NASAL CPAP THERAPY IN THE OBSTRUCTIVE SLEEP APNOEA SYNDROME

T K Lim

SYNOPSIS

I evaluated the use of nasal continuous positive airway pressure (Nasal CPAP) to treat nine patients with the obstructive sleep apnoea syndrome. There were 8 Chinese and one Indian man. Nasal CPAP was applied using a nasal mask and variable positive end expiratory pressure valve (Sleep-easy Respironics Inc.) in single overnight studies after 3 to 4 hours of baseline recording. Sleep events were monitored with ear oximetry (Biox IIA) and impedance plethysmography (Respisomnograph). Eight out of nine patients achieved quiet sleep and regular breathing at appropriate levels of nasal CPAP (8 to 15 cm H_2O). One patient could not tolerate nasal CPAP. The pitfalls and limitations in the use of nasal CPAP for the treatment of sleep apnoea are discussed.

SING MED J. 1988; 29:157–163

INTRODUCTION

The obstructive sleep apnoea (OSA) syndrome is a well defined group of disorders of breathing during sleep characterised by repeated episodes of upper airway obstruction associated with loud snoring, arterial desaturation and resulting in sleep fragmentation and daytime somnolence (1,2). The upper airway in patients with OSA is narrowed and functionally unstable, it tends to collapse during sleep when fall in pharyngeal muscular tone results in loss of wall rigidity and distending pressure.

Sullivan et al introduced a novel and ingenious method to maintain upper airway patency in patients with severe OSA by applying nasal continuous positive pressure (CPAP) with a tight fitting custom-made nasal mask(3). Nasal CPAP have been used as an effective modality for the long term treatment in many patients with OSA with good results(4).

While polysomnography is the 'gold standard" technique for the laboratory assessment of the OSA syndrome, there is still no agreement on what is the most cost-effective approach to the clinical evaluation of this syndrome and moreover, there is even less agreement on the application of nasal CPAP in its management.

In this paper, I describe the use of nasal CPAP therapy in a group of 9 Oriental patients with OSA. The clinical data and sleep observations of 5 subjects had been described in a previous report(5).

Department of Medicine
National University Hospital
Kent Ridge
Singapore 0511

T K Lim, M Med (Int Med)
Lecturer

PATIENTS AND METHODS

All patients had clinical histories, physical features, pulmonary function abnormalities and overnight studies of respiratory movement and ear oximetry which were characteristic of the OSA syndrome(1,2,5). The clinical features, awake pulmonary function data and overnight studies of 5 of the 9 patients had been described in a previous report(5). Repeat sleep studies in these 5 subjects showed oxygen desaturation on ear-oximetry of either similar or even greater severity when compared to records made 2 years previously.

Patients were admitted to the laboratory for a single overnight study to evaluate the use of Nasal CPAP. Baseline recordings of ribcage and abdominal wall motion (Respisomnograph Inc.) and ear oximetry (Biox IIA) were made during the first 3 to 4 hours of sleep to establish the presence of repeated apneoas/hypopneas, loud snoring, paradoxical breathing and significant arterial desaturation, this was followed application of CPAP from a tight-fitting nose mask. The CPAP was delivered from a high-flow low-pressure blower (Sleep-easy, Respironics) via a t-piece. The pressure was continuously monitored by a water manometer and regulated by a positive end-expiratory pressure valve until snoring ceased and quiet, regular breathing ensued. This was usually established in about 10 to 15 minutes and with a CPAP of about 10 to 15 cm H_2O. After another 3 to 4 hours of stable sleep recording on nasal CPAP, the airway pressures were gradually reduced until loud snoring and arterial desaturation was again noted.

The Respisomnograph system used to monitor breathing patterns was a microprocessor based system which identified the apnoeic or hypopnoeic events and provided a graphic record.

The author was attendent at bedside through out most of the recording periods and especially when CPAP was in use. Observer fatigue precluded consecutive night studies.

Table 1
ANTHROPOMETRIC DATA

Subjects No.	Name	Age (yrs)	Height (cm)	Weight (kg)
1.	LCA*	49	164	96
2.	PJ*	47	165	82
3.	PKS*	39	171	110
4.	SSH*	35	178	123
5.	TT*	53	168	122
6.	YSK	58	156	66
7.	LAH	55	165	77
8.	CKC	30	163	83
9.	PHY	60	163	68

*Clinical and sleep data described in reference 5

RESULTS

All 9 patients were middle aged men, their anthropometric and pulmonary function data are shown in table 1. They gave typical histories of loud snoring during sleep and daytime excessive somnolence. The clinical and sleep observations of the first five patients have been described in more detail in a previous report(5). The results of diagnostic sleep studies are in table 2. All patients demonstrated the characteristic features of obstructive sleep apnoea with repeated episodes of arterial desaturation and loud snoring associated with cycles of paradoxical breathing during apnoeic or hypopnoeic periods followed by hyperventilation(1,2).

