Headache	257	30.4% (31/102)	32.5% (25/77)	40.9% (18/44)	38.9% (13/34)	0.597
Dry mouth	265	42.9% (45/105)	34.2% (27/79)	55.3% (26/47)	55.9% (19/34)	0.057
Tiredness	269	64.5% (69/107)	59.5% (47/79)	60.4% (29/48)	65.7% (23/35)	0.867
Body Aches	259	42.2% (43/102)	34.2% (26/76)	35.6% (16/45)	63.9% (23/36)	0.021

Table 13 shows percentage of subjects in each age group who responded positively to the particular OSA symptom. Next to each of these percentages, the number within each age group who responded positively is shown as a fraction of the total number within that age group. As is seen in table 13, there was a significant association between age and snoring and age and witnessed apnoeas. Pair wise comparison revealed that snoring prevalence in the 20-30 category was significantly different to the 41-50 category as well as to the greater than 50 category, further it was noted that the 31-40 category was significantly different to the greater than 50 category.

Pair wise comparison conducted in respect of alence of witnessed apnoeas revealed that the 20-30 category was significantly different to the 31-40 category as well as to the greater than 50 category. The 31-40 category was significantly different to the greater than 50 category.

No significant differences were noted between age and the other symptoms.

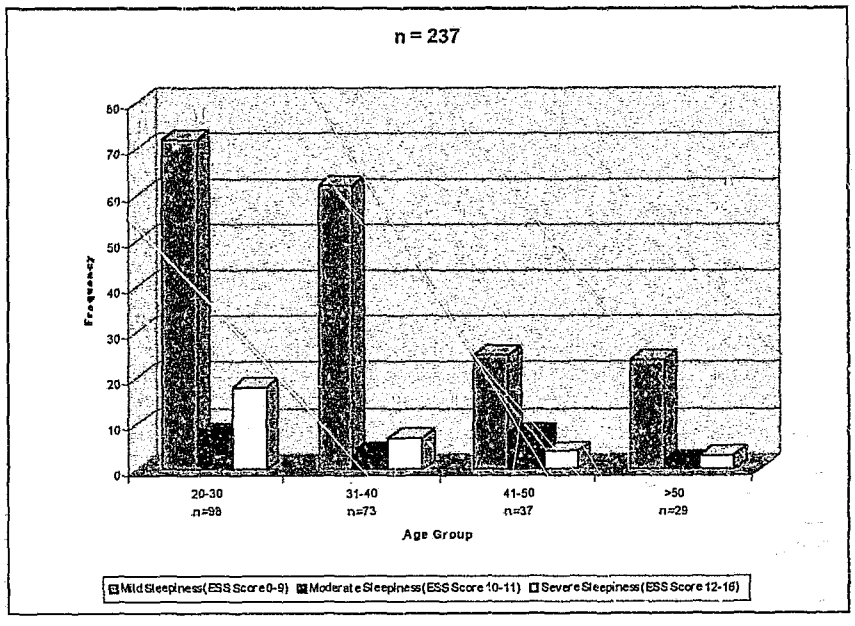
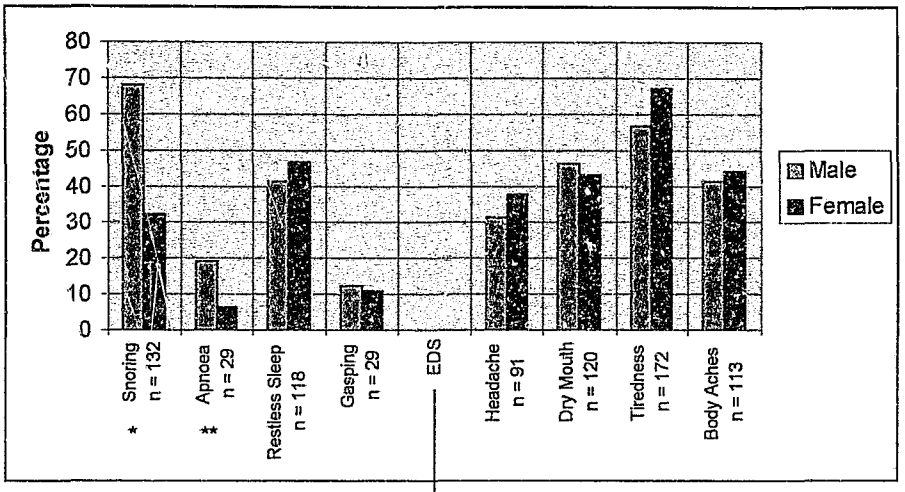


Figure 2 – Frequency of EDS According to Age

Figure 2 shows the frequency of EDS according to age. There were no significant relationship noted between the different age groups with respect to EDS. It is interesting to note the relatively high frequency of severe excessive day time sleepiness in the 20-30 age group.

The relationship of gender and OSA symptoms: A graph of gender versus OSA symptoms is shown in figure 3.



* Significant p = 0.000, ** Significant p = 0.003

Figure 3 – OSA Symptoms According to Gender

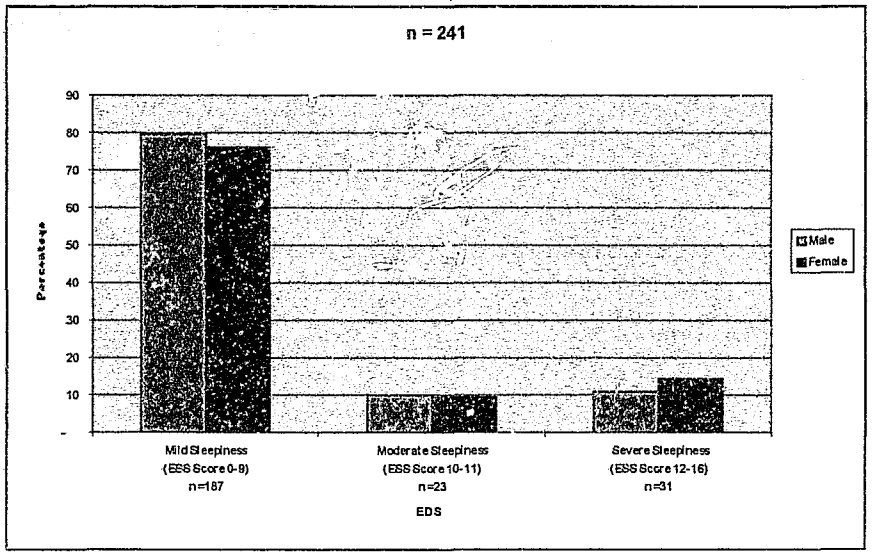


Figure 4 –EDS Symptoms According to Gender

Pearson Chi-Squared test results indicate that men have a significant increase in snoring and witnessed apnoeas when compared with women. As can be seen in figure 3, it was found that 68.0% (83 of 122) of men reported snoring whereas only 32.5% (49 of 151) of women reported snoring. It should be noted, however, that of the people who snored 62.9% (83 of 132) were male and 37.1% (49 of 132) were female. Of the men 18.9% (20 of 106) reported apnoeas whereas only 6.4% (9 of 140) of women reported apnoeas. Of the total who reported witnessed apnoeas 68.9% (20 out of 29) were male and 31.0% (9 of 29) were female. Figure 4 shows the EDS symptoms according to gender. No statistically significant relationship were noted in EDS with respect to gender. Although no statistically significant differences were noted in the other symptomatology associated with OSA, it appears clinically significant that of the subjects reporting restless sleep, headache, tiredness and body aches nearly 60.0% were women and nearly 40.0% were men.

