SLEEP APNOEA SYNDROME - A REPORT OF 14 CASES

T H Cheong, Y T Wang, S C Poh

ABSTRACT

Patients with sleep apnoea syndrome suffer considerable morbidity and an increased mortality. We reviewed the characteristics of 14 patients with sleep apnoea syndrome (11 males and 3 females) who were studied since 1986. All were ≤ 60 years of age with the majority in their 4th and 5th decade. Obesity was present in 8 patients (57%) and hypertension in 6 (43%).

Overnight sleep studies showed that 11 patients had obstructive sleep apnoea, 2 had central and one had predominantly mixed sleep apnoea. Ten patients (71%) had some form of nose and/ or throat pathology. Tonsillectomy seemed an effective therapeutic procedure in those with upper airway obstruction due to enlarged tonsils. Four out of 5 patients had significant symptomatic improvement post-tonsillectomy. Nasal continuous positive airway pressure was also effective in alleviating apnoeas and relieving symptoms in 4 other patients who had no obvious upper airway obstruction.

A high proportion of our patients had obstructive sleep apnoea due to enlarged tonsils. Tonsillectomy offered a simple and effective therapy for such patients. Nasal continuous positive airway pressure was also effective in the treatment of obstructive sleep apnoea.

Keywords : Sleep apnoea syndrome, obstructive sleep apnoea, upper airway obstruction, enlarged tonsils, nasal continuous positive airway pressure

SINGAPORE MED J 1990 ; Vol 31: 350 - 354

INTRODUCTION

Sleep apnoea syndrome is characterised by recurrent apnoeas and hypopnoeas during nocturnal sleep (> 5 hours of sleep) [1]. These respiratory disturbances are usually accompanied by oxyhaemoglobin desaturation and disruption of sleep pattern. In general, there are 3 main types of sleep apnoea syndromes which, according to the presence or absence of respiratory movements, are classified into obstructive, central or mixed sleep apnoea. Patients with obstructive sleep apnoea (OSA), which is the most common of the three types, suffer considerable morbidity, and recent data have supported the impression that they have an increased risk of dying as well [2,3].

Department of Medicine III
Tan Tock Seng Hospital
Moulmein Road
Singapore 1130

T H Cheong, MBBS, MRCP (UK), M Med (Int Med) (S'pore)
Registrar

Y T Wang, MBBS, MRCP (UK), AM (S'pore)
Consultant Physician

S C Poh, MBBS (Mal), AM (S'pore), FRCP (Edin)
Clinical Professor and Head

Correspondence to : Dr T H Cheong

Sleep apnoea syndrome has previously been reported in Singapore [4,5]. This paper represents our experience with 14 patients with sleep apnoea syndrome seen at the Department of Medicine (III) Tan Tock Seng Hospital since 1986.

METHODS

Patients

Fourteen patients (11 males and 3 females) underwent nocturnal sleep study in the ward. They had complained of symptoms suggestive of sleep apnoea syndrome; daytime hypersomnolence, disruptive loud snoring at night, early morning headaches and/or apnoeas during sleep observed by a bed-partner.

Baseline Evaluation

A detailed medical and physical examination were obtained in each patient. Routine spirometry and flow volume curves were performed using a dry rolling seal spirometer (Gould 900 IV, Gould Inc., Cleveland, OH). Arterial blood gas (awake) was also analysed (Radiometer ABL 30, Copenhagen).

Sleep Studies

All patients underwent overnight sleep studies. Oronasal airflow was measured by a thermistor (Somniprobe, Somnotec Inc., Van Nuys, CA). Thoraco-abdominal movements were qualitatively measured with the use of respiratory inductive phlethysmography (Respitrace

Systems, Respitrace Corp., Ardsley, N.Y.) after calibration with the isovolume manoeuvre. Arterial oxyhaemoglobin saturation ($Sa0_2$) and heart rate were measured with a pulse ear oximeter(Ohmeda Biox III, Ohmeda Corp., Boulder, CO). All variables were recorded on a multichannel recorder (Gould 2800S, Gould Inc., Cleveland, OH). The patients were observed throughout the night by an attending doctor and was asked the next morning if he slept during the night. The sleep study was repeated if the patient claimed or was observed to have slept poorly during the study.