Breathing Patterns

Figure 1 shows the records of breathing patterns during sleep for patient #2. Each record was an epoch of 5 minutes, the time was indicated on upper right of each panel and the mean percentage oxygen saturation (% sat.) was noted on upper left. Single breaths were represented by the vertical lines and they end on the continuous line which represented the level of the end-tidal position. The discontinuous line at the bottom of each panel indicated paradoxical breathing if the value recorded fell below 0.
Note in figure 1 (upper panel) while the patient was asleep and snoring loudly, that episodes of hypopnoea was accompanied by variation in end-tidal lung volume, paradoxical breathing and arterial desaturation. When nasal CPAP of + 10 cm H_2O was applied (lower panel) quiet regular breathing was observed with no signs of variable end-tidal lung volume, respiratory paradox or fall in oxygen saturation.

Figure 2 shows the breathing pattern records of patient #4 who had classical obstructive apnoea and severe oxygen desaturation (upper panel) which was completely controlled by nasal CPAP of + 13 cm H_2O (lower panel).
Similar features may be noted in the recordings of patient #7 in figure 3. He showed a mixed pattern of apnoea and hypopnoea which was well controlled by nasal CPAP of + 8 cm H_2O.

Ear-Oximetry

Figure 4 shows a continuous overnight recording of percent oxygen saturation by ear-oximetry in patient #3. It runs from right to left and was started at 2200H. Nasal CPAP was applied after about 2.5 H. of baseline recording which showed the typical repeated "dips" in oxygen saturation which had been described in severe obstructive sleep apnoea. During the 3 H. of the nasal CPAP therapy oxygen saturation was well maintained at about 94%. Note in addition, that oxygen desaturation may be paradoxically more severe during application of nasal CPAP (⬇) this was obviated at higher levels of airway pressure.

Figures 5 to 8 are recordings of overnight oxygen saturation with and without nasal CPAP in patients #1, #7, #8 and #6 respectively. They show that nasal CPAP effectively prevented any episodes of arterial desaturation in all subjects.

As shown in table 2, 8 out of 9 patients tolerated levels of nasal CPAP which maintained patency of airways in sleep and achieved normal breathing and oxygen saturation. Only the last patient could not sleep with the nose mask because he felt suffocated. he was also the oldest person in this group. Patients #1 and #6 have responded well to nasal CPAP at home.

DISCUSSION

The syndrome of obstructive sleep apnoea is caused by repeated episodes of upper airway occlusion at the pharyngeal level during sleep accompanied by pro found falls in arterial oxygen tension and arousal from sleep. While many factors may be implicated in the production of upper airway occlusion, therapeutic maneuvers have aimed at increasing the size and or the stability of the oro-naso-pharynx during sleep(6).

Simple measures such as weight loss, surgery for nasal occlusion or tonsillar enlargement and avoidance of depressant drugs and alcohol may alone be adequate for some patients. The majority will need some form of specific treatment for OSA.

Positive airway pressure applied through the nose is a proven way of eliminating upper airway obstruction in the OSA syndrome and have been successfully used in many centres in long-term treatment. This report confirms its effectiveness in Oriental patients and at the same time points out some of the problems which may be encountered in its application.

The optimum airway pressure varies widely between patients and must be established individually in overnight studies (table 2). Inadequate pressures may result in enhanced airway instability and greater desaturation (figure 4). A leak in the nose mask may precipitate airway collapse which may be hazardous as these patients fall into a 'rebound" REM or unarousable sleep during the first few nights on CPAP(3).

While no deaths have so far been reported in the use of CPAP, severe desaturation had occurred in older delivery systems which had high impedance(6). Moreover, higher pressures may be required when the patient enters REM sleep. This might account for the brief unstable episode encountered in patient #1, figure 5.

I conclude that while nasal CPAP is effective therapy for the obstructive sleep apnoea syndrome in Oriental patients, the appropriate pressures needed must be established in careful overnight studies with continuous monitoring of the patient. Indications for the use of nasal CPAP in mild disease and its place in long term treatment need to be determined by further studies.

Fig. 1. Breathing pattern recordings in patient #2. Showing obstructive hypopnoea and its control with nasal CPAP. See text for detailed description.