The relationship of OSA symptoms according to ethnic group: Below are ethnic groups variances with respect to different symptoms of OSA.

Table 14 – OSA Symptoms According to Ethnic Group

	N	White	Black	Coloured	Indian	P, *=Significant
Snoring	109	52.9% (55/104)	41.7% (10/24)	30.0% (6/20)	54.3% (38/70)	0.193
Apnoea	28	17.2% (17/99)	4.3% (1/23)	5.9% (1/17)	14.8% (9/61)	0.312
Restless Sleep	87	37.0% (37/100)	44.0% (11/25)	45.0% (9/20)	46.2% (30/65)	0.664
Gasping	23	12.0% (12/100)	8.7% (2/23)	11.1% (2/18)	11.7% (7/60)	0.976
Head Ache	73	32.1% (34/106)	38.5% (10/26)	42.1% (8/19)	34.4% (21/61)	0.815
Dry Mouth	97	48.1% (51/106)	33.3% (9/27)	30.0% (6/20)	47.0% (31/66)	0.285
Tiredness	134	60.4% (64/106)	74.4% (19/27)	60.0% (12/20)	59.1% (39/66)	0.772
Body Aches	93	32.1% (34/106)	48.0% (12/25)	55.0% (11/20)	58.1% (36/62)	0.007 *

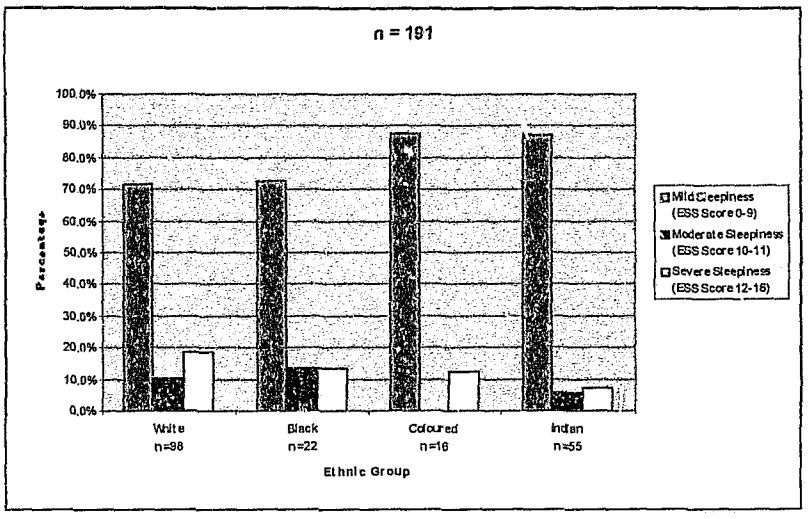


Figure 5 – EDS According to Ethnic Group

Table 14 shows OSA symptoms according to ethnic groups, in order to understand the table it is important to note that it relates to the percentage within the ethnic group who reported the symptom, e.g. 52.9% (55 of 104) Whites reported snoring whereas of the people who snored 50.5% (55 of 109) were White. Chi-Squared analysis revealed that significant association existed between ethnicity and body aches and pains ($p < 0.05$). Pairwise comparisons were conducted to assess the nature of the significant differences and it was found that the most significant difference were between Whites and Indians (32.1% (34 of 106) and 58.1% (36 of 62) respectively) and Whites and Coloureds (32.1% (34 of 106) and 55.0% (11 of 20) respectively).

Figure 5 shows the frequency of EDS according to ethnic group. No significant relationship was noted between the ethnic groups with respect to EDS.

4.3.3 Objective 3: The link between body mass index and OSA symptoms.

Individual parameters of height and weight of each subject were used to calculate the body mass index (BMI). The mean body mass index (BMI) for this population in this study was found to be 25.49 kg/m^2 with standard deviation of 6.1. The BMI distribution results of this study were as follows:

Table 15 – Body Mass Index Frequency

	Weight	Height	BMI
n	286	257	251
Mean	69.94 kg	167.70 cms	25.49 kg/m^2
Std. Error of Mean	0.96 kg	0.85 cms	0.38 kg/m^2
Std. Deviation	16.16 kg	13.66 cms	6.10 kg/m^2

Table 15 represents the frequency of data obtained with respect to body mass index, namely weight and height. Body mass index could only be calculated on 251 subjects as these were the only ones that supplied both parameters needed. The mean body mass index was found to be 25.49 kg/m^2 for the study population.

Anova tests performed for snoring and for witnessed apnoeas indicated a significant positive correlation ($P < 0.01$ and $P < 0.005$ respectively) for these variables with BMI. No significant correlation was found for BMI and any of the other variables of OSA symptoms.

When the data of obese (BMI > 25 kg/m^2) subjects only was subjected to t-test, it was noted that there was a significant positive relationship between BMI and 'waking up with dry mouth' ($t = 0.0193$); no significant relationship was noted for

the other symptoms of OSA vs. BMI for obese patients. In this study, it was found that 48% (82 of 172) of obese patients (BMI > 25 kg/m^2) reported snoring.

4.3.4 Objective 4: the familial association of OSA symptoms.

There was a 56.6% (158 of 279) prevalence of a positive family history of snoring in the sample studied, 14.5% (36 of 248) for a family history of witnessed apnoeas, 28.2% (73 of 259) for a family history of restless sleep and 11.6% (29 of 249) for a positive family history of gasping.

Chi-Squared analysis revealed a significant correlation between the four major symptoms and family history of snoring, witnessed apnoeas, restless sleep and gasping as per table 16 below.

Table 16 – Positive Family History of OSA Symptom in the Presence of that Symptom in the Patients Studied

Symptom	n	Positive Family History of each Symptom	P
Snoring	131	70.2 % (92)	0.000 (<0.005)
Witnessed apnoeas	29	34.5% (10)	0.000 (<0.005)
Restless Sleep	109	39.4% (43)	0.000 (<0.005)
Gasping	26	42.3% (11)	0.000 (<0.005)

A positive family history of snoring was found in 70.2% (92 of 131) of the patients who snored, 80.0% (24 of 30) of patients with witnessed apnoeas, 63.4%

(71 of 112) of patients with restless sleep and 71.4% (20 of 28) of patients who complained of gasping.

4.3.5 Objective 5: The association of OSA symptoms with co-morbidities such as hypertension, heartburn, depression, angina, stroke and heart failure.

COMORBIDITIES ASSOCIATED WITH OSA

Below (table 17) are the prevalence rates for the comorbidities associated with OSA in this study.

Table 17-Frequency of Patients with OSA Comorbidities

	n	Frequency	%
Heartburn or Reflux	275	76	27.6
Depression	270	65	24.1
High Blood Pressure	277	59	21.3
Angina	269	8	3.0
Heart Failure	268	6	2.2
Stroke	270	1	0.4

As can be seen in table 17, the most prevalent associated comorbidities reported were heartburn or reflux at 27.6% (76 of 275), depression at 24.1% (65 of 270) and high blood pressure at 21.3% (59 of 277).

