Sleep apnoea/hypopnoea syndrome

JKW Chan, A Ho, RCC Leung, CKW Lai

Forty patients were evaluated using sleep studies for assessment of possible sleep apnoea/hypopnoea syndrome. Twenty-six patients had significant sleep apnoea based on polysomnography. Most of the patients with this syndrome had at least two of the three major symptoms of obstructive sleep apnoea—witnessed apnoeas, heavy snoring, and excessive daytime somnolence. The most common associated medical illnesses were hypertension (47%), chronic obstructive airways disease (16%), respiratory failure (15%), and allergic rhinitis (12%). All 26 patients with sleep apnoea/hypopnoea syndrome were treated with either continuous positive airways pressure or bilevel positive airways pressure with significant improvement. Minimum percentage arterial oxygen saturation increased significantly, with a median difference of 25.5 (interquartile range, 10 to 36; $P < 0.01$) and the apnoea/hypopnoea index decreased significantly with a median difference of 23.0 (interquartile range, 15 to 44; $P < 0.01$). The clinical features of this syndrome in Hong Kong are similar to those reported in the West.

HKMJ 1996;2:34-39

Key words: Sleep apnea syndromes; Snoring

Introduction

The sleep apnoea/hypopnoea syndrome (SAHS) has long been recognised in the West as a major cause of morbidity with a tendency to appear in obese, middle-aged men.[1] The introduction of nasal continuous positive airways pressure (CPAP) and uvulopalatopharyngoplasty (UPPP) in 1981 provided effective means of treatment for SAHS, and have since obviated the need for tracheostomy in many patients.[2,3]

The prevalence of SAHS in Southeast Asia is not well defined, although the clinical spectrum and treatment methods involved have been previously described.[4] An earlier study conducted in Hong Kong Chinese suggests that SAHS is not uncommon.[5] Epidemiological studies in Japan in a cohort of industrial workers show a prevalence of SAHS of 17%.[6] There are inter-population variations in the prevalence of SAHS. For instance, the prevalence of SAHS is high in Polynesians and South Islanders, compared with Caucasians, and is possibly related to an inherited pattern of obesity and body habitus.[7]

With increased recognition and awareness of SAHS in recent years—in particular its associations with vascular diseases such as hypertension, ischaemic heart disease, stroke, pulmonary hypertension, and right heart failure—the early diagnosis and treatment of this disease have assumed greater clinical significance.[8-11]

This paper is a retrospective review of the sleep studies performed at the Prince of Wales Hospital in 1994, and is an attempt to outline the characteristics of the patients presenting for sleep studies and the outcome of treatment. The Prince of Wales Hospital is a major teaching hospital in Hong Kong and serves a population of approximately 1.3 million.

Subjects and methods

Data was obtained retrospectively from case notes and printed sleep study reports held in patient medical records. From February 1994 through January 1995, 40 patients underwent a total of 112 sleep studies in the Department of Medicine at the hospital. Most of the patients underwent multiple studies which were performed either using a PolyG recorder (CNS Inc., Chanhassen, Mn, US) [41 studies] or using a portable pulse oximeter (Ohmeda Biox 3700, Louisville, Co,

Department of Medicine, Prince of Wales Hospital, Shatin, Hong Kong
JKW Chan, MB, BS
A Ho, MB, ChB, MRCP
RCC Leung, MD, FRACP
CKW Lai, DM, FRCP(Edin)
Correspondence to: Dr JKW Chan

US) [71 studies]. The polyG recorder monitored chest and abdominal respiratory movements indirectly by inductance plethysmography. The summation of the two respiratory channels, i.e. thoracic and abdominal, which correlated with tidal volume, was also derived. Respiratory airflow was measured using a nasal thermistor attached to the patient's nose. Heart rate and ECG were monitored via three ECG leads. Finger pulse oximetry was recorded using the pulse oximeter. Sleep staging was not recorded.

Apnoea was defined as at least 90% reduction in respiratory airflow lasting for more than 10 seconds with at least 4% oxygen desaturation. Hypopnoea was defined as a 50% to 90% reduction in respiratory airflow lasting for more than 10 seconds with at least 4% oxygen desaturation. Apnoea/hypopnoea was classified as obstructive when the thoracic and abdominal channels showed activity, and as central when both of these channels showed no activity. The apnoea/hypopnoea index (AHI) was defined as the number of combined apnoeas and hypopnoeas per hour of sleep.