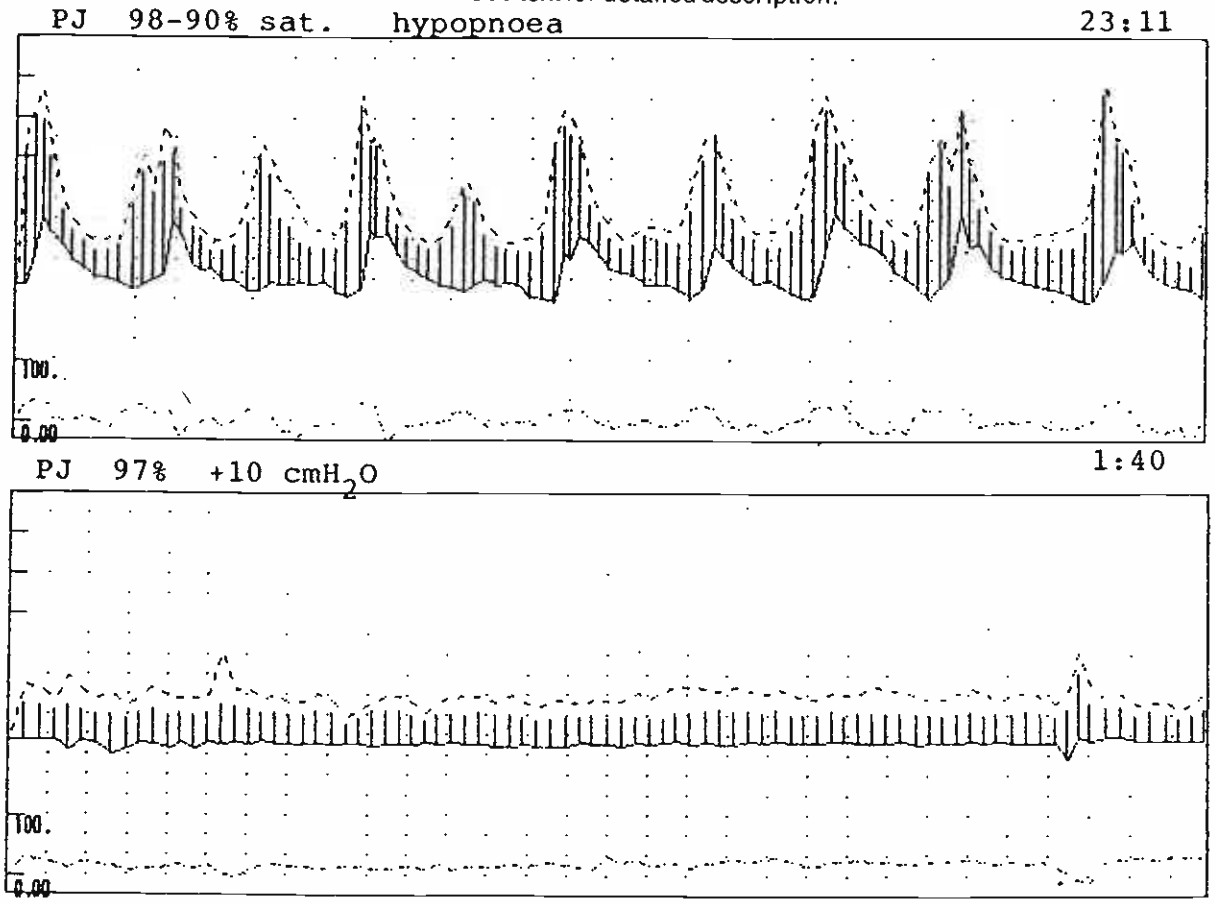


Fig. 2. Breathing pattern recordings in patient #4.