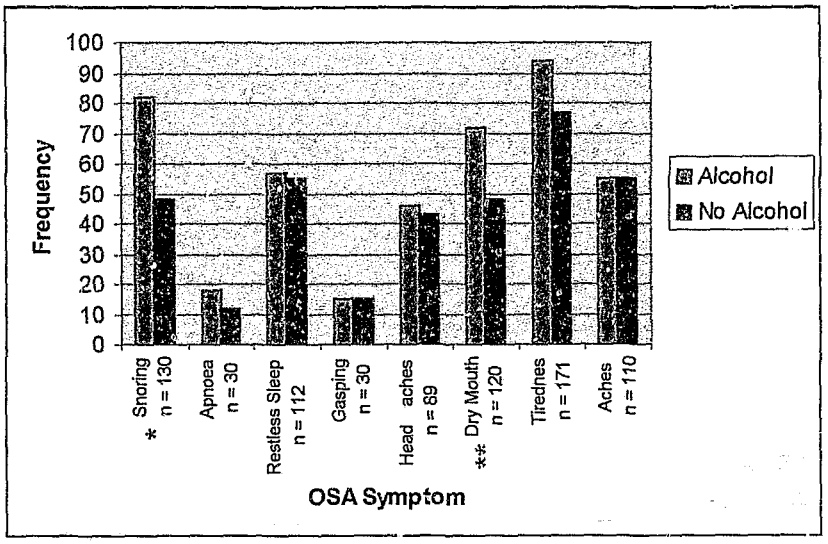
Table 18 – The Association of OSA symptoms to Comorbidities

	OSA SYMPTOMS							
OSA Co-morbidity	**Snoring**	**No Snoring**	**Witnessed apnoeas**	**No Witnessed apnoeas**	**Restless Sleep**	**No Restless Sleep**	**Gasping**	**No Gasping**
H/T	28.8% (36/125) *p=0.004	15.2% (15/99)	27.6% (8/29)	20.0% (33/165)	21.9% (23/105)	20.0% (24/120)	37.0% (10/27)	18.4% (34/185)
Heartburn	36.8% (46/125) *p=0.002	15.3% (15/98)	44.8% (13/29)	24.2% (40/165)	32.1% (34/106)	22.7% (27/119)	42.9% (12/28)	27.6% (51/185)
Depression	26.6% (22/124)	17.5% (17/97)	39.3% (11/28) *p=0.047	19.5% (32/164)	34.0% (35/103) *p=0.003	14.4% (17/118)	48.1% (13/27) *p=0.000	17.5% (32/183)
Angina	4.9% (6/122)	2.0% (2/98)	7.1% (2/28)	2.4% (4/164)	3.9% (4/102)	2.5% (3/119)	4.0% (1/25)	2.7% (5/184)
Stroke	0.8% (1/123)	0.0% (0/98)	0.0% (0/28)	0.6% (1/165)	0.0% (0/103)	0.8% (1/119)	0.0% (0/26)	0.5% (1/184)
CCF	4.0% (5/124)	1.0% (1/96)	7.1% (2/28)	1.8% (3/163)	2.9% (3/103)	2.5% (3/118)	3.8% (1/26)	1.6% (3/182)

Table 18 shows the association of OSA symptoms to comorbidities. The table indicates the prevalence of the comorbidity within each symptom group, e.g. 28.8% (36 of 125) of the subjects who snored reported hypertension, whereas only 15.2% (15 of 99) of those who did not snore reported hypertension. There was a significant difference noted in the prevalence of hypertension and heartburn in patients who snored vs. those who did not snore ($p<0.005$ for hypertension and $p < 0.005$ for heartburn). It was further noted that witnessed apnoeas, gasping and restless sleep were positively correlated with depression ($p < 0.05$ for witnessed apnoeas, $p < 0.001$ for gasping and $p < 0.005$ for restless sleep).

In this study, of the hypertensive patients it was noted that 66.7% (36 of 54) reported snoring as a symptom, which was statistically significant compared to those who did not snore, 27.8% (15 of 54) ($p < 0.05$).

4.3.6 Objective 6: The association of life style factors and OSA symptoms.



* Significant P = 0.000, ** Significant P = 0.033

Figure 6 – Frequency of Alcohol Consumption According to OSA Symptoms

As can be seen in figure 6, there was a statistically significant increase in snoring ($p = 0.000$) and waking up with a dry mouth ($p = 0.033$) in the alcohol consuming patients as compared with the non alcohol consuming patients. No other significant differences were noted with respect to the other symptoms.

No significant difference was noted in alcohol consumption with respect to EDS.

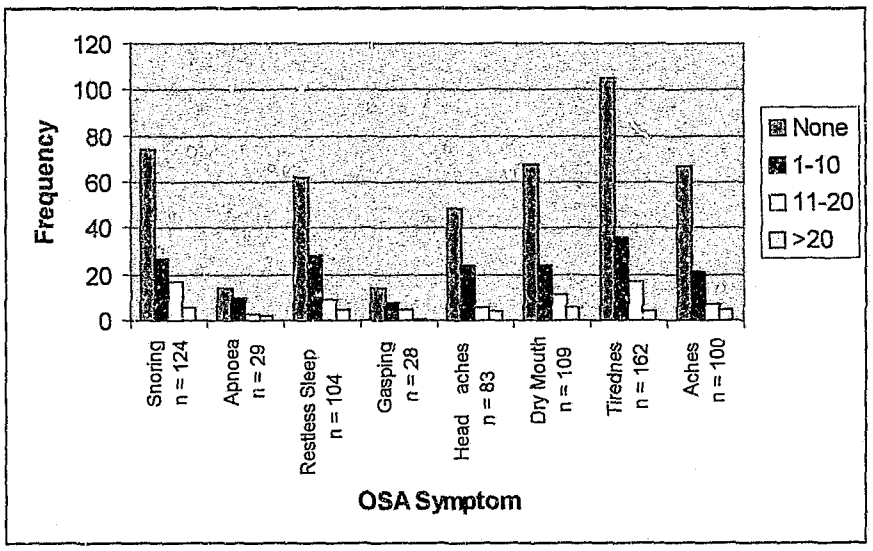


Figure 7 – Frequency of Smoking According to OSA Symptoms

Figure 7 represents the frequency of smoking according to OSA symptoms. With respect to smoking, there were no statistically significant differences noted in OSA symptoms. These observations were checked with the Fisher's Exact Test and again no significant findings were noted.

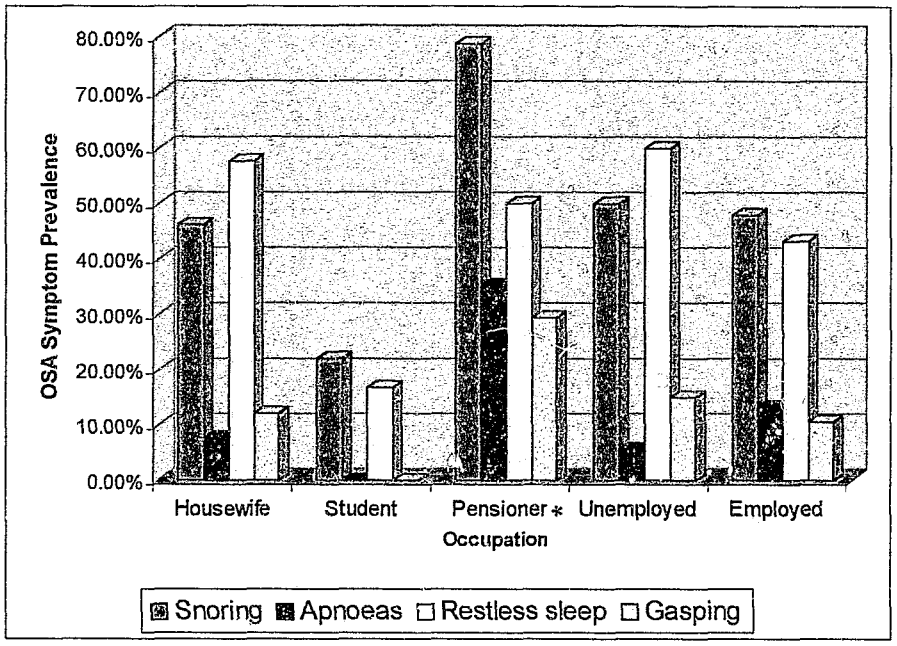
No statistically significant differences were noted between smokers and non smokers with respect to EDS.