Significant SAHS was defined as an AHI greater than 20 with clinical symptoms suggestive of sleep apnoea. We also included patients who had AHIs between 10 and 20 and severe symptoms such as heavy snoring and excessive daytime somnolence. Sleep apnoea/hypopnoea syndrome was stratified according to the degree of oxygen desaturation (minimum % SaO_2 > 85; between 70 to 85; < 70 were classified as mild, moderate, and severe SAHS, respectively) and AHI (10 to 20, mild; 20 to 40, moderate; > 40, severe).[12] All patients with clinically significant SAHS as defined above were treated with either CPAP or bilevel positive airways pressure (BiPAP). The optimal CPAP pressure was titrated by polysomnography or overnight oximetry with the aim of eliminating most hypopnoeas/apnoeas (AHI < 5) and/or of maintaining overnight SaO_2 at more than 90%. The PolyG recorder had been previously validated against conventional polysomnography and showed good agreement.[13] The Fischer's exact test was used for comparison of proportions. The Wilcoxon matched-pairs signed ranks test was used for the comparison of medians in paired samples. All other data are given as mean and standard deviation values.

Results

Thirty-two men and eight women (mean age, 45.3 y) participated in a total of 112 studies. Men had a mean body mass index (BMI) of 31.1 compared with a BMI of 27.1 for women. Twenty-six patients had clinically significant sleep apnoea on polysomnography and were similar demographically to the rest of the group (Table 1). Although there were many presenting symptoms, the three cardinal symptoms—obstructive sleep apnoea (OSA), loud snoring, witnessed apnoeas, and excessive daytime sleepiness—were commonly reported in patients both with and without SAHS. A number of medical illnesses were present in these patients (Table 2). Hypertension was common in the group with SAHS (47%).

Table 1. Sex, age and weight characteristics of sleep study subjects

	OSA* (n = 26)	no OSA (n = 14)
Male: female ratio	5.5 : 1	2.5 : 1
Mean ± (SD) age (y)	44.9 ± 10.8	46.2 ± 14.3
Mean ± (SD) BMI†	30.8 ± 4.6	29.4 ± 4.7

* OSA Obstructive sleep apnoea
† BMI Body mass index

Four of the six patients with hypercapnic respiratory failure had a background of chronic obstructive airways disease (COAD), one had pulmonary fibrosis and another had a chest wall deformity, post-thoracoplasty. The six patients with respiratory failure had a mean PaO_2 of 7.7 ±0.7 (SD) KPa and a mean $PaCO_2$ of 7.5 ± 1.6 (SD) KPa on room air, with clinical signs of pulmonary hypertension and right heart failure. Two patients had stable left ventricular failure due to hypertension and ischaemic heart disease, respectively.

The 26 patients with significant sleep apnoea on baseline sleep study underwent pressure titration studies (13 patients with polysomnography, 13 with overnight oximetry) on either CPAP (24 patients) or BiPAP (two patients). A comparison of the presenting symptoms of patients with and without OSA, revealed that snoring was reported in 100% of patients with OSA and in 86% of those without; 31% of those with OSA reported witnessed apnoeic episodes versus 7% in those without; 12% of those with OSA reported snoring as the sole complaint versus 36% in those without OSA. The results were not statistically significant due to the relatively small sample size.

Of the 26 patients with sleep apnoea, 17 had severe disease, seven had moderate disease, and two had mild disease based on the criteria of minimum % SaO_2

and AHI. Three patients had significant central apnoeas in addition to obstructive apnoeas. One patient had no evidence of significant sleep apnoea to account for his daytime somnolence and subsequently underwent a multiple sleep latency test which showed a mean sleep latency of 3.3 minutes with evidence of rapid eye movement sleep at sleep onset, suggestive of narcolepsy.