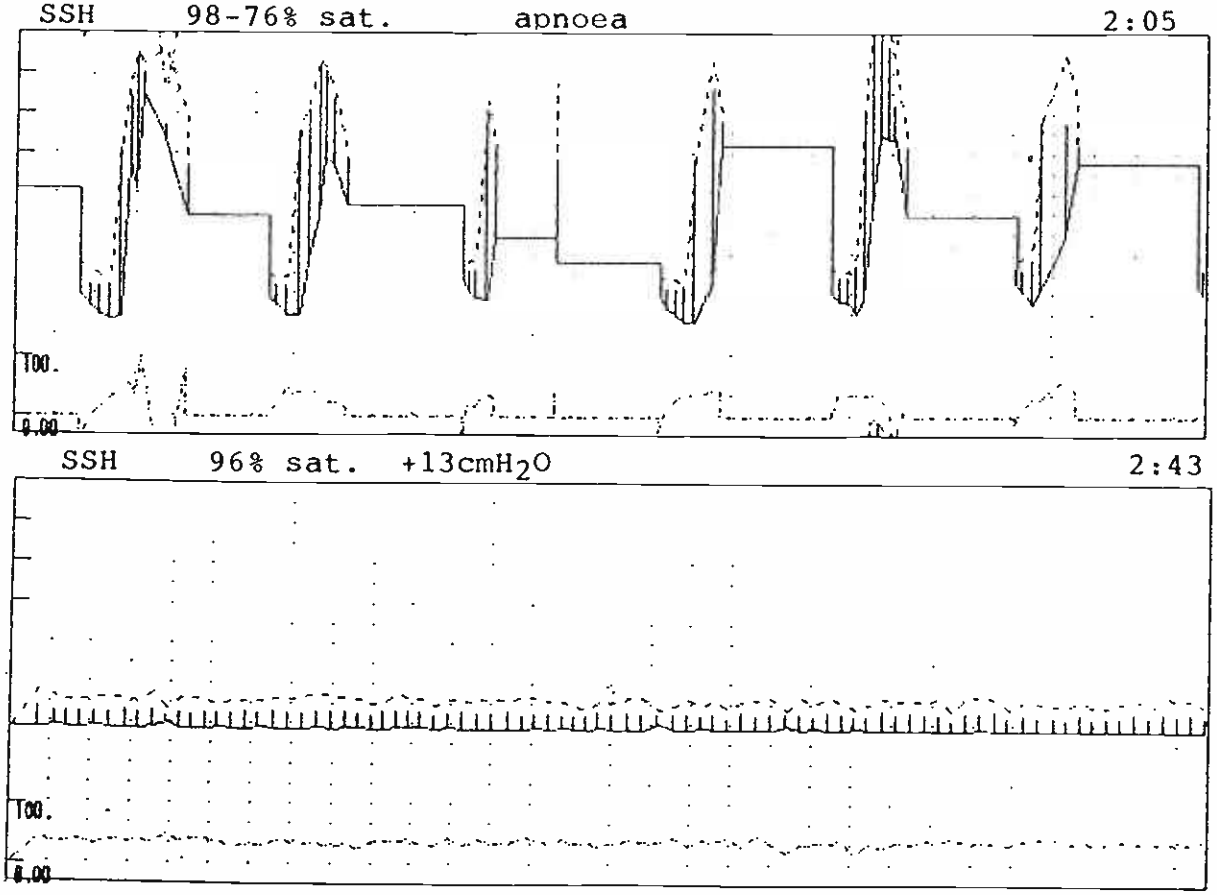


Fig. 3. Breathing pattern recordings in patient #7.