4.3.7 Objective 7: The association of Socio-Economic and Education factors with OSA symptoms.

Table 19 – Education Level and Witnessed Apnoeas

	Education Level			
	Grade 1-12	n	Tertiary	n
Witnessed apnoea	33.0%	10	67.0%	20
No Witnessed apnoea	55.1%	119	44.9%	97

A correlation between education level and the presence of witnessed apnoeas was noted; as education level increased, the level of witnessed apnoeas also increased ($p < 0.05$).. There was no significant difference noted in the level of education with respect to snoring or the other OSA symptoms.



* Significant ($p<0.05$)

Figure 8 – Prevalence of OSA Symptoms According to Occupation

Table 20 – Prevalence of OSA Symptoms According to Occupation

	Housewife	Student	Pensioner	Unemployed	Employed
Snoring	46.4% (13/28)	22.2% (4/18)	78.9% (15/19)	50.0% (10/20)	47.9% (90/188)
Apnoeas	7.7% (2/26)	0.0% (0/18)	35.3% (6/17)	5.6% (1/18)	13.1% (22/168)
Restless sleep	57.7% (15/26)	16.7% (3/18)	50.0% (9/18)	60.0% (12/20)	43.2% (80/185)
Gasping	12.0% (3/25)	0.0% (0/18)	29.4% (5/17)	15.0% (3/20)	10.5% (18/171)

Figure 8 and Table 20 represent the prevalence of OSA symptoms within various occupational groups. Of the housewives 46.4% (13 of 28) reported snoring. There was a statistically significant increase in the prevalence of witnessed apnoeas in pensioners compared with the other occupation groups. There were no other significant differences.

Further comparisons were done for OSA symptoms vs. EDS:

Table 21 – Prevalence of OSA Symptoms According to EDS

	EDS		
	Mild (ESS Score 0-9) n = 189	Moderate (ESS Score 10-11) n = 24	Severe (ESS score 12-16) n = 32
Snoring	44.5%	56.5%	48.4%
Witnessed apnoeas	7.8% *	14.3%	26.7% *
Restless Sleep	41 5%	40.9%	40.0%
Gasping	9.9%	14.3%	16.7%
Head Aches	31.3%	45.5%	35.5%
Dry Mouth	38.5%	56.5%	50.0%
Tiredness	55.1% **	73.9%	78.1% **
Aches	40.1%	47.8%	45.2%

*Significant $p<0.01$, ** significant $p < 0.05$

According to table 21, with increasing levels of excessive daytime sleepiness, there was a significant increase in the prevalence of both witnessed apnoeas ($p < 0.01$) and tiredness ($p < 0.05$). Pairwise comparisons further revealed that there was a significant difference in the prevalence of witnessed apnoeas and tiredness for mild versus severe EDS.

Chapter 5

Discussion, Conclusion and Recommendations

5.1 Discussion

Several important findings emerged from this study.

OSA symptoms are currently highly prevalent in urban environments in South Africa. Snoring had a prevalence rate of 48.6% in the population studied. There was a high prevalence rate in the population studied for restless sleep (44.6%), tiredness on waking (63.3%), dry mouth on waking (45.1%) and waking with body aches and pains (42.5%). The prevalence rate in the population studied for witnessed apnoeas was 12.4%, waking up gasping was 11.8%, morning headaches 35.1% and moderate and severe excessive daytime sleepiness categories were 9.8% and 13.1% respectively. The prevalence of snoring, witnessed apnoeas, restless sleep and gasping had increased over the previous five year period.

A relatively high percentage (32.0%) of subjects reported that they considered that they actually had a sleeping problem. The most common problem reported in the group who reported having a sleep problem was insomnia (27.8%), snoring (24.7%), restlessness (11.3%), broken sleep (6.2%) and witnessed apnoeas (2.1%). A total of 37.8% of the subjects woke up more than 3 times per night. The most common reasons for waking up were found to be either to go to the toilet or drink water or to eat (53.2%), for no reason (11.0%), to tend to children (10.0%) or getting

disturbed (6.8%). Only 11.8% of the subjects had previously consulted a doctor about a sleep problem.

Although OSA symptoms are present in both sexes there is a statistically higher prevalence in male gender for snoring (68.0% vs. 32.5%) and witnessed apnoeas (18.9% vs. 6.4%). Of those who snored 62.9% were male and 37.1% were female.

Some of the symptoms of OSA were found to increase with increasing age, BMI and alcohol consumption. Snoring and witnessed apnoeas were found to increase in prevalence with increasing age; snoring and witnessed apnoeas with BMI; and snoring and waking up with a dry mouth with alcohol consumption. No relationship was found between smoking and OSA symptoms.

There were no significant differences between the ethnic groups for the major obstructive sleep apnoea symptoms. However, Indians and Coloureds were noted to have significantly higher prevalence rates of body aches and pains compared to Whites.

There was a significant difference in family history in patients with and without OSA symptoms. The results revealed a significant correlation for all four major symptoms and family history of each symptom. It was further found that 70.2% of the patients who reported snoring and 80.0% of patients who reported witnessed apnoeas had a positive family history of snorin

Depression was positively correlated with witnessed apnoeas, gasping and restless sleep. Hypertension and heartburn were positively correlated with a snoring history.

A significant positive relationship was noted between increased education level and witnessed apnoeas as well as being a pensioner and witnessed apnoeas.

This study looked at the prevalence of snoring and sleep apnoea symptoms in several general practice populations in Johannesburg. It did not attempt to determine the prevalence of obstructive sleep apnoea per se. Any attempt to determine the prevalence of OSA in the general population by questionnaire is complicated by the fact that there is no definition of the syndrome upon which there is general agreement and the inability to accurately predict the level of sleep apnoea from the symptoms only. Thus to determine the prevalence of OSA itself, we would have to perform an overnight polysomnogram on each member of our sample population. Nonetheless the prevalence rates obtained for the symptoms of OSA in this study compare well with overseas studies cited.[11,19]

Perhaps most importantly the small number of practices (5) used as well as their geographical location makes one cautious about generalising the findings; although attempt was made to make the study subjects as socio-demographically diverse as possible, due to the survey population being mostly white, with a high degree of tertiary education and employment rate compared to the general population, as well as the fact that by its very nature, the poor and unemployed would generally not be able to afford private general practitioner visits, the survey may not provide an accurate estimate of the prevalence of OSA symptoms in the whole of South African primary care practice. Nevertheless the high prevalence

rate of OSA symptoms found in this study is remarkable and can lead to further research about the prevalence of OSA itself in South Africa.