In general, all patients had objective improvement of their sleep apnoea on CPAP or BiPAP, (Figs 1 and 2). Baseline median % SaO_2 increased significantly after treatment from 62.5 to 90.0 without supplemental oxygen (n = 26; median difference, 25.5; interquartile range, 10 to 36, $P < 0.01$) and median AHI decreased significantly from 38.0 to 5.0 (n = 13; median difference, 23.0; interquartile range, 15.0 to 44.0; $P < 0.01$). Four patients (three with COAD, one with pulmonary fibrosis) needed supplemental oxygen in addition to CPAP to maintain oxygen saturation above 90% while sleeping. On discharge from hospital, 24 of 26 patients went home on either CPAP (22 patients), with a mean pressure of 9.3 ± 3.2 (SD) cmH_2O or BiPAP (two patients). The inspiratory positive airways pressure and expiratory positive airways pressure were 6/2 and 13/10 respectively, for the two patients on BiPAP.

All 24 patients returned to the outpatient clinic for follow up approximately four to six weeks following discharge. A total of seven patients discontinued treatment for various reasons. Five were unable to tolerate the machine, because of poor mask fitting, excessive pressure, sore nose and dry mouth. One patient no longer needed CPAP after successful weight reduction and another claimed financial difficulties as the

Table 2. Presenting symptoms and associated medical illnesses of sleep study subjects

Presenting symptoms	OSA* (n = 26) No. (%)	no OSA (n = 14) No. (%)	P value
Snoring	26 (100)	12 (86)	0.12
Excessive daytime somnolence	23 (88)	11 (79)	0.65
Witnessed apnoeas	8 (31)	1 (7)	0.12
Concentration and memory deficit	11 (43)	6 (43)	0.97
Nocturnal choking episodes	5 (19)	3 (21)	1.00
Daytime lethargy/diminished libido	9 (35)	3 (21)	0.48
Nocturia	7 (27)	2 (14)	0.45
Morning headaches	4 (15)	2 (14)	1.00
Atypical chest pain	3 (12)	1 (7)	1.00
Sleep disruption/arousals	2 (8)	2 (14)	0.60
Associated medical illnesses			
Hypertension	12 (47)	2 (14)	0.08
COAD†	4 (16)	1 (7)	0.64
Allergic rhinitis	3 (12)	2 (14)	1.00
Diabetes mellitus	3 (12)	1 (7)	1.00
Respiratory failure	4 (15)	2 (14)	1.00
Left ventricular failure	1 (4)	1 (7)	1.00
Ischaemic heart disease	1 (4)	1 (7)	1.00
Cerebrovascular accident	1 (4)	0 (0)	1.00

* OSA Obstructive sleep apnoea
† COAD Chronic obstructive airways disease

Fig 1. Effect of treatment with CPAP/BiPAP on mininum %SaO_2 in 26 patients with obstructive sleep apnoea. Bars indicate median values.