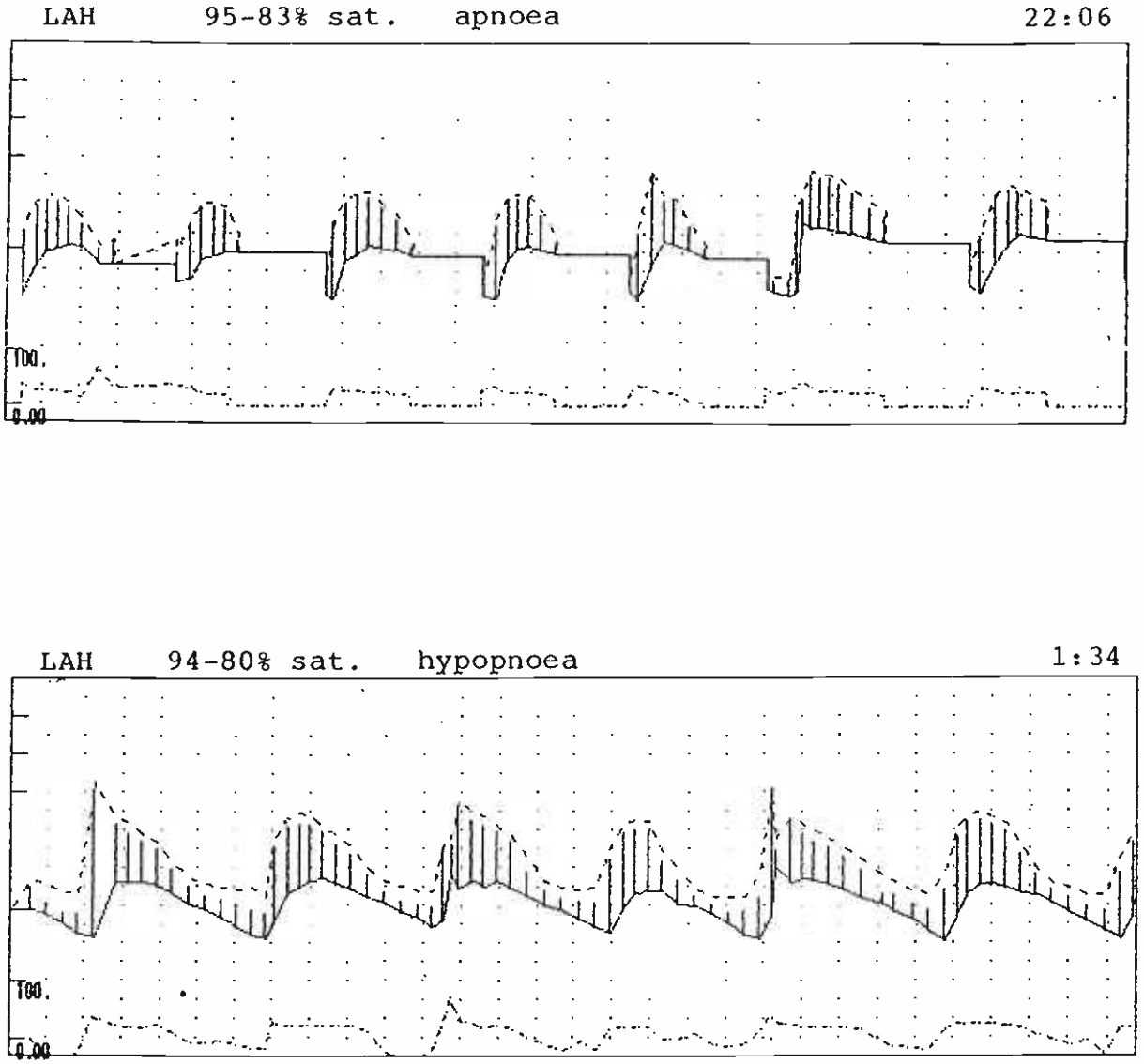


Fig. 4. Overnight recording of percent oxygen saturation (sat) in patient # 3. It was started at 2200 H and runs from right to left. The hatched bar indicate use of nasal CPAP. Note more severe desaturation (⬇) when in adequate airway pressure was applied.