In addition, sleep studies were repeated in 6 subjects who had specific therapy; four with the use of nasal continuous positive airway pressure(nCPAP) and 2 after tonsillectomy. Nasal CPAP was provided with a Sleep Easy II system (Respironics Inc., Monroeville, PA) through a tightly fitting nasal mask. The airway pressure was monitored with a calibrated pressure transducer (Validyne MP45-871, Northridge, CA) connected to a port of the nasal mask by rigid tubing. Positive airway pressure titration was performed in order to determine the airway pressure which alleviated the apnoeas.

Table I

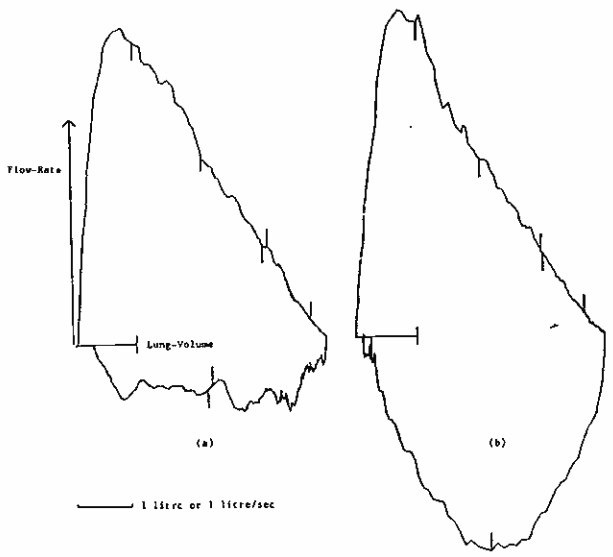
Physical characteristics and blood gas results for 14 patients with sleep apnoea syndrome.

	Mean ± S.E.M	Range
Male : Female	11 : 3	-
Age	37±3	13 - 60
B.M.I.*	34 ± 2	22 - 45
pH	7.38 ± 0.01	7.34 - 7.43
PaO_2	74 ± 4	42 - 96
Pa CO_2	45 ± 2	35 - 58
FEV_1/FVC (%)	85 ± 2	74 - 94
Haemoglobin (g)	15.7 ± 0.6	10.7 - 18.6

* B.M.I = body mass index (weight (kg)/height^2 (m^2))

Fig 1

Flow-volume loop of one patient showing typical upper airway obstructive pattern. Y-axis denotes flow-rate and X-axis the lung volume. Expiratory loop is above the X-axis while the inspiratory loop is below it. Note the 'flattened' inspiratory loop before tonsillectomy (a), and the improvement seen after operation (b).



Analysis of Sleep Data

Apnoea was defined as the cessation of oro-nasal airflow for at least 10 seconds in duration. Obstructive apnoea was defined as the cessation of oro-nasal airflow despite the presence of respiratory movements as measured by inductive plethysmography while central apnoea was the absence of both oro-nasal airflow and respiratory movements. Mixed apnoea was defined as the occurrence of central apnoea early in the episode of apnoea followed by obstructive apnoea later in the episode. A hypopnoea was scored qualitatively if there was a reduction of oro-nasal airflow (compared to the baseline thermistor output) accompanied with a desaturation of ≥ 4%. The apnoea/hypopnoea index (AHI) was derived from the ratio of the total number of apnoeas/ hypopnoeas observed to the total sleep time in hours. Total sleep time was estimated from the total bedtime. Allowing for potential difference between the 2 values, the calculated AHI would underestimate rather than overestimate the value.

