Three Dimensional Imaging Based Diagnosis for Obstructive Sleep Apnoea: A Conceptual Framework

[1]Syed M. S. Islam, [1]Mithran S. Goonewardene and [2]Paul Sillifant

[1] School of Dentistry, The University of Western Australia, 35 Stirling Highway, Crawley, WA 6009, Australia
[2] Department of Oral and Maxillofacial Surgery, Royal Perth Hospital, 197 Wellington Street, Perth, WA 6000, Australia
[1]`{syed.islam,mithran.goonewardene}@uwa.edu.au`, [2]paulsillifant@hotmail.com

Abstract. Obstructive Sleep Apnoea (OSA) is a disorder in which repetitive periodic cessation of breathing for 10 seconds or more occurs during sleep despite increased effort to breathe. It leads to day-time sleepiness, poorer health, increased healthcare and higher work-related and road accidents costing the national economy billions of dollars per year. Early intervention may improve health outcomes for the sufferers. In this article, a hierarchical diagnostic approach is proposed in which at first a quick and safe three-dimensional (3D) surface imaging based technique is used to identify patients susceptible to OSA, thereby allowing a cost-effective patient screening. The susceptible patients are referred for volume imaging such as Cone Beam Computed Tomography (CBCT) from which the airway and other hard-tissue anatomical features can be extracted. Age and gender specific 3D facial norms and different thresholds have been proposed to compute against which individualized features can be judged to determine the presence of OSA. Finally, the severity of OSA is measured by polysomnography sleep study only for those patients who are confirmed for OSA by both surface and volume image-based analysis.

1 Introduction

Sleep apnoea is a serious health issue with significant public health implications [6, 13]. There are three types of sleep apnoea: obstructive (OSA), central (CSA) and mixed (combination of the two). In OSA (84% of cases), mechanical factors play an integral role in the reduction of airflow despite continued respiratory effort [1]. In CSA (0.4% of cases) the physiological respiratory control processes fail to maintain the required respiratory function for optimal health.

OSA is characterised by the presence of apnoeas (i.e. a complete cessation of breathing despite respiratory effort) or hypopneas, defined as greater than 30% reduction in chest and/or abdominal expansion during breathing or shallow breathing lasting at least 10 seconds combined with at least a 4% reduction in oxygen desaturation. Numerous indices have been developed to express the severity of sleep apnoea diagnosed using polysomnography and include the apnoea index (AI) which represents the total number of apnoeas per hour and the Apnoea-Hypopnea Index (AHI), which represents the total combined apnoeas and hypopnoeas per hour. The AHI has been divided into severity scales: mild ($5 < \text{AHI} < 15$), moderate ($15 < \text{AHI} < 30$) and severe ($\text{AHI} > 30$). Additional indices that have been utilised include sleep arousals (Respiratory Disturbance Index) and subjective patient perceptions of sleep impact on daytime activities (Epworth Sleepiness Scale).

During apnoeic episodes, arterial blood oxygen saturation decreases, and sympathetic activity and blood pressure increases. Each apnoeic episode ends with an arousal from sleep, resulting in marked fragmentation of sleep in affected individuals. Excessive daytime sleepiness is a major consequence of OSA. OSA has also been linked to significant conditions such as hypertension [16, 9], ischaemic heart disease and stroke [18], premature death [17], and impairment of cognitive functions [8] which may contribute to motor vehicle and workplace related accidents (comparable to functioning while intoxicated) [7]. A study from The University of British Columbia demonstrated that a person with OSA is twice as likely to be involved in a motor vehicle accident [19]. For untreated individuals, it has been established that there is a 37% higher 5-year morbidity and mortality rate [14].

It is estimated that 775,000 Australians (4.7% of the adult population) suffer from OSA [15]. The Busselton (Australia) Health Survey [2] of 294 men aged 40 to 65 years revealed that about 26% of individuals have mild and 10% have severe levels of sleep apnoea. The total financial and non-financial burden of OSA in Australia was estimated as 21.2 billion dollars in 2010 including direct health care cost of $575.42 million and indirect health care cost (due to lost productivity, deadweight loss, workplace/motor vehicle accidents, social security payments etc.) of $2.6 billion [18]. In U.S. it was estimated in 2008 that the average additional annual health care cost of an untreated sleep apnoea patient is US $1,336 contributing an estimated total of $3.4 billion/year additional medical costs [1].