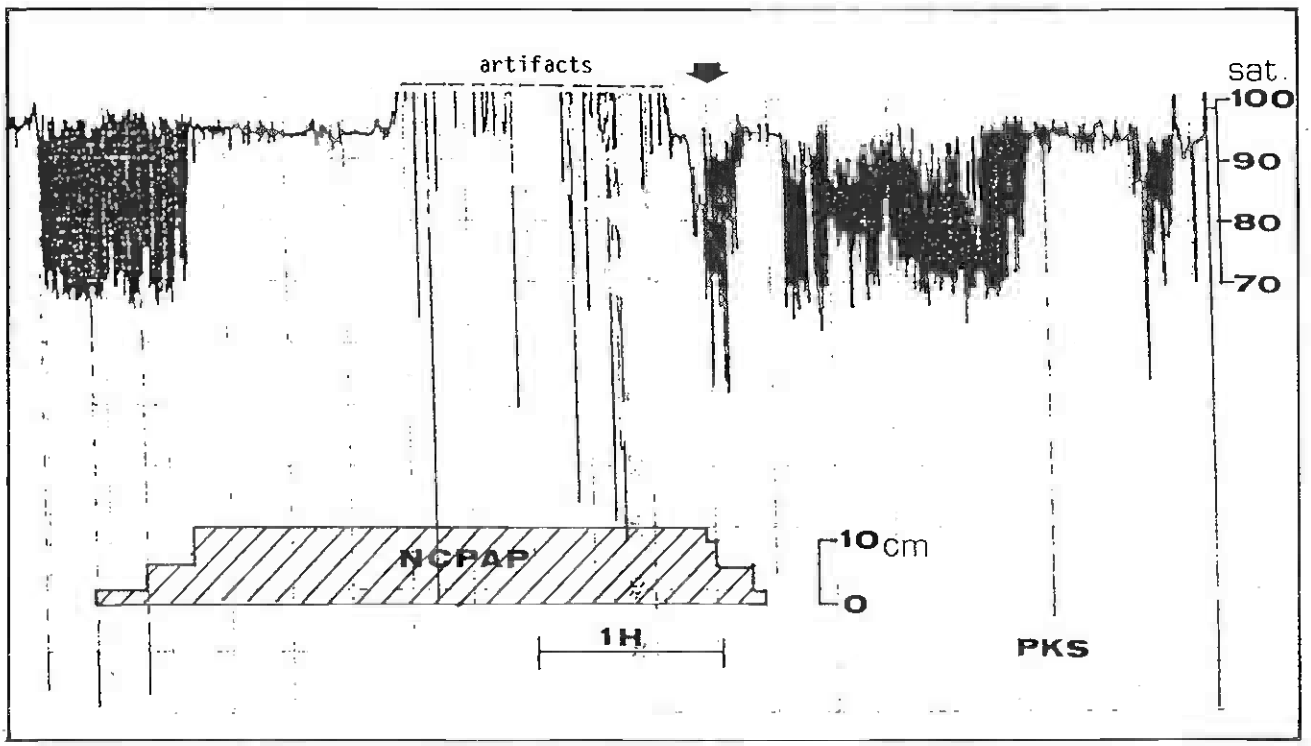


Fig. 5. Ear oxi-meter recording in patient # 1. See comments on this patient under "DISCUSSION".

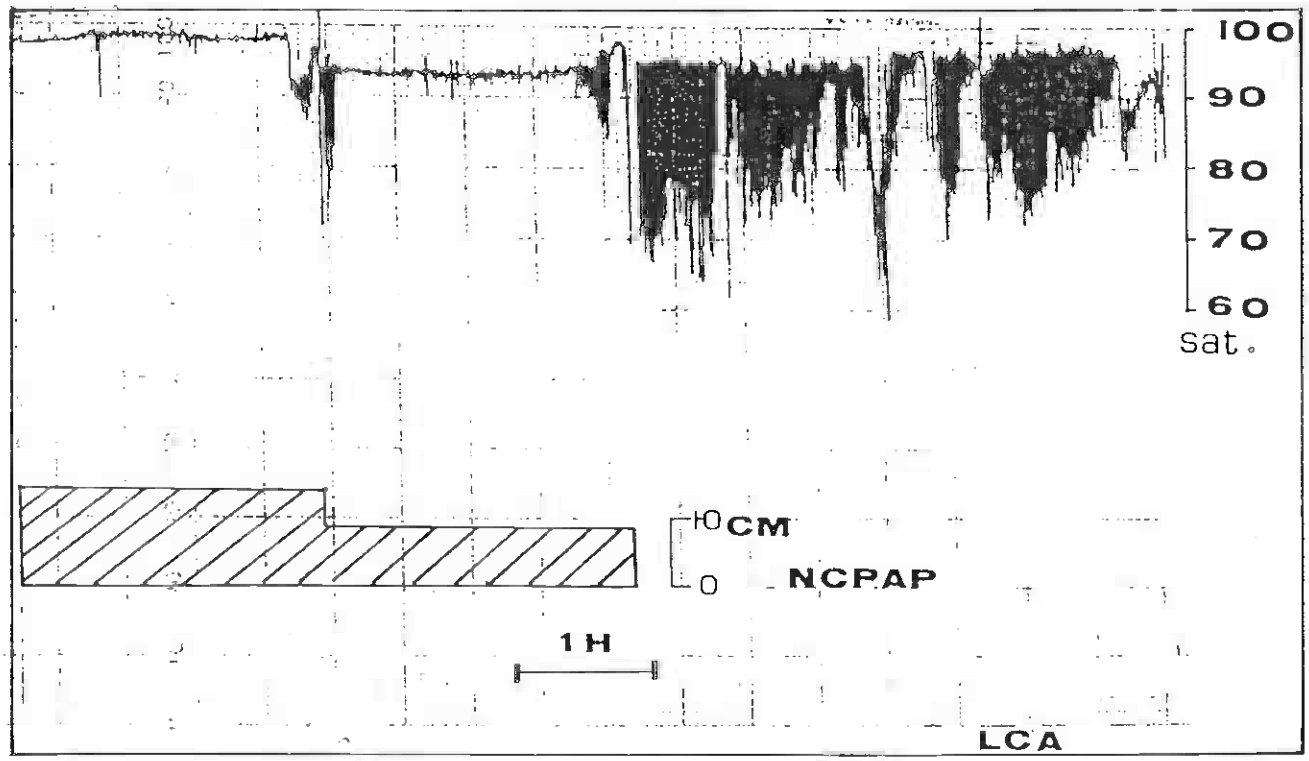


Table 2
SLEEP DATA

Subject No.	Min. O_2 Sat(%)	ΔO_2 Sat(%)	Apnoea per H.	NCPAP cmH_2O
1.	70	95 - 68	120	10
2.	88	98 - 90	126	10
3.	56	96 - 67	80	12.5
4.	62	96 - 76	66	13
5.	58	94 - 68	60	15
6.	58	95 - 66	68	10
7.	82	94 - 82	76	8
8.	72	95 - 76	120	15
9.	78	94 - 85	43	--