Other indices measured included the maximum desaturation (the difference between the highest and lowest SaO_2 values during an apnoeic spell), the lowest SaO_2 reached, and the maximum duration of apnoea

Table II

Sleep parameters for 14 patients with sleep apnoea syndrome before therapy

	Mean ± S.E.M.	Range
Total no. of apnoeas/hypopnoeas	433 ± 62	83 - 935
Sleep time (h)	7.5 ± 0.2	6 - 9
A. H. I.	58 ± 8	12 - 117
Maximum duration of apnoea (s)	47 ± 6	20 - 90
Maximum desaturation (%)	33 ± 4	10 - 50
Lowest SaO_2(%)	58 ± 6	12 - 86

Fig 2

Flow-volume loop of one patient. Note the 'saw-tooth' pattern along the expiratory loop (arrow).

Table III
Effect of tonsillectomy on two patients with obstructive sleep apnoea syndrome.

	Patient 1 (TPS) before	(after)*	Patient 2 (AB) before	(after)*
Total no. of apnoeas	581	*(99)*	433	*(98)*
A.H.I	89	*(15)*	60	*(15)*
Maximum Desaturation (%)	50	*(16)*	50	*(30)*
Lowest SaO_2 (%)	42	*(78)*	12	*(60)*
Maximum duration of apnoea	63	*(28)*	50	*(100)*

* *(numbers)* refer to corresponding data after tonsillectomy

(Table III). All results were reported as mean ± standard error of the mean (S.E.M.).

RESULTS

Baseline Evaluation

The mean age of the patients was 36 ± 3 years (S.E.M.) with the majority in their 4th or 5th decade. One patient was a 13 year old girl who had lymphomatous infiltration of the tonsils. Eight patients (57%) were obese as they had a body mass index >30 [weight (kg)/height^2 (m^2)] (6). Five patients (36%) had hypertension while three (21%) (2 of whom had hypertension) had diabetes mellitus. All patients had daytime hypersomnolence and 3 of them had been involved in road traffic accidents as a consequence of their falling asleep while driving.

The mean values for arterial blood gas analysis and spirometry were within normal limits (Table I). Three male patients were hypercapnic at rest ($PaCO_2$ of 53.6, 55.2 and 58 mmHg). Two of them were obese and one of them was also polycythaemic with a haemoglobin of 18.6 g. Ten patients (71%) had some form of nasal and/or throat pathology eg. nasal obstruction, deviated nasal septum, nasopharyngeal cyst or enlarged tonsils. Four such patients had grossly enlarged tonsils described by the otolaryngologist as 'kissing tonsils'. Seven patients showed an upper airway obstructive pattern in their flow volume curves (Fig 1). A 'saw-tooth' pattern was seen in the expiratory limbs of the flow-volume curves of 2 patients (Fig 2). This sign, once previously described as typically seen in OSA [7] was later found to be both insensitive and nonspecific for OSA.

Sleep Studies Analysis

Sleep studies revealed that 11 patients (79%) had obstructive, 2 had central and one, mixed sleep apnoea syndrome. One of the 2 patients with central sleep apnoea had idiopathic cerebellar degeneration and epilepsy while 2 patients with OSA had suffered from previous cerebrovascular accidents.

On the average, the patients slept 7.5 ± 0.2 h and had 83 to 935 apnoeas/ hypopnoeas during the night (Table II). The mean AHI was 58 ± 8 and varied individually from 12 to 117 during the sleep study. In general, patients with obstructive sleep apnoea had a higher apnoeic index than those with other types of apnoea. The maximum duration of apnoea ranged from 20 to 90 seconds and the maximum desaturation averaged 33 ± 4% (Table II). Two patients had a fall of 50% points in SaO_2 during apnoeas. The mean of the lowest O_2 saturation observed was 58 ± 6% with the minimum at 12% in one patient. However, due to technical limitations, oximeter readings < 50% may not be accurate [8]. Nevertheless, it did show that the patient was severely hypoxaemic during the apnoeas. In general, patients with OSA had longer apnoeas and greater desaturation than the rest.