In this article, we introduce a novel quantitative diagnostic method for OSA based on the combination of two approaches related to two different imaging modalities (surface and volume). The first approach is based on the analysis of a three-dimensional surface scan of a subject (using e.g. a 3dMD face scanner). We propose to extract quantitative facial features from the scan to differentiate between facial morphologies of OSA patients and normal non-apnoeic individuals. The relative position of the upper and lower jaws to the skull base and in turn to each other can be assessed as represented by the external facial appearance. These facial features can be evaluated to determine the relationship between facial morphology and the severity of OSA. 3D surface facial scanning has the advantage of being a non-invasive imaging tool which does not require exposure to ionizing radiation. The second approach relates to the application of state-of-the-art dental imaging in the form of a Cone Beam CT to obtain a 3D (volumetric) representation of the hard and soft tissues. The determination of the morphology (shape and structure) of the airway of OSA patients should help in revealing any significant deviations from the airway of normal individuals. As Cone Beam CT is a readily available imaging tool in most clinics, the proposed diagnostic method is easily accessible with many control non-OSA patients imaged for unrelated dental anomalies. The overall outcome of the article is the development of improved conservative diagnostic methods which will be accessible to wider patient groups and will contribute in early intervention.

The rest of the article is organized as follows. Various approaches currently used for the diagnosis of OSA is described in Section 2. The conceptual framework for our proposed approach is elaborated in Section 3. Proposal for the evaluation of the new diagnostic method is discussed in Section 4 followed by the conclusions in Section 5.

2 Existing Diagnostic Approaches for OSA

OSA is seen more frequently in older males and is related to many predisposing factors such as increased Body Mass Index (BMI), increased neck circumference, smoking, alcohol consumption and enlarged tonsils and adenoids. Clinicians also recognise specific dentofacial deformities which predispose individuals to the development of OSA. The obvious retrusion or underdevelopment of the lower jaw and and/or the upper jaw alerts the clinician to the possibility of a patient susceptible to OSA.

Today, overnight polysomnography remains the ‘gold standard’ diagnostic method for OSA. It is a monitored sleep study to record biophysiological changes that occur during sleep. Measurements include electroencephalogram, electrooculograms, submental electromyogram, oronasal airflow, chest wall motion, and arterial oxygen saturation. In addition to the significant inconvenience to the patient, polysomnography requires sophisticated specialist facilities, technical and scientific staff and sleep clinicians, which are commonly not available in all regions.

Imaging techniques have been considered as useful adjunctive tools to diagnose and plan the treatment of OSA, with the radiographic head film (cephalometric) analysis being the most convenient and widely used [3]. However, the cephalometric analysis is inherently limited because of its two dimensional imaging and the lack of information about the airway volume and dimensions [5]. In addition, measurements are obtained with the patient in the upright position which may not accurately reflect the distortion of the airway in the supine sleeping position. This may create an underestimation of the degree and pattern of airway narrowing and/or collapse. Lee et al. [10, 11, 12] analysed facial characteristics to predict OSA with an accuracy of 76.1% using 2D photographic and cephalometric images. These have limitations compared to 3D surface and volume data. For example, while they demonstrated a relationship between facial structural measurements such as alar width and intercanthal distance, they did not assess 3D positional relationships of the relevant structural components representing the underlying jaw base, which is the focus of this article.

During the last few years, there has been significant interest in developing conservative, cost-effective, patient-convenient and widely applicable methods to diagnose and treat OSA. Although the morphology of patients diagnosed with OSA has been well documented using two dimensional (2D) imaging techniques, and to a much lesser degree using 3D imaging techniques, no specific stratified evaluation has demonstrated the impact of progressive distortions of the maxillomandibular structures on airflow and sleep performance.

3 Proposed Methods and Techniques

Considering the cost effectiveness and the simplicity, we propose a hierarchical framework for diagnosing OSA. We would like to keep the cheaper and widely accessible measures at the beginning and thus screening out a number of patients before suggesting for more expensive and exhaustive approaches. The detailed framework is described in this section.

3.1 Statistical Design

A null hypothesis for developing the new diagnostic approach can be defined as follows: there will be a statistically significant difference in the proportion of patients who are correctly diagnosed with OSA using the new method as compared to the gold standard.

The sample size for the above hypothesis can conservatively be estimated using an expected sensitivity (probability of correctly identifying a patient as positive by the proposed approach given they have OSA) of 0.85 and specificity (probability of correctly identifying a patient as negative by the new approach given they do not have OSA) of 0.95, and a 95% confidence level. A sample size of 100 OSA patients and 100 non-OSA participants would provide a 0.07 precision for sensitivity and 0.04 precision for specificity.