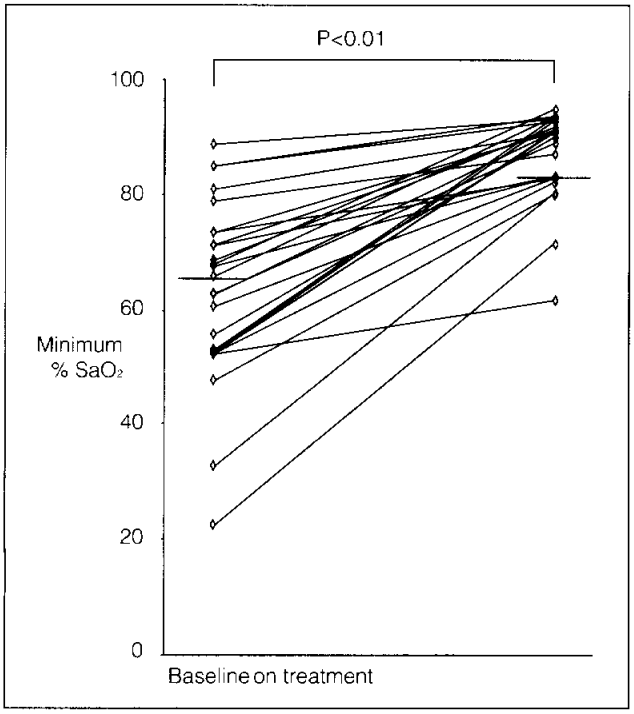
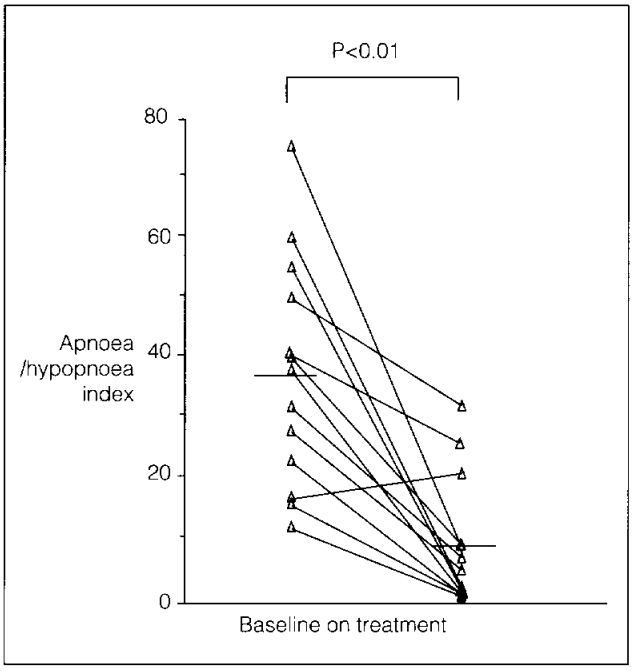


Fig 2. Effect of treatment with CPAP/BiPAP on apnoea/hypopnoea index in 13 patients with obstructive sleep apnoea. Bars indicate median values.



reason for stopping treatment. Eight patients entered a dietary programme to lose weight and one patient was referred to the Ear, Nose and Throat clinic for surgical assessment after an unsuccessful trial using CPAP.

Discussion

It is now evident that SAHS is one of the the most common chronic disorders, the overall prevalence of the OSA syndrome being approximately 9% in Western countries.[14] A large epidemiologcal study estimates that 4% of men and 2% of women in the middle-aged workforce have SAHS.[15] The prevalence of SAHS in Southeast Asian countries and among the Chinese population is not well defined. With advances in knowledge of the condition and sleep monitoring technology, it has become increasingly recognised in clinical practice.

The male to female ratio of 5.5:1 in patients with SAHS in our series was lower than the previously reported ratio of 10:1, although it has been suggested that the prevalence rate in women may be underestimated.[16] Obesity is a known risk factor for SAHS. In this study, the patients were obese with a mean BMI of 30.8. The majority of patients with SAHS had at least two of the three cardinal symptoms of SAHS, i.e. snoring, excessive daytime sleepiness, and witnessed apnoeas. This may be partly due to self-selection because patients with these symptoms were more likely to be referred to a respiratory clinic for sleep studies.

There were no significant differences in the incidence of snoring, daytime somnolence, and witnessed apnoeas between patients with and without OSA, probably as a result of the small sample size. The usual precipitating factor for presentation was loud snoring, often with complaints from a patient's bed partner. Some patients had additional factors such as heavy alcohol use, micrognathia, and previous UPPP which may alter the upper airway anatomy and augment the degree of sleep apnoea.

Commonly associated medical illnesses in patients with SAHS included hypertension, COAD with hypercapnic respiratory failure, allergic rhinitis, and diabetes mellitus. Surprisingly, there were relatively few patients with other cardiovascular diseases such as ischaemic heart disease and stroke. Patients with allergic rhinitis often had symptoms of nasal congestion and post-nasal drip which can worsen sleep apnoea by causing the patient to mouth-breathe, thus exacerbating their snoring.

Approximately two-thirds of patients admitted for assessment had evidence of OSA on their diagnostic studies and were then treated with CPAP or BiPAP. Stratification of the severity of SAHS according to

minimum SaO_2 and AHI showed that almost two-thirds of our patients with OSA were in the severe category, possibly as a result of selection bias as most patients who were admitted for sleep studies often have more severe symptoms and disease.

