TONSILLECTOMY RELIEVES HYPERSOMNOLENCE

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SYNOPSIS

We report a 52 year old man who presented with excessive sleepiness and upper airway obstruction due to grossly enlarged tonsils. Following tonsillectomy, the hypersomnolence was completely relieved.

INTRODUCTION

Obstructive sleep apnoea syndrome is characterised by periodic cessation of breathing resulting from cyclical obstruction of the upper airways during sleep. (1) Each episode may last for half a minute or more and is terminated by the patient's arousal. The disturbed sleep results in daytime somnolence which is a frequent presenting complaint. We report here a man with hypersomnolence and upper airway obstruction due to enlarged tonsils.

CASE REPORT

A 52 year old man Chinese man was referred to this hospital in July 1984 for excessive sleepiness for 8 years. More recently, the symptom had worsened. He fell asleep many times during the day and had even fallen asleep at the wheel while awaiting traffic lights to turn green. He snored heavily at night, and periods when breathing stopped completely had been observed by his relatives. He had gained nearly 13.5 kg over the past 3 years. In 1981, he had a left renal calculus removed, and hypertension was discovered then. He smoked 20 cigarettes a day for many years and consumed alcohol occasionally. On examination, he was obese (80 kg). Blood pressure was 190/110 mmHg and pulse was 100 pm and regular. The heart, lungs and abdomen were normal, and no neurological abnormalities were present. Both tonsils were greatly enlarged leaving a narrow pharyngeal orifice of a cm or two across. In the ward, he was noticed to fall asleep very easily and several times during the day. Even during a daytime nap, periods of apnoea with paradoxical abdominal and rib cage movements were observed. During the night, he snored heavily, and several apnoeic spells lasting 15-20 seconds each were noted.

Lung volumes and expiratory flow rates were normal (see table 1), but a flow-volume loop revealed variable extrathoracic upper airway obstruction (see figure 1). Arterial blood gases are shown in table 1. Haemoglobin was 18 gm%. Other investigations including blood urea, serum electrolyte and serum thyroxine levels, and electrocardiogram, chest radiograph and tracheal tomograms did not reveal any abnormalties.

The hypertension was controlled with propranolol and chlorothiazide. Tonsillectomy was performed on 29.08.84. This was followed by improvement of the flow-volume loop (see figure 1) and dramatic relief of the hypersomnolence. After a year of follow-up he has remained well and the relatives have noted improvement in his mental alertness.

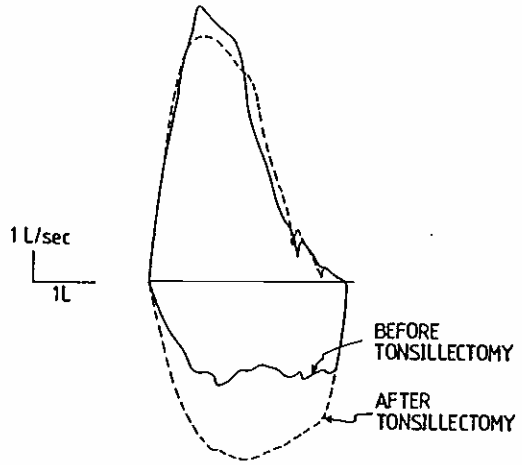

Figure 1. Flow-volume loop showing flattening of the inspiratory loop before tonsillectomy. After tonsillectomy, the inspiratory loop deepened indicating relief of the extrathoracic airway obstruction.

DISCUSSION

The obstructive sleep apnoea syndrome is a sleep breathng disorder characterised by multiple episodes of apnoea during a night's sleep, each episode lasting at least 10 seconds (2). This is accompanied by daytime hypersomnolence, resulting from the interrupted sleep at night. During the spells of apnoea, arterial oxygen saturation may fall to as low as 68% (3), and both pulmonary and systemic arterial pressures are frequently raised, and severe bradycardia has been recorded (4). In later stages, the patient may suffer behavioural changes, sustained pulmonary hypertension, cor pulmonale, and polycythaemia. The disease may terminate as cardiac failure or sudden