3.2 Determination of Norms and Thresholds

This approach requires a prior set up of age and gender specific facial norms (nn) used as references. For that purpose, we propose to compute the age and gender specific average faces from a large sample of non-OSA subjects. In addition to these average-face norms, we also propose to determine some other thresholds associated with other discriminating features as illustrated in Fig. 1 and explained below.

Threshold t_1 can be established as follows from 3D ear to ear facial surface images (e.g. Fig. 2) of the 100 patients diagnosed with OSA by polysomnography. The face area can be detected and cropped and various surface features (e.g. length of the maxilla, mandible and chin and the circumference of the neck) can be extracted. The relative shape ratios (RSRs) of these different features (e.g. length of maxilla with respect to the mandible and that of maxilla and mandible compared to the forehead and neck) can be computed. These features then can

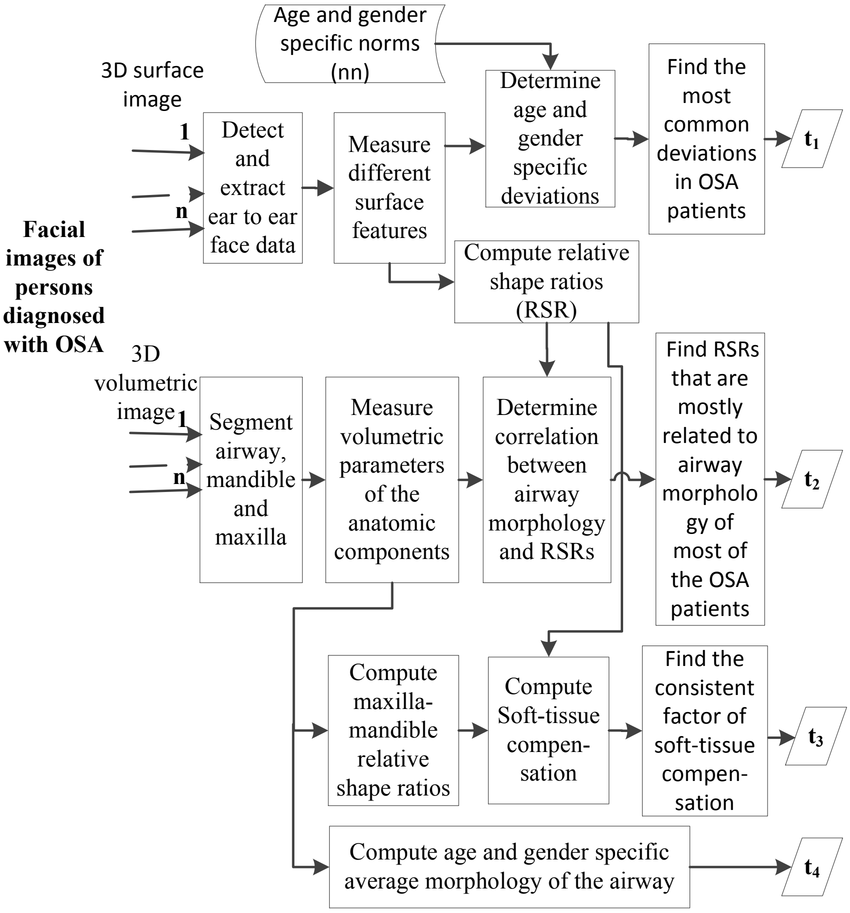

Fig. 1. Block diagram of the computation of different thresholds (t_1, t_2, t_3, and t_4) used in the proposed diagnostic algorithm.

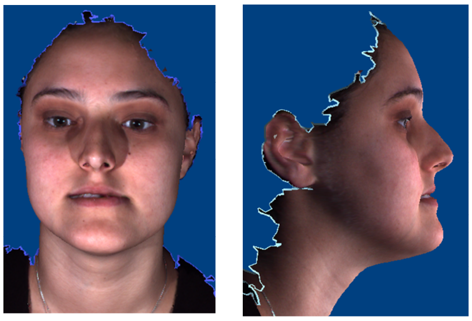

Fig. 2. 3D textured image of a person's frontal (left) and right profile.

be compared with the age and gender specific norms to outline any deviations from the norms. The threshold t_1 can then be derived from these deviations.