The link between SAHS and hypertension is well-established. Hypertension was found in as many as 50% of patients presenting for the first time with sleep apnoea.[17] In this study, there were more patients with a previous diagnosis of hypertension in the severe group compared with the moderate and mild groups combined, although the mean blood pressure recorded during admission did not differ among the three groups. Of the two patients with BMI values less than 27 who had severe disease, one had previous UPPP which may have altered the anatomy of the laryngeal inlet and upper airway, predisposing the patient to OSA. The second patient had micrognathia and a strong family history of snoring. Three patients had clinically significant central apnoea, of which one had type 2 respiratory failure and congestive cardiac failure (CCF). The apnoeic episodes were not characteristic of the Cheyne-Stokes breathing pattern usually associated with CCF. Recent studies have shown that CPAP is able to abolish the central apnoeas and improve left ventricular ejection fraction in patients with CCF.[18]

Two patients were successfully treated with BiPAP. One could not tolerate the high CPAP pressure of 13 cmH_2O required and changed to BiPAP; the other patient had type 2 respiratory failure with nocturnal hypoventilation, which was not controlled on CPAP and supplemental oxygen. Some patients had more than one baseline study because the first study was non-diagnostic due to poor sleep (the "first night effect") or technical problems. Patients with OSA had on average two studies on CPAP/BiPAP to determine the optimal pressure required to abolish obstruction. Nasal CPAP is an effective treatment for SAHS but may be difficult to use for many reasons. Only two-thirds of patients prescribed CPAP for SAHS use the device long term and many use it only for limited hours during sleep.[19,20] However, there is clinical evidence of improved daytime cognitive function in patients using CPAP for only 3.4 hours nightly.[21]

Patients with severe SAHS usually tolerate CPAP better than do milder cases because of the improvement in symptoms they experience with regular use.[22] However, all five patients in our series who could not tolerate CPAP and discontinued its use had severe disease. Most complained of nasal congestion, the discomfort of the face mask, and the inconvenience of being connected to a machine. Nasal congestion due to mouth leaks may be overcome by a carefully applied chin strap, humidification, or by using a full-face mask.

Successful weight reduction may enable the CPAP pressure to be lowered, thus improving compliance by making it more comfortable to use.[20] Discomfort from expiration against high positive pressures can be reduced by using BiPAP, although lower pressure during expiration may compromise the efficacy of the pneumatic splint and the patency of the upper airway.[23] For patients who have failed CPAP or simply cannot tolerate the device after an appropriate trial period (usually four to six weeks), surgical procedures such as UPPP and maxillofacial surgery may be beneficial in selected individuals.[24] Mandibular advancement using a dental prosthesis and various tongue-retaining devices are currently under investigation with promising results as alternative modes of treatment in mild to moderate SAHS.[25,26]

The characteristics of patients admitted for sleep studies to the Prince of Wales Hospital conform with those reported in the literature, as many of these patients were middle-aged, obese, and hypertensive. Treatment with CPAP is effective in most patients, although compliance is a major problem. Further studies on the prevalence of SAHS in Hong Kong are warranted in order to estimate the magnitude of the problem in the community. Sleep-disordered breathing is frequently underdiagnosed and, if left untreated, will remain a major cause of morbidity and mortality both on its own as well as in association with hypertension, heart failure, and vascular diseases such as ischaemic heart disease and stroke.

References

1. Soldatos CR, Lugaresi E. Nosology and prevalence of sleep disorders. Semin Neurol 1987;7:236-42.
2. Sullivan CE, Berthon-Jones M, Issa FG, Eves l. Reversal of obstructive sleep apnoea by continuous positive airway pressure applied through the nares. Lancet 1981;2:862-5.
3. Fujita S, Conway W, Zorick F, Roth T. Surgical correction of anatomic abnormalities in obstructive sleep apnoea syndrome: uvulopalatopharyngoplasty. Otolaryngol Head Neck Surg 1981;89:923-34.
4. Chiu HF, Chen CN. Sleep apnoea syndrome: a study of 5 cases. Singapore Med J 1991;31:466-8.
5. Ip M, So SY, Lam WK. Obstructive sleep apnoea syndrome: a rare entity in Hong Kong Chinese? J Hong Kong Med Assoc 1989;41:191-4.
6. Wataru H, Chiyohiko S, Shinichi O, et al. Prevalence of sleep apnoea syndrome in Japanese industrial workers using a home sleep monitor. Sleep 1993;16:126-7.