TABLE 1 LUNG FUNCTION AND ARTERIAL BLOOD GAS DATA

Forced expired volume in first second	2.74L	(2.78)*
Forced vital capacity	3.05L	(3.32)
Ratio of forced expired volume in first second to forced vital capacity	90%	(70)
Functional residual capacity	2.14L	(2.12)
Residual volume	1.59L	(1.43)
Total lung capacity	5.03L	(5.11)
Maximal midexpiratory flow rate	6.08L/sec	(3.27)
Diffusing capacity for carbon monoxide (single breath method, mmol/kpa/min)	10.4	(7.7)
Arterial pH	7.34	
Arterial oxygen tension	82 mmHg	
Arterial CO2 tension	41 mmHg	
Arterial oxygen saturation	94%	
Standard bicarbonate level	21.7 mmol/L	

*figures in ( ) = predicted normal values.
Predicted normal values for volumes and flow rates are taken from Poh SC and Chia M. Respiratory function in normal adult Chinese in Singapore. Singapore Med J 1969; 10: 265-71; and predicted normal for diffusing capacity from Bates DV, Macklem PT, Christie RV. Respiratory function in disease. WB Saunders. 1971: 93.

nocturnal death (1, 5). Typically, the patient (usually a male) snores in his sleep. The snoring gets louder and louder, and is then abruptly silenced by complete upper airway occlusion. During the apnoea (and occlusion), the patient is seen to be struggling to breathe until arousal occurs when an explosive inspiratory snort marks the end of the apnoeic spell. This is followed by quieter breathing until the next cycle ensues. The explanation for such a cycle follows.

It is often not appreciated that the upper airway muscles (eg genioglossus) are as much respiratory muscles as are the diaphragm and intercostals (6). Electromyographic (EMG) studies of the diaphragm have shown phasic activity coincident with inspiration. With CO2 stimulated breathing diaphragm EMG activity increases as the CO2 level increases, reflecting an increasing ventilatory drive resulting from the chemical (CO2) stimulus (7). This same phasic EMG activity, and EMG response of CO2 stimulated breathing is present in the genioglossus, indicating that it is a respiratory muscle (8). The purpose of this phasic upper airway muscle activity is to hold the upper airway open during inspiration, when negative pressures tend to collapse the extrathoracic airways. During rapid eye movement sleep, all skeletal muscles, except the diaphragm, "sleep" and become hypotonic. With loss of upper airway tone, the splinting effect during inspiration is diminished, so that with each inspiration, the upper airways are sucked inward. In normal people, this may manifest as snoring. When the upper airways are already anatomically narrowed, eg by enlarged tonsils, further dynamic narrowing may lead to complete occlusion, and thus an apnoeic episode. The progressively narrowing lumen with each inspiration explains the loud snoring prior to the apnoeic silence which marks the occlusive period. During this period, there is an increasing ventilatory drive, and inspiratory efforts, as measured by pleural pressure swings, are greatly increased in an attempt to overcome the occlusion. The patient arouses and regains control of the upper airway which then opens (9). This opening, together with the increased inspiratory efforts, typically results in an explosive first breath after the apnoeic spell. The patient then drifts into deeper sleep again and re-enters the apnoea cycle. In one night's sleep several hundred such episodes may occur. During each episode, the arterial oxygen saturation falls. The occult sleep deprivation and hypoxaemia may explain the daytime somnolence, polycythaemia and pulmonary hypertension.

Although not documented by a formal sleep study, we believe that our patient had the obstructive sleep apnoea syndrome. He was severely hypersomnolent, obese, and had functional evidence of upper airway obstruction. He had hypertension which is frequently found in such patients (10). From visual observation, he was seen to have repeated episodes of apnoea during which he was seen to be struggling to breathe. Tosillectomy relieved the upper airway obstruction and completely abolished the hypersomnolence. Adenotonsillectomy has been reported to relieve sleep apnoea in four children who had adenotonsillar obstruction (11).

The treatment of this disorder includs avoidance of sedatives and alcohol, weight loss in the obese (1), nocturnal oxygen in selected patients (12), and of course removal of any lesions that may compromise the airway. Tracheostomy successively treats this condition but is disfiguring. Uvulopalatopharyngoplasty, which tightens up lax upper airways, has been advocated by some (13). Protryptiline, which abolishes rapid eye movement sleep, has been helpful in some cases (14). An interesting mode of treatment is the turnal use of nasal positive airway pressure which may work by preventing inspiratory collapse of the airways (15).

Significantly, the patient himself rarely complains of disturbed sleep. It is often the spouse who is unable to sleep because of the loud snoring, snorting and restlessness of the sleeping patient and it is the spouse who brings attention to the problem.

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