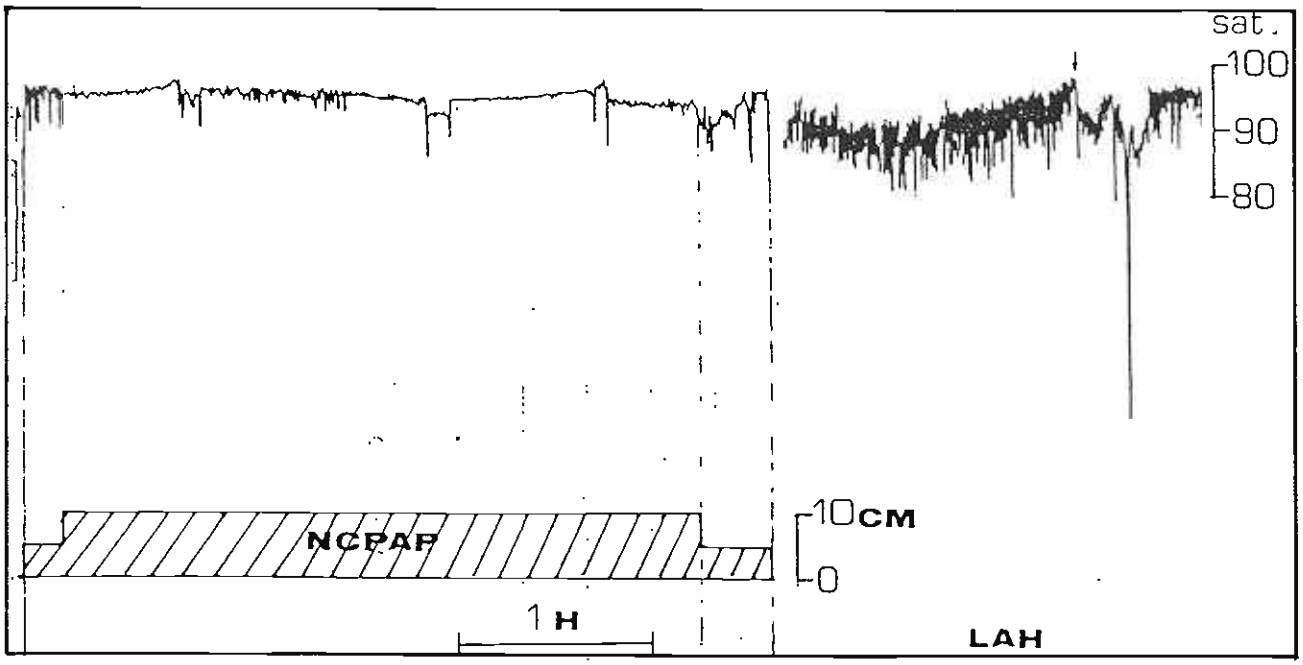
Min. O_2 : lowest saturation point

ΔO_2 : average fall in arterial saturation during apnoea or hypopnoea

Apnoea : mean numbers of apnoea or hypopnoea per hour.

NCPAP : optimum airway pressure for quiet breathing.

Fig. 6. Ear oxi-meter recording in patient # 7.



ACKNOWLEDGEMENT

I would like to thank the various colleagues who referred patients for sleep study and Ms Siti Dahniar for expert typing of the manuscript.

Fig. 7. Ear oxi-meter recording in patient # 8.