Effect of Therapy

As the upper airway obstruction was likely to be due to the enlarged tonsils in some patients with OSA, tonsillectomy was offered to those patients with this problem. Of the 5 patients who had tonsillectomy, 3 had marked symptomatic improvement while one died. This unfortunate patient was an obese diabetic who collapsed in the recovery room after the operation. The postmortem did not identify the specific cause of death and his death was attributed to 'cardiorespiratory failure'.

We studied the effect of tonsillectomy in 2 patients by repeating sleep studies post-operatively. Marked improvement was seen in the parameters measured, although in one patient (AB) the maximum duration of apnoea was increased post-tonsillectomy (Table III). This patient had obstructive apnoeas pre-operatively, and demonstrated mostly hypopnoeas after the operation. Although some of these episodes were longer in duration, oxyhaemoglobin desaturation was significantly less. Even though apnoeic activity was still present post-operatively, these 2 patients were clinically well and no longer complained of the disabling hypersomnolence.

Sleep studies with the use of nCPAP were repeated in 4 patients with OSA. Dramatic improvements were noted in the parametres measured (Table IV). Apnoeas were significantly reduced in numbers ($p < 0.05$, paired t- test) with concomitant improvement in oxygenation, ie. less desaturation. Mean airway pressure required to alleviate the apnoeas/ hypopnoeas was 10 cmH_2O. One patient who subsequently bought a nCPAP machine experienced significant improvement in her condition with daily nocturnal usage.

Table IV
Sleep parameters before and with nCPAP usage in 4 patients with obstructive sleep apnoea.

Patient	A.H.I	Maximum desaturation (%)	Lowest SaO_2 (%)	Maximum duration of apnoea	Airway pressure cm H_2O
YSK	36 *(5.4)**	40 *(20)*	32 *(68)*	90 *(50)*	10
IS	117 *(3.6)*	26 *(20)*	52 *(74)*	30 *(20)*	8
HAK	50 *(5)*	48 *(12)*	44 *(84)*	60 *(40)*	7
SCS	63 *(18)*	26 *(6)*	66 *(90)*	80 *(20)*	15
Mean	67 *(8)*	37 *(15)*	54 (74)	65 *(33)*	10

* *(numbers) refer to corresponding data with nCPAP usage*

One of the two patients who had central apnoea died in his sleep while in the hospital before any treatment could be given. This patient had epilepsy, idiopathic cerebellar degeneration and was hypercapnic at rest. No postmortem was done. Two patients (one with central and the other with mixed sleep apnoea) defaulted from follow-up and their outcomes are unknown.

DISCUSSION

The physical characteristics and the distribution of the different types of sleep apnoea syndrome in our series of patients are in agreement with most other published series [1]. The male preponderance and the more common occurrence of OSA are well recognised [1,9,10]. Two of the 3 female patients were post-menopausal while the third was a 13 year old girl with lymphoma involving the tonsils. Excessive daytime sleepiness and loud nocturnal snoring were outstanding symptoms. Indeed 2 of our patients were involved in multiple road traffic accidents because they fell asleep while driving. Interestingly, most of our patients had accepted their chronic symptoms as quite 'normal' and therefore had delayed seeking medical attention.

Two obese male patients were hypercapnic at rest and one of them was also polycythaemic. The FEV_1/FVC ratios were normal in these 2 patients. They could have had 'Pickwickian' syndrome with central hypoventilation. Their ventilatory response to hypercapnia however was not done. Obesity was common in our patients. It has been reported that obesity was associated with a more severe form of disease [11]. Although moderate weight loss has been shown to be effective in treating OSA [12], we encountered problems in trying to motivate our obese patients to lose weight.