Three more thresholds can be determined from 3D volumetric images which can be acquired using a Cone Beam CT scanner from the same patients above. The volumetric data of the airway (Fig. 3) and other anatomical features can be segmented from these data using commercial software such as Dolphin, 3dMD-vultus and 3D Slicer. Different volumetric parameters can be measured and statistically correlated with the facial RSRs computed from the facial surface images. The RSR (of each age and gender group) with the highest correlation factor can be used as a threshold (t_2). The relative shape ratio of maxilla and mandible computed from volumetric data can be compared with those obtained from surface data (3dMD) to evaluate the most common soft-tissue compensation factor (t_3). The average morphology of the airway (threshold, t_4) of the different age and gender subgroups can be computed using the above software or computer programming using MATLAB.

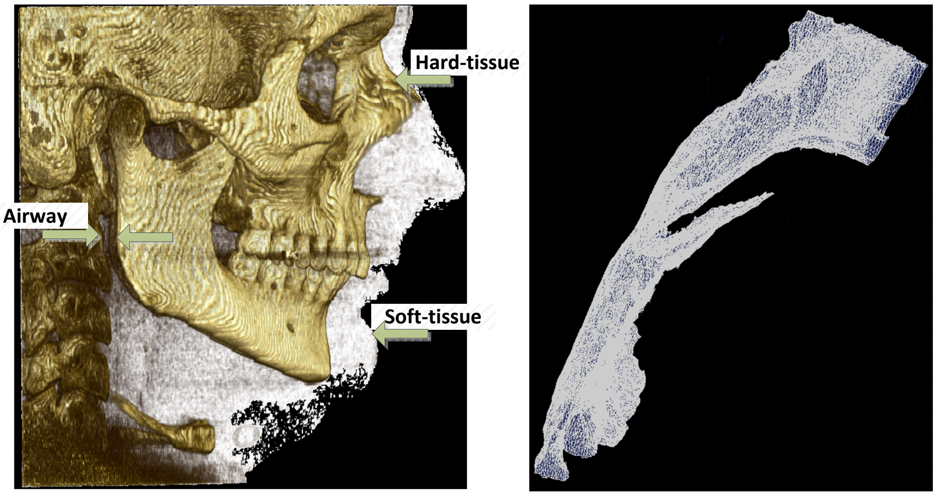

Fig. 3. 3D volumetric image of an OSA patient and his digitally segmented airway represented in wireframe model.

3.3 Diagnosis Using the New Approach

As illustrated in Fig. 4, in the proposed diagnostic framework, a subject presenting for an OSA test will firstly be diagnosed using a surface image. A 3dMD scan (e.g. Fig. 2) will be taken using the 3dMD Facial Scan System. The captured image data will be represented as a 3D surface mesh. Then quantitative facial shape features and ratios will be extracted or derived from the surface data.

An individualized norm will be determined based on the age and gender specific norms (nn) to localize and quantify any shape deviations (d_1) of the facial