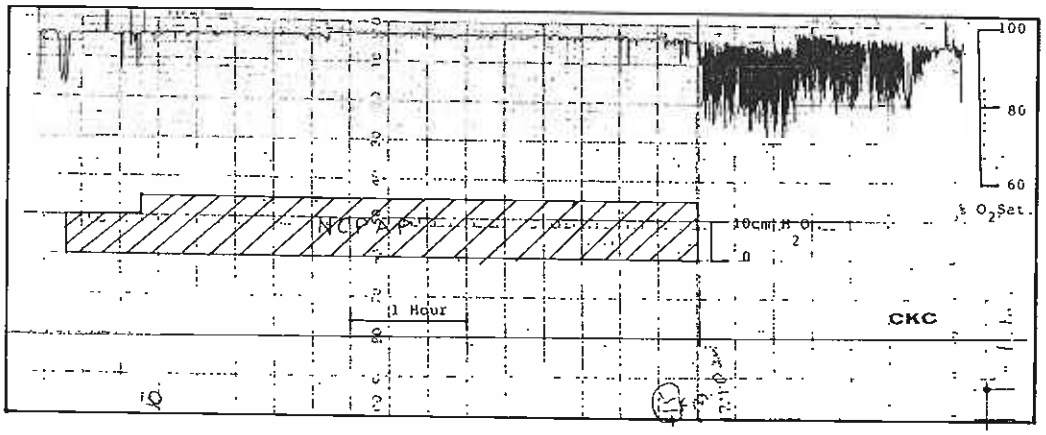
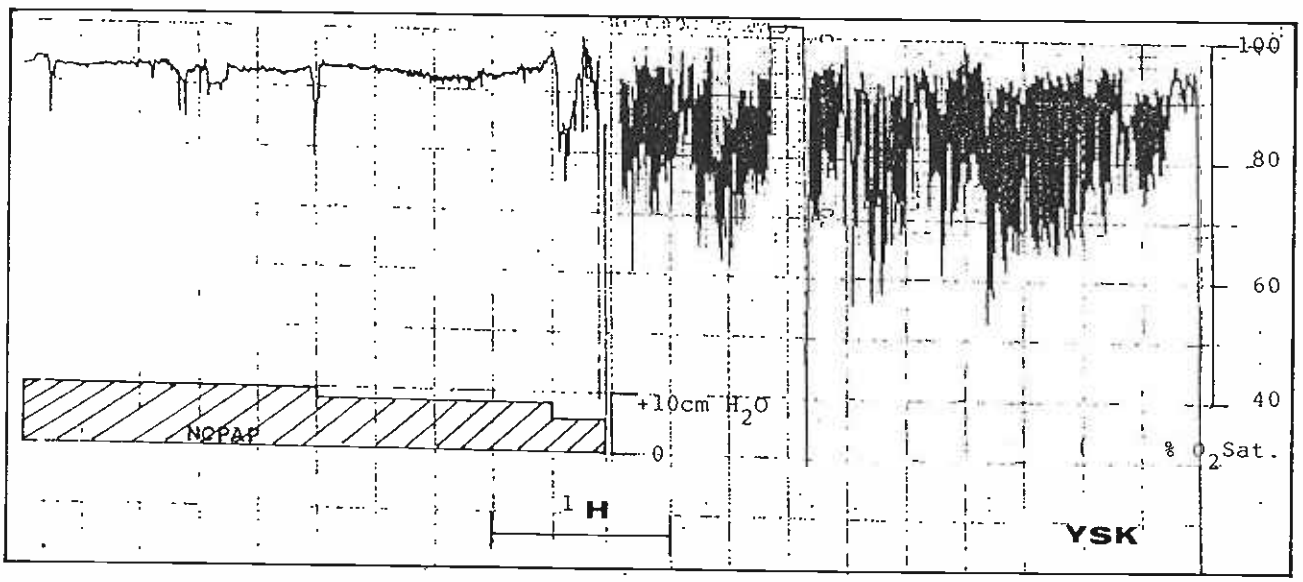


Fig. 8. Ear oxi-meter recording in patient # 6.



REFERENCES

1. Guilleminault C, Tikian A, Dement WC. The sleep apnoea syndromes. Annu Rev Med 1976; 27:465–84.
2. Strohl KP, Saunders NA and Sullivan CE. Sleep Apnoea Syndromes. In: Sleep and Breathing eds. Saunders NA and Sullivan CE. Marcel Dekker, N.Y. 1984; 365–402.
3. Sullivan CE, Berthon-Jones M, Issa FG, Eves L. Reversal of obstructive sleep apnoea by continuous positive airway pressure applied through the nose. Lancet 1981; 1:862–66.
4. Rapoport DM, Sorkin B, Garay SM and Golding RM. Reversal of the "pickwickian syndrome" by long term use of nocturnal nasal-airway pressure. N Engl J Med 1982; 38:237–41.
5. Lim TK and Tan WC. The obstructive sleep apnoea Syndrome: Case studies. Ann Acad Med Singapore 1985; 14:435–8.
6. Strohl KP, Cherniak NS and Gothe B. Physiologic basis of therapy for sleep apnoea. State of Art. Am Rev Respir Dis 1986; 134:791–802.