7. Grunstein RR, Lawrence S, Spies JM, et al. Snoring in paradise: the Western Samoa sleep study. Eur Respir J 1989;2(5 Suppl):4015S.
8. Koskenvuo M, Kaprio J, Partinen M, Langivainio H, Sarna S, Heikkila K. Snoring as a risk factor for hypertension and angina pectoris. Lancet 1985;1:893-6.
9. Partinen M, Putkonen PT, Kaprio J, Koskenvuo M, Hilakivi I. Sleep disorders in relation to coronary heart disease. Acta Med Scand Suppl 1982;660:69-83.
10. Partinen M, Palomaki H. Snoring and cerebral infarction. Lancet 1985;1:1325-6.
11. Waller PC, Bhopal RS. Is snoring a cause of vascular disease?: an epidemiological review. Lancet 1989;1:143-6.
12. McNamara SG, Cistulli PA, Strohl KP, Sullivan CE. Clinical aspects of sleep apnoea. In: Saunders NA, Sullivan CE, editors. Sleep and breathing: lung biology in health and disease. 2nd ed. Marcel Dekker: New York, 1994:493-519.
13. Man GC. Validation of a portable sleep apnoea monitoring device (PolyG). Proceedings of the American Lung Association and the American Thoracic Society Annual Conference; 1994 May; Boston. Boston: The American Lung Association and the American Thoracic Society, 1994.
14. Bearpark H, Elliot L, Grunstein RR, et al. Snoring and sleep apnea: a population study in Australian men. Am J Respir Crit Care Med 1995;151:1459-65.
15. Terry Y, Palta M, Dempsey J, et al. The occurrence of sleep-disordered breathing among middle-aged adults. N Engl J Med 1993;328:1230-5.
16. Ambroghetti A, Olson LG, Saunders NA. Differences in the symptoms of men and women with obstructive aleep apnoea. Aust NZ J Med 1991;21:863-6.
17. Millman R, Redline S, Carlisle C, Assaf A, Levinson P. Daytime hypertension in obstructive sleep apnoea: prevalence and contributing risk factors. Chest 1991;99:861-6.
18. Bradley T, Takasaki Y, Orr D, Popkin J, Liu P, Rutherford R. Sleep apnoea in patients with left ventricular dysfuntion: beneficial effects of nasal CPAP. In: Issa FG, editor. Sleep and respiration. New York: Wiley-Liss, Inc. 1990:363-70.
19. Rolfe I, Olsen L, Saunders N. Long term acceptance of continuous positive airways pressure in obstructive sleep apnoea. Am Rev Respir Dis 1991;144:1130-3.
20. Waldhorn R, Herrick T, Nguyen M, O'Donnell A, Sodero J, Potolicchio S. Long term compliance with nasal continuous positive airway pressure therapy of obstructive sleep apnoea. Chest 1990;97:33-8.
21. Engleman HM, Martin SE, Deary IJ, Douglas NJ. Effect of continuous positive airways pressure treatment on daytime function in sleep apnoea/hypopnoea syndrome. Lancet 1994;343:572-5.
22. Gugger M, Vock P. Effect of reduced expiratory pressure on pharyngeal size during nasal continuous airway pressure in patients with sleep apnoea: evaluation by continuous computed tomography. Thorax 1992;47:809-13.
23. Harman EM, Wynne JW, Block AJ. The effect of weight loss on sleep-disordered breathing and oxygen desaturation in morbidly obese men. Chest 1982;82:291-4.
24. Riley RW, Powell NB, Guilleminault C. Maxillofacial surgery and obstructive sleep apnoea: a review of 80 patients. Otolaryngol Head Neck Surg 1989;101:353-61.
25. Knudson RC, Meyer JB, Montalvo R. Dental prothesis for sleep apnoea patients. J Prosthet Dent 1992;68:109-11.
26. Cartwright RD, Samuelson CF. The effect of non-surgical treatment for obstructive sleep apnoea. JAMA 1982;248:705-9.