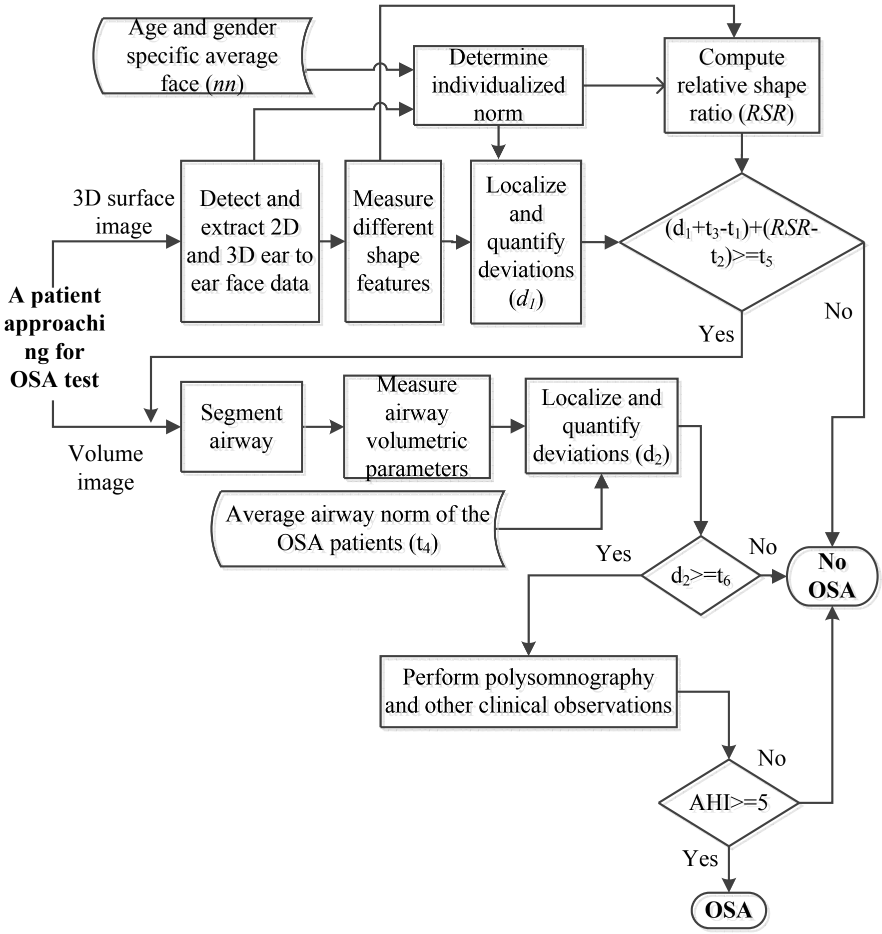

Fig. 4. Block diagram of the proposed diagnostic methods.

shape of the patient relative to a non-OSA subject of the same gender and age group. These shape deviations along with a soft-tissue compensation factor (t_3), will be compared with the morphological threshold t_1. Furthermore, the RSR of the patient will also be compared with the threshold t_2. After computation of these thresholds and deviations obtained from the analysis of the surface image only, patients will be primary identified as a candidate for OSA if the summation of the following two differences is greater than or equal to an empirically evaluated threshold t_5: (i) the difference of the soft-tissue deviations including any soft-tissue compensations from the most common deviations in OSA patients and, (ii) the difference of patients' relative shape ratio from the similar ratio of the most of the OSA patients. The condition can be mathematically represented as in Equation 1.

$$(d_1 + t_3 - t_1) + (RSR - t_2) >= t_5 \tag{1}$$

The potential subjects will then be exposed to a Cone Beam CT scan for a segmental airway assessment, and volumetric parameters of the airway will be measured. Comparing the average airway norm of OSA patients (t_4), the deviation (d_2) in the airway will be calculated. If the subjects' deviations are greater than or equal to an empirically determined threshold t_6, they will be recommended for a polysomnographic sleep study and other clinical observations in order to finally confirm the presence and severity of OSA expressed in AHI.

4 Evaluation of the Proposed Diagnostic Method

A comparison of the new 3D imaging-based diagnostic method with findings from polysomnography can be performed through a test for difference in proportions for the paired-sample design [4]. The diagnosis can be defined as successful if AHI (found using polysomnographic sleep study) of the positively diagnosed patients (using the proposed method) is found to be greater than 5 (the threshold measure of apnoea).

The test for difference in proportions for the paired-sample design can then be used to reject the hypothesis that there will be a statistically significant difference in the proportion of patients who are correctly diagnosed with OSA using the proposed method as compared to the gold standard (polysomnography). If there is no statistically significant difference in the proportion of patients who benefit from the proposed 3D image-based approach, then it should be widely adopted. The test can be specifically described as follows:

1. For the 200 randomized subjects, apply the 3D imaging-based diagnosis approach (response Y1) and standard polysomnography procedure (matched control, response Y2) [4].
2. Define a failure by a 'miss' and 'false alarm' (adopting the terminology of detection theory), i.e. if the subject is diagnosed with the proposed approach while they are not diagnosed using polysomnography, then it is a false alarm. We then determine the proportion of cases when the proposed method resulted in a success (P1) and when polysomnography resulted in success (P2).

3. If the proportions P1 and P2 computed above are equal, then reject the hypothesis that the proportion of successes is the same for our 3D imaging-based diagnosis method and polysomnography. (test statistics is unit-normally distributed; the exact formula is given in [4]).

5 Conclusions

The proposed conservative, cost-effective, patient-convenient and widely applicable methods to diagnose OSA will facilitate more accessible diagnosis of a larger number of patient groups than is possible with polysomnography and will enhance early intervention.