One of the aims of the study was to obtain results from several different ethnic groups and to shed light on the presence or absence of racial differences with respect to sleep apnoea symptoms. It was hoped that 100 completed questionnaires per practice would be collected that would result in roughly equal racial distribution. However 100 was collected from Lenasia South (mainly Indian base), 68 from Eldorado Park (mainly Black and Coloured base) and 136 from Randburg (mainly White base). Of the 304 completed questionnaires obtained only 234 answered the ethnic group question and of those 46.2% were White, 31.2% were Indian, 12.0% were Black and 10.7% were Coloured. Due to the small sample size of the Black and Coloured race groups, the results can not easily be generalised to these population groups. Further studies addressing different race groups may be justified.

The study sample was a convenient sample of patients attending several general practices in the greater Johannesburg area. As such the prevalence rates reported here may over estimate community prevalence; however, they do suggest that symptoms of OSA are prevalent in the population studied and that further study may be warranted. It will be important to perform large-scale community-based epidemiological studies to determine age and sex specific prevalence rates now that the symptoms are found to be so prevalent in a general practice set up.

The data reported in this study are consistent with previous international studies demonstrating that OSA occurs more commonly in older males[1,16,17,29], but that unlike the literature, in the local context, prevalence rates were higher in the White and Indian race groups. There were high prevalence rates found for restless sleep, tiredness on waking, drymouth on waking and body aches and pains and future

studies will need to focus on the reasons for such high prevalence rates as the economic implications of reduced productivity associated with tiredness and the other symptoms mentioned above seems alarming. While 77.1% of the study population fell in the category of normal to mild EDS (ESS score of 0-9) as much as 9.8% and 13.1% fell under the categories of moderate and severe EDS (ESS score of 10-16). This too requires future studies ascertaining reasons and management solutions for such apparently high rates of sleepiness found in this study.

Table 22 – Comparison of Results to USA Community Sample

	USA community sample[19]		**This study**	
	Male	Female	Male	Female
Snoring disturbing others	68.0%	50.0%	68.0%	32.5%
Loud snoring	34.0%	22.0%		
Apnoeas	4.0%	2.0%	18.9%	6.4%
Gasping	17.0%	6.0%	12.1%	11.1%
EDS (moderate and severe)	20.0%	17.0%	20.6%	23.8%

Table 23 – Comparison of Results to Other International Studies

	International studies[11,22]		**This study**	
	Male	Female	Male	Female
Snoring[11]	51.6%	33.0%	68.0%	32.5%
Apnoeas[11]	33.4%	12.6%	18.9%	6.4%
Snoring[22]	25.0%	15.0%	68.0%	32.5%

Tables 22 and 23 present a number of comparative data from international studies. Although the prevalence rates vary considerably from study to study, especially with the rate of apnoeas (justifying further studies), it can be seen that 68% of males and 50% of females reported snoring disturbing others in a USA community

sample compared to this study where 68% of males and 32.5% of females reported snoring.

In the analysis of the prevalence of EDS it was noted that as many as 3.4% of the research population were likely or very likely to fall asleep in a car while stopped for a few minutes in the traffic. This has alarming safety implications at a national level.

The four significant symptoms of OSA considered in this study were all found to increase in prevalence over a five year period. This is corroborated in a study by Pendlebury in which the natural evolution of moderate sleep apnoea was studied.[17] The results showed that a significant increase in AHI occurred in a group of patients with mild to moderate OSA over a mean of 17 months which was not related to an increase in BMI. This is further confirmed by two other studies cited by Pendlebury.[17] Unfortunately in this study, BMI progression over a five year period could not be assessed in order to ascertain whether the apparent increase in reported OSA symptoms was related to a parallel rise of BMI. Such data would also have been impossible to collect in this study as patients would probably have not known accurately what their weights were five years ago. It is speculated by Pendlebury that the mechanism of progression of OSA in the absence of weight gain may involve several functional factors such as ventilatory instability or snoring induced trauma to the upper airway.[17]

Looking at the progression of OSA symptoms in this study, it is interesting to note that the greatest increase over the five year period was related to restless sleep (42.0% change in five years). A growing body of data suggests that poor overall health and quality of life are consequences of untreated OSA.[19] Restless sleep may have increased as a consequence of untreated OSA and this may indirectly explain the large increase. Other reasons for the higher increasing rates of restless

sleep reported, may include increased number of children, increased number of jobs held, shift work and psychological stresses such as increased expense of living and greater commitments.

There was a significant relationship between age and snoring and age and witnessed apnoeas. According to the literature age was a significant risk factor for OSA[27] and increased prevalence of OSA and snoring was associated with increasing age. These findings were confirmed in this study, where increasing age was associated with increasing prevalence of certain symptoms, but significant differences were noted for witnessed apnoeas and snoring only as mentioned.

Statistical analysis revealed significant differences between males and females with respect to snoring and witnessed apnoeas with men higher than women. This is as expected from the literature.[1,11,17,27] Although no statistically significant differences were noted in men and women with respect to EDS, it appears clinically significant that excessive daytime sleepiness appears higher in females than in males. Future studies for this observation is warranted.

The ratio of snoring in men vs. women was equal in this study with that found by Kingman et al, who reported male – female ratio of approximately 2:1 to 3:1 in the general population.[1] In this study the ratio was found to be 2:1 (68.0% of men and 32.5% of women reported snoring in this study).

Although no statistically significant differences were noted in the other symptomatology associated with OSA, it appears clinically significant that 60.0% of women versus nearly 40.0% of men reported restless sleep, headache, tiredness and body aches. This is cause for concern and future studies elucidating the reasons for such apparently high prevalence rates in both sexes as well as the disproportionately high prevalence rates of the above symptoms in the female

group need to be undertaken. If as many as 40.0% of the adult male population and 60.0% of adult female population actually experience the above reported symptoms, there are alarming clinical and economic implications, to say the least.

The results revealed that significant association existed between various ethnic groups with respect to body aches and pains only. This association was most significant in Indians and Coloureds (58.1% and 55.0% respectively) as compared to Whites (32.1%). No significant relationship was noted between ethnicity and EDS. That there was no significant association between ethnicity and the major symptoms of OSA (snoring, gasping, witnessed apnoeas, restless sleep and excessive daytime sleepiness) was unexpected. The international literature indicate a higher prevalence of age adjusted OSA in Blacks compared to Whites,[1,3,14] and of OSA in Mexican-Americans and Pacific Islanders (non Caucasians) compared to Caucasians. Although body aches and pains was found to be associated with ethnicity it would appear that the reason for the body aches and pains in certain populations is not necessarily a consequence of OSA symptoms, as the major symptoms of OSA in the different ethnic groups indicate (i.e., no significant association between ethnicity and the major symptoms of OSA). It may be speculated that the association found in this study between body aches and pains and ethnicity (Coloured and Indian race groups) may possibly be explained on the basis of factors other than OSA, possibly economic, social and life style conditions.

Body mass index (BMI) is a convenient method of defining obesity for both clinical and epidemiological purposes.[34] Obesity as defined, by a body mass index higher than 25 kg/m^2, has a prevalence of 36% generally in men and 26% in women in the literature.[35] When defined by a body mass index higher than 30, it is 6% in both sexes.[35] A BMI of 28 corresponds to a weight 20% above the ideal body weight.[34] In this study the mean body mass index was 25.49 kg/m^2 with a

median of 24.8 kg/m^2, pointing to the fact that nearly half of the study population fell in the obese range. Anova tests performed for snoring and body mass index and for witnessed apnoeas and body mass index indicated a significant positive correlation, ie with increased BMI there was a significant increase in snoring and witnessed apnoeas. This implies that people with a high BMI are more likely to have OSA and may require further screening.. This is corroborated by Kingman et al who show that obesity as defined by BMI > 28 was present in 60% to 90% of OSA patients evaluated at sleep clinics.