Sleep studies showed that most of our patients had severe OSA (Table II) with many apnoeas and associated severe desaturation. Those patients with OSA tended to be more obese and had longer apnoeas and greater desaturation than those who had predominantly central sleep apnoea. The recurrent hypoxaemic episodes in OSA patients have been shown to increase blood pressure as well as precipitating cardiac arrhythmias and conduction disturbances [13]. The frequent association between hypertension and OSA was also observed in this series as 5 patients (36%) had elevated blood pressure. The prevalence of systemic hypertension in OSA has been reported to range between 48 and 96% [14-16]. Likewise, studies have shown that as many as 30% of hypertensive patients have significant sleep apnoea [17].

An interesting feature in this series was the high incidence of nasal and/ or throat pathology. Seventy-one per cent of the patients had some form of nasal and/or throat problem and 8 of the 11 patients(73%) with OSA had enlarged tonsils. The tonsils may have contributed towards the upper airway obstruction and the abnormal flow volume loops (Fig 1). We speculate that this unusually high incidence of upper airway pathology is due to the lower incidence of morbid obesity in our population compared to that in the West. The pathogenesis of obstructive sleep apnoea is probably multifactorial but occurs particularly in the setting of obesity and compromised upper airway anatomy. If obesity is less common in our population, then upper airway disease would appear to be a commoner cause of obstructive sleep apnoea. Other than a small series of 6 patients with OSA due to enlarged tonsils [18], most series published do not seem to encounter such a high incidence of upper airway problems [19]. Due to high incidence of enlarged tonsils in our patients, tonsillectomy was offered as a therapeutic measure. It proved effective in relieving symptoms and reducing the number of apnoeas as well as improving oxygenation (Table III). Tonsillectomy is a much simpler and less extensive operation than uvulopalato-pharyngoplasty, which is often performed in the West as a surgical therapy for OSA [20].

Two deaths were encountered in our series. One was an obese patient with OSA who died in the recovery room after tonsillectomy. Postmortem did not show any obvious cause although respiratory embarrassment was suspected as the patient was already extubated. The other fatality was a patient who had central sleep apnoea and he died in his sleep. He may have succumbed to a period of prolonged apnoea or a fatal episode of cardiac arrhythmia. Such arrhythmias have been known to occur during apnoea [13,19]. Although the natural history of this condition is not entirely clear, recent data have appeared which supports the impression that patients with obstructive sleep apnoea syndrome have an increased risk of dying [2, 3]. Therefore specific therapy with either tonsillectomy in those with enlarged tonsils or with nCPAP are important in OSA. Daytime hypersomnolence by itself is severely disabling and warrants treatment [10].

Since its introduction in 1981 [21], nCPAP has been proven to be effective in the treatment of OSA. By acting as a pneumatic splint, the upper airway is kept patent throughout respiration. This beneficial effect was clearly demonstrated in the 4 patients in which nCPAP was tried. Total number of apnoeas and oxygenation were improved significantly and symptoms were relieved. The reason for residual apnoeic activity (Table IV) during nCPAP usage was because nCPAP titration was performed in the early part of the sleep study and some apnoeas were inevitably present during this manoeuvre. These apnoeas were included in the analysis of the sleep recordings. The beneficial effect of nCPAP in improving survival in patients with OSA was recently reported [2].

In conclusion, the sleep apnoea syndrome like many other medical conditions can be diagnosed only if one is aware of its symptom complex. The following features should alert one to the possibility of the sleep apnoea syndrome :

1) daytime hypersomnolence
2) loud nocturnal snoring
3) male sex
4) upper airway pathology
5) obesity
6) hypertension.

If these features are present, the patient should undergo nocturnal polysomnography which is diagnostic and allows assessment of the severity of condition.

Although our series was small, what was interesting was the high incidence of nasal and/or throat pathology and the likely role that these played in the pathogenesis of the upper airway obstruction during sleep. At the time of writing, we have another 16 patients with OSA that have recently been studied and are undergoing therapy. A high incidence of nasal and throat pathology is also noted in them and we hope to report on these patients in the near future.