The purpose of the proposed diagnostic approach is not to replace the sleep studies but to screen and then to stratify adult OSA patients for various modes of treatment based on the anatomical features and airflow measurements. Importantly, the proposed approach will also provide guidance to clinicians who manage significant jaw structure problems in children with occasionally irreversible conventional orthodontics with little regard for the consequences of leaving the child prone to developing sleep apnoea with their underlying jaw structure remaining atypical. The specific patterns of jaw morphology can be identified during the diagnosis in individuals who would be considered for surgical management of their jaw deformity in adulthood rather than attempting to compensate the teeth for the jaw structure. Clinicians may then modify the way in which they advise patients with more severe jaw structure problems based on the impact on predisposition to OSA. Moreover, after screening, morphologically predisposed patients may be warned about lifestyle habits which may contribute to the possibility of developing OSA at a later age.

Acknowledgments

This research is sponsored by two Special Donation grants from the Australian Society of Orthodontists Foundation of Research and Education (PG51311900 and PG51312000) and, by a Research Development Award (PG12104373) and a Research Collaboration Award (PG12105002) from the University of Western Australia.

Bibliography

[1] Aarab G, Lobbezoo F, Hamburger H, Naeije M (2010) Effects of an oral appliance with different mandibular protrusion positions at a constant vertical dimension on obstructive sleep apnea. Clinical Oral Investigations 14(3)

[2] Bearpark H, Elliott L, Grunstein R, et al (1995) Snoring and sleep apnea. a population study in australian men. Am J Respir Crit Care Med 151:1459–1465

[3] Conley R (2011) Evidence for dental and dental specialty treatment of obstructive sleep apnoea. part 1: the adult osa patient and part 2: the paediatric and adolescent patient. J Oral Rehabil 38(2):136–56

[4] Connor R (1987) Sample size for testing differences in proportions for the paired-sample design. Biometrics 43(11):207–211

[5] Doff M, Hoekema A, Pruim G, van der Hoeven J, de Bont L, Stegenga B (2009) Effects of a mandibular advancement device on the upper airway morphology: a cephalometric analysis. J Oral Rehabil 36(5):330–7

[6] George C (2001) Reduction in motor vehicle collisions following treatment of sleep apnoea with nasal cpap. Thorax 56(7):508–512, DOI 10.1136/thorax.56.7.508

[7] Horne J, Reyner L (1995) Sleep related vehicle accidents. Bmj 310(6979):565–7

[8] Klitzman D, Miller A (1994) Obstructive sleep apnea syndrome: complications and sequelae. Mt Sinai J Med 61(2):113–21

[9] Lavie P, Herer P, Hoffstein V (2008) Obstructive sleep apnoea syndrome as a risk factor for hypertension: population study. Bmj 320(7233):479–82

[10] Lee R, Chan A, Grunstein R, Cistulli P (2009) Craniofacial phenotyping in obstructive sleep apnea - a novel quantitative photographic approach. Sleep 32(1):37–45

[11] Lee R, Petocz P, Prvan T, Chan A, Grunstein R, Cistulli P (2009) Prediction of obstructive sleep apnea with craniofacial photographic analysis. Sleep 32(1):46–52

[12] Lee R, Sutherland K, Chan A, Grunstein R, Cistulli P (2010) Relationship between surface facial dimensions and upper airway structures in obstructive sleep apnea. Sleep 33(9):1249–1254

[13] Lee W, Nagubadi S, Kryger M, Mokhlesi B (2008) Epidemiology of obstructive sleep apnea: a population-based perspective. Expert Rev Respir Med 2(3):349–364

[14] Marti S, Sampol G, Munoz X, Torres F, Roca A, Lloberes P, et al (2002) Mortality in severe sleep apnoea/hypopnoea syndrome patients: impact of treatment. Eur Respir J 20(6):1511–8

[15] Morgenthaler T, Kagramanov V, Hanak V, et al (2006) Complex sleep apnea syndrome: is it a unique clinical syndrome? Sleep 29:1203–1209

[16] Nieto F, Young T, Lind B, Shahar E, Samet J, Redline S, et al (2000) Obstructive sleep apnoea syndrome as a risk factor for hypertension: population study. Jama 283(14):1829–36

[17] Rapoport D (1994) Treatment of sleep apnea syndromes. Mt Sinai J Med 61(2):123–30

[18] Report DAE (2011) Re-awakening australia - the economic cost of sleep disorders in australia. URL http://www.sleephealthfoundation.org.au/research.html

[19] ScienceDaily (2008) Sleep apnea doubles car crash risk, study shows. retrieved on oct 30, 2012. URL http://http://www.sciencedaily.com/releases/2008/02/080218214401.htm