It is further corroborated by Shinohara et al, that 60% of obese subjects were found to exhibit OSA.[29] As fat accumulation around the neck, reduction of lung volume and the structural alteration of pharyngeal muscle fibres have been considered causes for the narrowing of the upper airway, a major cause of OSA[29], it follows logically that an association between BMI and snoring was noted in this study. The association between BMI and dry mouth may be due to the presence of OSA, diabetes or the metabolic syndrome in patients with increased BMI.

Several studies have stressed the importance of rather measuring the neck circumference index (NCI)[27] or the waist-hip ratio,[1] and have found that these measurements had a much better correlation with OSA than BMI. However, BMI was used in this study as a method of defining obesity[34] due to the convenience of not needing cumbersome measuring instruments and thus allowing for the use of a self administered questionnaire as a measuring instrument and the limitation was taken note of.

Of the patients who snored, 70.2% had a positive family history of snoring while of the patients who had witnessed apnoeas as many as 80% had a positive family history of snoring. Patients with restless sleep and gasping had 63.4% and 71.4% positive family history of snoring respectively. In this study there was a

significant difference in family history in patients with and without OSA symptoms, with snoring having the highest familial association (70.2% of the snorers had a positive family history of snoring). As snoring is a major symptom of OSA,[1,3,14,19] the implications of managing patients in general practice with a positive family history of snoring are noteworthy. This is as expected from the literature, where three authors unequivocally point out familial and genetic risk as having a significant association with OSA.[1,3,14] As previously mentioned such familial risk may be the result of similarities in facial structure affecting upper airway dynamic in sleep.[14] Estimates are that 40% to 50% of the variance in apnoeic activity can be attributed to familial factors.[1] While significant, the overall magnitude of the relationship between familial and genetic factors and OSA does not appear to be sufficiently strong to justify screening of asymptomatic family members, but probably justifies screening of family members of patients diagnosed with OSA that report snoring.

With regards to associated comorbidities, the prevalence of witnessed apnoeas, gasping and restless sleep were positively correlated with depression. This may be due to a cause or effect phenomenon. Patients who are depressed may be using sedative medications or sleep promoting hypnotic medications which can directly affect apnoea expression and sleep pattern.[6] Alternatively, it may be due to the fact that patients with apnoeas sleep poorly and the latter may result in secondary depression.[11]

There was also a significant increase in the prevalence of hypertension as well as heartburn in patients who had a snoring history. This confirms previous studies where a significant relationship was found between hypertension and OSA.[1,3,6,31] Prevalence of day time hypertension in patients with OSA has been reported to exceed 40%.[3,6] In this study the prevalence of hypertension in patients who reported the symptom of waking up gasping, a significant predictive symptom of

OSA,[27] was found to be 37%, thus nearly matching prevalence rates reported in the literature.[6]

The significant association of heartburn in patients with snoring may be explained on the basis of a common denominator, namely obesity, being associated with both snoring and reflux oesophagitis, due mainly to increased pharyngeal fatty tissue and higher abdominal pressure respectively. Heartburn may also be explained by the presence of negative pressure produced in the thorax during witnessed apnoeas.

There was a statistically significant positive correlation between alcohol consumption and snoring and alcohol consumption and waking up with a dry mouth. It should be noted that as alcohol preferentially suppresses the activity of upper airway dilator muscles in comparison to the diaphragm, and therefore has been shown to increase upper airway resistance,[3] it may be associated with snoring, as found in the present study. No other significant relationships were noted between alcohol and other OSA symptoms.

That there was no association between alcohol consumption and witnessed apnoeas in this study was unexpected as the literature points out several studies in which alcohol use has been significantly associated with apnoeas.[1,3,14] The differences in this study and those cited in the literature, although not obvious could relate to the level of alcohol use in the various studies or to the variability in the categorization of alcohol consumption. The statistically significant association of alcohol consumption in patients reporting dry mouth in the morning may be explained by several factors, namely the presence of diabetes, diuresis secondary to alcohol, the coexistence of obesity and OSA resulting in snoring, witnessed apnoeas and therefore a dry mouth, or as part of a hangover.

With respect to smoking no statistically significant relationships were noted with OSA symptoms (including EDS). This was unexpected as smoking has been linked to snoring possibly by producing pharyngeal inflammation.[1] Although statistically not significant, the numbers do appear to be of clinical significance as it was observed that in the 11-20 cigarettes category, 13.7% were snorers compared to 6.3% that did not snore. This may be significant when considering a larger number of patients.

A significant statistical relationship was noted between increased education level and presence of witnessed apnoeas.

There was also a statistically significant relationship between occupation and witnessed apnoeas with the highest prevalence of witnessed apnoeas noted in pensioners at 35.3%. As pensioners are by definition greater than 60 years old, this may be an age related effect and not related to occupation. As has been previously shown, significant factors affecting this may be related to increased age, male gender or obesity with increased BMI.

This study has remodeled my practice as a family physician. It has made me consider the possibility that with these prevalence rates locally, every patient needs to be screened for OSA symptoms in order not to overlook this often missed diagnosis. This study has laid the path for several new studies to be undertaken by postgraduates with a special interest in sleep disorders.

5.2 Conclusion

It appears from this study, undertaken for the first time in South Africa to examine the prevalence of snoring and OSA symptoms, that these symptoms are highly prevalent and have similar significant associations to those cited in the literature.

There was a predominant male gender association with snoring and witnessed apnoeas confirming the literature findings.[1,17] OSA symptoms of snoring and witnessed apnoeas increased with increasing age.[1,2,29] This study found a significant difference between 'White' and 'Coloured' race groups and 'White' and 'Indian' race groups with respect to body aches and pains, prevalence rates were significantly higher for Coloured and Indian races. Unlike studies cited no association was found between ethnicity and the other OSA symptoms. This was unexpected from the literature, where an association with snoring is described with Blacks, Mexican Americans and Pacific Islanders.[1] Snoring and witnessed apnoeas correlated with increasing BMI in this study, thus disagreeing with literature findings where visceral adipose tissue and neck circumference index (NCI) were seen as better markers of obesity associated with OSA.[1,4,27,29]

Familial associations of OSA symptoms found in this study corroborated with those cited in the literature.[1,3] With respect to comorbidities, depression was positively correlated with witnessed apnoeas, gasping and restless sleep while hypertension and heartburn correlated with snoring, again corroborating studies cited.[1,3,6,31]

Symptoms of OSA (snoring and waking up with a dry mouth) correlated with alcohol consumption, thus confirming the literature findings.[3,14] However, contrary to literature no relationship was noted with smoking.[1]

It appears from this study that snoring and OSA symptoms in South Africa have a similar prevalence and similar associations to that described in the literature. In view of the fact that 64.3% of subjects reported waking up at night and 15.7% reported waking up four or more times, it was surprising to find that only 11.8% of the subjects had previously consulted a doctor about a sleep problem. Thus it is recommended that the physician should routinely ask all adult patients direct specific questions about sleep problems. Stated differently, the first step in detecting OSA is acknowledging that it may be a problem for any given patient. It behooves all clinicians at primary care and speciality level to take cognizance of the above findings and to maintain a high index of suspicion for the diagnosis of OSA, when treating not only patients with the more obvious symptoms, eg, snoring and witnessed apnoeas, but also patients presenting with hypertension, heartburn and depression, in whom the treatment of OSA may reverse their medical condition.[3,11] This study provides insight into the necessity for implementing change in our approach to similar patients.