REFERENCES

1. Guilleminault C, Tilkian A, Dement WC: The sleep apnoea syndromes. Ann Rev Med 1976; 27: 465-84.
2. He J, Kryger MH, Zorick FJ, Conway W, Roth T: Mortality and apnoea index in obstructive sleep apnoea. Chest 1988; 94: 9-14.
3. Partinen M, Jamieson A, Guilleminault C. Long term outcome for obstructive sleep apnoea syndrome patients: mortality. Chest 1988; 94; 1200-4.
4. Lim TK. The obstructive sleep apnoea syndrome: Case studies. Ann Acad Med 1985; 14: 435-8.
5. Wang YT, Tan TH, Choo MH, Chan RKC. Tonsillectomy relieves hypersomnolence. Singapore Med J 1986; 27: 347-9.
6. Bray GA. Definitions, measurements and classification of the syndromes of obesity. Int J Obes 1978; 2: 99-112.
7. Sanders MH, Martin RJ, Pennock BE, Rogers RM: The detection of sleep apnoea in the awake patient: The 'saw-tooth' sign. JAMA 1981; 245: 2414-8.
8. Ohmeda Biox III pulse oximeter operating/service manual. Boulder, CO., Ohmeda Corp. 1983:50.
9. Kales A, Vela-Bueno, Kales JD: Sleep disorders: Sleep apnoea and Narcolepsy. Ann Intern Med 1987; 106: 434-43.
10. Weil JV, Cherniak NS, Dempsey JA et al: Respiratory disorders of sleep. Am Rev Respir Dis 1987; 136: 755-61.
11. Sullivan CE, Issa FG: Obstructive sleep apnoea. In: Kryger MH ed. Clin Chest Med 1985; 6: 633-50.
12. Suratt PM, McTier RF, Findley LJ, Pohl SL, Wilhoit SC: Changes in breathing and the pharynx after weight loss in obstructive sleep apnoea. Chest 1987; 92: 631-7.
13. Shepard JW. Cardiopulmonary changes in obstructive sleep apnoea. In: Kryer MH, Roth T, Dement WC, eds. Principles and practice of sleep medicine. Philadelphia: WB Saunders Co, 1989: 537-55.
14. Burack B, Pollak C, Borowiecki et al. The hypersomnia-sleep apnoea syndrome (HSA): a reversible major cardiovascular hazard. Circulation 1977; 56: 177.
15. Guilleminault C, van den Hoed J, Mitler MM. Clinical overview of the sleep apnoea syndromes. In: Guilleminault C, Dement WC eds. Sleep apnoea syndromes. New York: Alan R Liss, 1978: 1-12.
16. Shepard JW Jr, Garrison MW, Grither DA et al. Relationship of ventricular ectopy to nocturnal 0_2 desaturation in patients with obstructive sleep apnoea. Chest 1985; 88: 335-40.
17. Kales A, Bixler EO, Cadieux RJ, Schneck DW et al: Sleep apnoea in a hypertensive population. Lancet 1984; ii: 1005-8.
18. Orr WC, Martin RJ: Obstructive sleep apnoea associated with tonsilar hypertrophy in adults. Arch Intern Med 1981; 141: 990-2.
19. Guilleminault C, Connolly S, Winkle RH: Cardiac arrhythmia and conduction disturbances during sleep in 400 patients with sleep apnoea syndrome. Am J Cardiol 1983; 52: 490-4.
20. Strohl KP, Cherniack NS, Gothe B. Physiologic basis of therapy of sleep apnoea. Am Rev Respir Dis 1989; 134: 791-802.
21. Sullivan CE, Issa FG, Berthon-Jones M, Eves L. Reversal of obstructive sleep apnoea by continuous positive airway pressure applied through the nares. Lancet 1981; 1: 862-5.