Based on the findings of this study, it is felt that OSA symptoms must be considered to be of significance in South Africa irrespective of race. We conclude that OSA symptoms are common in the population studied with prevalence rates comparable to overseas rates.

5.3 Recommendations for further study

Although this study examined the prevalence of OSA *symptoms* in adult general practice in South Africa, further studies need to be undertaken to establish the actual prevalence of OSA itself in South Africa.

Furthermore, examining the results, it is evident that in trying to answer one research question several other research questions for future analysis have arisen.

It is recommended that further studies be performed to elucidate the reasons for the high prevalence rates of tiredness on waking and excessive daytime sleepiness of 63.3% and 22.9% respectively in this study, as this is of clinical significance and has tremendous economic implications. As previously mentioned in the discussion an alarmingly high prevalence was noted for symptoms of tiredness on waking, morning headaches, restless sleep and waking with body aches and pains. This also has serious implications and consequences and it is recommended that future studies look into the above findings.

Studies need to be performed on the mechanism of progression of OSA in the absence of weight gain and other confounding variables.

Formal studies with comparison groups for ethnic variation and OSA have not been performed. Comorbid factors such as obesity, hypertension and non insulin dependant diabetes are more prevalent in certain ethnic groups and the prevalence of OSA relative to comorbidity and race is not known.[1]

Knowing how prevalent OSA symptoms are at a local level it would be of tremendous interest to screen patient populations at related speciality clinics eg, Hypertension, Cardiology, Gastro entrology and Psychiatry clinics for the main symptoms of OSA and further test the likely candidates for OSA. The cost saving implications would be tremendous if treating OSA may reverse the medical condition.

Since recognising OSA will affect the use of healthcare resources and the cost to the patient or the healthcare system for subsequent diagnosis and therapy, research

of practitioners' knowledge and education about OSA and its related symptoms, as well as their experience with OSA is warranted. This will hopefully result in an increased awareness of this problem and an increased willingness to screen at an earlier stage, for OSA symptoms, thus speeding up diagnosis and management of patients with OSA or OSA comorbidities.

Behavioural and performance deficits associated with OSA could carry a considerable hidden cost. The economic impact of sleepiness has been calculated as the sum of morbidity and mortality for motor vehicle crashes and industrial accidents.[1] Reports using a variety of indirect sources and assumptions estimate an indirect cost in the billions of dollors.[1] The degree to which OSA contributes to this figure may be significant; it is recommended that further studies be undertaken to determine these indirect costs associated with OSA or OSA symptoms such as sleepiness at a local level in order to point out the priorities for education of the public and medical sectors and to lead to a more vigilant recognition of the effects of OSA.

In summary, knowing that snoring and OSA symptoms are highly prevalent at a local level, practitioners need to routinely and directly look for the presence of snoring and OSA symptoms in order to increase the chance of accurately identifying and appropriately managing patients subjected to OSA or its comorbidities, which although often overlooked have tremendously far reaching medicopsychosocial, economic and public saftely implications.

REFERENCES:

1) Kingman PS, Redline S. Recognition of Obstructive Sleep Apnoea. American Journal of Respiratory Critical Care Medicine 1996; 154 : 279-289.

2) Kryger MR, Roth T, Dement WC. Principles and Practise of Sleep Medicine. London: WB Saunders Company, 1989.

3) Pack AI. Obstructive Sleep Apnoea. In: Advances in Internal Medicine 1994; 39 : 517-569.

4) Lavie P. Incidence of Sleep Apnoea in a Presumably Healthy Working Population; A Significant Relationship with Excessive Daytime Sleepiness. Sleep 1983; 6 : 3112-318.

5) George CP, Nickerson, Miller T, Kryger M. Sleep Apnoea Patients have more Automobile Accidents (letter). Lancet 1987; 8556 : 447.

6) Fletcher EC. The Relationship between Systemic Hypertension and Obstructive Sleep Apnoea: Facts and Theory. American Journal of Medicine 1995; 98 : 118-128.

7) Waller PC, Bhopal RS. Is Snoring a Cause of Vascular Disease? Lancet 1989; 1 : 143-146.

8) D'Alessandro R, Magelli C, Gamberuni G. Snoring Every Night as a Risk Factor for Myocardial Infarction : a Case Control Study. British Medical Journal 1990; 300 : 1557-1558.

9) Suzuki M, Otsuka K, Guilleminault C. Long Term Nasal Continuos Positive Pressure Administration can Normalise Hypertension in Obstructive Sleep Apnoea Patients. Sleep 1993; 16 : 526-529.

10) Chan HS, Chiu HFK, Tse LKK, Woo KS. Obstructive Sleep Apnoea Presenting with Nocturnal Angina, Heart Failure, and Near Miss Sudden Death. Chest 1991; 99 : 1023-1025.

11) Grunstein RR et al. Impact of Self Reported Sleep Breathing Disturbances on Psychosocial Performance in the Swedish Obese Subjects (SOS) study. Sleep 1995; 18 (8) : 635-643.

12) Greensberg G, Watson R, Depule D. Neuropsychological Dysfunction in Sleep Apnoea. Sleep 1987; 10 : 254-262.

13) Naegele B et al. Deficits of Cognitive Executive Functions in Patients with Sleep Apnoea Syndrome. Sleep 1995; 18 (1) : 43-52.

14) Mc Namara SG, Gunstein RR, Sullivan CE. Obstructive Sleep Apnoea. Thorax 1993; 48 : 754-762.

15) Stradling JR. Obstructive Sleep Apnoea and Driving. British Medical Journal 1989; 298 (6679) : 904-905.

16) Rediine S, Young T. Epidemiology and Natural History of Obstructive Sleep Apnoea. Ear Nose Throat Journal 1993; 72 : 20-26.

17) Pendlebury ST, Pepin JL, Veale D, Levy P. Natural Evolution of Moderate Sleep Apnoea Syndrome : Significant Progression Over a Mean of 17 Months. Thorax 1997; 52 : 872-878.

18) Johnston TS, Halberstadt J. Phantom of the Night: Overcome Sleep Apnoea Syndrome and Snoring. Cambridge: New Technology Publishing, 1994 : 27-44.

19) Redline S, Kingman PS. Recognition and Consequences of Obstructive Sleep Apnoea Hypopnoea Syndrome. Sleep Disorders 1998; 19 (1) : 1-19.

20) Barnes DJ. Sleep Apnoea, Obstructive. MIMS Disease Index. Pretoria : Times Media Ltd, 1992.

21) Johns MW. A New Method for Measuring Day Time Sleepiness : The Epworth Sleepiness Scale. Sleep 1991; 14 : 540-545.

22) Lugaresi E et al. Some Epidemiological Data on Snoring and Cardiocirculatory Disturbances. Sleep 1980; 3 : 221-224.

23) Koskenmo M et al. Snoring as a Risk Factor for Ischaemic Heart Disease and Stroke in Men. British Medical Journal 1987; 294 : 16-19.

24) Gislason T, Aberg H, Taube A. Snoring and Systemic Hypertension – an Epidemiological Study. Acta Medica Scandinavia 1987; 222 : 415-421.

25) Norton PG, Dunn EV. Snoring as a Risk Factor for Disease: an Epidemiological Survey. British Medical Journal 1985; 231 : 630-632.

26) Flemons WW, Remmers JE. The Diagnosis of Sleep Apnoea: Questionnaire and Home Studies; Methods of Diagnosing Sleep Apnoea. Sleep 1996; 19 : 243-247.

27) Pillar G, Peled N, Katz N, Lavie P. Predictive Value of Specific Risk Factors, Symptoms and Signs, in Diagnosing Obstructive Sleep Apnoea and its Severity. Journal of Sleep Respiration 1992; 3 : 241-244.

28) Franceschi M et al. Excessive Daytime Sleepiness: a One Year Study in an Unselected Inpatient Population. Sleep 1982; 5 : 239-47.

29) Shinohara E et al. Visceral Fat Accumulation as an Important Risk Factor for Obstructive Sleep Apnoea Syndrome in Obese Subjects. Journal of Internal Medicine 1997; 241 : 11-18.

30) Strools RA, Guillemin ault AI, Dement WC. Traffic Accidents in Commercial Long Haul Truck Drivers: the Influence of Sleep Disordered Breathing and Obesity. Sleep 1994; 17 : 619-623.

31) Khalil MM, Rifaie OA. Electrocardiographic changes in Obstructive Sleep Apnoea Syndrome. Respiratory Medicine 1998; 92 : 25-27.

32) Kapuniai LE et al. Identifying Sleep Apnoea from Self Reports. Sleep 1988; 11 : 430-436.

33) Strading JR, Gosby JH. Predictors and Prevalence of Obstructive Sleep Apnoea and Snoring in 1001 middle aged men. Thorax 1991; 46 : 85-90.

34) Rosenbaum M, Laibel RL, Hirsch J. Medical Progress: Obesity. New England Journal of Medicine 1997; 337 (6) : 396-408.

35) Ailhaud G, Guy-Grand B, Lafontan M, Ricquier D. Obesity in Europe 91. London: John Libbey and Company Ltd., 1992.

APPENDIX 1

QUESTIONNAIRE WITH COVERING LETTER

SNORING AND SLEEP APNOEA SURVEY

Dear patient,

With the assistance of your doctor, we are conducting a survey about the prevalence of snoring and sleep apnoea symptoms (holding one's breath during sleep). The information we gather will be used to help us understand how common this condition is in urban general practices in Johannesburg which will be to the benefit of both the patient and the doctor treating him/her. We would like you to help us by filling in the attached questionnaire. You may choose whether you want to fill in the questionnaire or not. You are assured that if you do not wish to fill in the form, your care with your doctor will not be affected in any way. Please note that the questionnaire is completely anonymous and confidential, your name will therefore not appear on the questionnaire and you cannot be identified in any way.

This study is being undertaken by Dr. Mary Rouhani, a postgraduate student at the Wits Department of Family Medicine and her supervisor Dr. Alison Bentley (a specialist in sleep medicine).

If you would like more information about sleep apnoea, please speak to your doctor. Information leaflets are also available at the reception counter, in this regard.

If you are willing to fill in the questionnaire, please do so now, before seeing your doctor. Unfortunately the form cannot be taken home and returned later. We would like you to fill in the questionnaire as carefully and completely as possible. If you do not wish to answer any question for whatever reason, please feel free to do so. When you have finished, please check to see that you have answered all the questions and then place your form in the box.

If you decide not to fill in the questionnaire, we still request you to place your form in the box provided.

Thank you for your time

Dr. M. Rouhani
MBBCh (Wits)

Sleep apnoea Research Questionnaire

1. How old are you? (tick appropriate box)

20-30	31-40	41-50	51-60	61-70	71-80	Over 81
2	3	4	5	6	7	8

2. Are you male or female? (tick box)

Male	Female

3. What is the last standard you passed at school? Standard _____

4. Please give details of any diplomas or degrees completed since school.

_____

5. What is your occupation? (tick appropriate box)

Housewife	Student	Pensioner/retired	Unemployed	Employed
H	S	P	U	O

If employed, give details, what type of job do you do?

_____

6. Which ethnic group do you belong to: _____

7. How much do you weigh? _____ kilograms or _____ pounds

8. How tall are you? _____ feet/inches or _____ Centimetres

9. Do you consider that you have a sleeping problem?

Yes	No

If yes, please specify_____

10. Have you ever consulted a doctor about a sleeping problem?

Yes	No

11. What time do you usually go to bed?:
Weekend _____ Weekday _____

12. What time do you usually fall asleep?
Weekend _____ Weekday _____
13. What time do you usually wake up in the morning?:
Weekend _____ Weekday _____
14. Do you wake up at night?

Yes	No

If yes how many times? _____

If yes for what reason? _____

If yes how long altogether? _____

15. Which of the following symptoms do you experience or which of the following symptoms have you been told that you have by a partner? (tick appropriate box)

Snoring	Yes	No	Do not know	S
Stop breathing while asleep (witnessed apnoeas)	Yes	No	Do not know	A
Restless sleep (frequent awakenings)	Yes	No	Do not know	R
Waking up gasping	Yes	No	Do not know	G

16. Looking back to five years ago.
Which of the following symptoms did you experience or which of the following symptoms were you told that you had by a partner? (tick appropriate box)

Snoring	Yes	No	Do not know	S
Stop breathing while asleep (witnessed apnoeas)	Yes	No	Do not know	A
Restless sleep (frequent awakenings)	Yes	No	Do not know	R
Waking up gasping	Yes	No	Do not know	G

17. Is there a family history of: (tick appropriate box)

Snoring	Yes	No	Do not know	S
Stop breathing while asleep (witnessed apnoeas)	Yes	No	Do not know	A
Restless sleep (frequent awakenings)	Yes	No	Do not know	R
Waking up gasping	Yes	No	Do not know	G

18. In the morning on waking do you have? (tick box)

Headache	Yes	No	H
Dry mouth	Yes	No	D
Tiredness	Yes	No	T
Bodily aches and pains	Yes	No	B

19. How likely are you to doze off or fall asleep in the following situations: (tick appropriate box)

	Never	Seldom	Likely	Very Likely	
Sitting and reading					R
Watching TV					T
Sitting, inactive in a public place					I
Passenger in a car for an hour without a break					P
Lying down to rest in the afternoon					L
Sitting and talking to somebody					S
Sitting quietly after lunch with no alcohol					Q
In a car, while stopped for a few minutes in the traffic					C

20. Have you ever been told that you have any of the following conditions?

High blood pressure	Yes	No	H
Heartburn or Reflux	Yes	No	R
Depression	Yes	No	D
Angina	Yes	No	A
Stroke	Yes	No	S
Heart failure	Yes	No	C

21. Please list below medication that you are taking plus the dosage.

Medication	Dose	Times per day

22. How many cigarettes do you smoke per day?

None	1-5	6-10	11-20	21-30	31-40	Over 40
0	5	10	20	30	40	50

23. Do you drink any alcohol?

Yes	No

If yes, how many glasses (beer, wine, spirits) do you drink? __ per day __ per week

24. Are you interested in more information regarding sleep apnoea syndrome?

Yes	No

If you have any questions about sleep apnoea please collect information leaflet at reception counter or ask your doctor.

Thank you for completing this questionnaire.

Author Rouhani-N M M
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