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Prevalence of sleep apnoea in patients over 40 years of age with spinal cord lesions

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Abstract

Twenty two patients over the age of 40 with stable spinal cord damage underwent overnight sleep studies to investigate the prevalence of sleep apnoea. Ten patients had some evidence of obstructive sleep apnoea (OSA). Hypoxic events were scored as number of dips of SaO_2 more than 4% below the preceding 10 minute average (> 4% SaO_2 dip rate). All the patients had more than five such dips per hour and six had clearly abnormal dip rates of more than 15 per hour. Two other patients had dip rates above 10 per hour without apnoeas but periods of central hypoventilation mainly during rapid eye movement (REM) sleep. OSA appears to be more common in older patients with spinal cord injury than in the general population. Possible relevant factors include patient selection, reduced ventilatory function secondary to spinal cord damage, sleep posture and medication.

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Sleep apnoea is conventionally defined as a cessation of airflow at the nose and mouth for more than 10 s during sleep.[1 2] It results from either no ventilatory effort or from obstruction to flow (obstructive sleep apnoea, OSA), which is more common.[3] Occlusion occurs in the upper airway at the level of the oropharynx.

Prime symptoms of OSA are snoring and daytime somnolence.[3] Other features[1 3] include intellectual deterioration, memory loss, poor judgement, pulmonary hypertension with right heart failure,[4] cardiac arrhythmias and, possibly, systemic hypertension.[5] OSA causes impaired psychological functioning, clinical morbidity and increased mortality, so it is important to identify because successful treatment is possible.

In the general population OSA is more common in males and the incidence increases with age, obesity (particularly neck obesity, measured as neck circumference), alcohol intake and age.[6] Sleeping position is an important factor and the supine posture is the most provocative.[7 8] A history of nasal stuffiness, cigarette smoking and snoring are also associated.[6] Underlying lung disease, greater obesity and impairment of ventilatory function all increase the extent and duration of hypoxia[1 3] and the development of cor pulmonale.[9]

In many patients with spinal cord injuries central control of some respiratory muscles is lost. In low cervical cord lesions, the commonest level damaged, ventilatory function depends largely on the inspiratory capacity of the diaphragm. The intercostal and abdominal muscle contribution is lost and changes in tone or spasms in these muscles may have a detrimental effect.[10] Measured lung function remains permanently well below predicted and would render such patients more susceptible to the effects of hypoventilation and apnoea. Apart from reduced ventilatory capacity, several other factors may be relevant to sleep induced alterations in breathing in those with spinal cord injuries. Patients with high spinal cord lesions are often unable to turn themselves without assistance, spending several hours in one position overnight. Nasal stuffiness, owing to interruption of the thoracic sympathetic outflow, is a problem in the acute phase, and to some degree may persist in the long term. Many of the drugs administered for control of spasticity and pain in these patients commonly cause sedation. These considerations led us to investigate the breathing disorders of sleep in older SCI patients.

Methods

Patient selection

Twenty four consecutive inpatients satisfying the basic criteria of age over 40 years and with lesions above T10 were studied. Neurological lesions were Frankel grade A, B or C[11] (that is, no functional motor activity below the level of the cord lesion) and had occurred more than three months earlier. Patients with other medical conditions likely to affect breathing, or suffering from known respiratory problems were excluded. Verbal consent was obtained after a full explanation of the study procedure. In one patient the study was not completed because of nursing problems and patient intolerance. Another study was technically unsatisfactory because of oximeter malfunction and thermistor displacement. The remaining 22 patients were studied adequately.

Community survey data, examining the prevalence of obstructive sleep apnoea in 1001 middle-aged men,[6] were used for comparison.

Techniques

Sleep analysis Sleep was staged conventionally[12] with standard placements of EEG, EMG and EOG electrodes. The raw signals were recorded onto an Oxford Medical, MPA I, multichannel tape recorder for subsequent analysis. Sleep analysis was first performed

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automatically on the Oxford Medical 9000 replay unit and important areas checked and reclassified manually. Derived from this were total sleep time, sleep efficiency, per cent slow wave sleep, per cent REM sleep and minutes of wake after sleep onset.

Respiratory analysis Respiration was measured using ribcage and abdominal strain gauges (mercury in silicone), as well as combined thermistors registering airflow from both nostrils and mouth. Oxygenation was measured continuously using either a Biox II ear oximeter or an Ohmeda 4700 finger oximeter. The use of these oximeters to detect hypoxaemic events and to track them sufficiently accurately for clinical purposes has been published elsewhere.[13 14 15] All these signals were also recorded on the MPA I tape recorder for subsequent computer analysis.

Characterisation of the respiratory events was centred around the oximeter results. Dips in arterial oxygen saturation (SaO_2) of 4% or more from a preceding 10 minute average (weighted to the higher values) were counted for the whole sleep period automatically by the Oxford Medical 9000 system. No account was taken of the length of the dips, most being less than one minute, as is characteristic of OSA. The causes of these hypoxic dips were assessed from the respiratory tracings. Absence or diminution of oronasal airflow with continuing ribcage or abdominal effort, or both, was taken to indicate hypoxic dips due to obstructive apnoea. A reduction in, or absence of, all three signals indicated hypoxic dips due to probable central events.

The hypoxic dips were expressed per hour of sleep. A dip rate of five per hour was used as an arbitrary dividing line[2] and rates in excess of 15 per hour were taken as indicative of clearly abnormal respiration during sleep.[16] In addition, cessation of oronasal airflow in excess of 10 s was documented and expressed per hour of sleep as the apnoea index.

Protocol

Patients were examined to confirm the neurological level and completeness of their spinal lesions and to exclude clinical respiratory complications. Note was made of age, cause and duration of paralysis, relevant past medical history, smoking habits, alcohol consumption and current medication. Neck circumference was measured with a standard tape measure at the level of the cricothyroid membrane. Full lung function tests or flow/volume loop measurements were performed in the sitting position.

The first two patients were studied in an individual side ward. Subsequent studies were performed on the ward without moving the patient. Monitoring was set up between 10 and 11 pm and patients went through their usual regime of overnight turns. Recording was discontinued by the nursing staff between 5 and 7 am. Nursing staff were asked to check and change oximeter placement as appropriate during the night.

Statistical analysis

The relationship between the degree of hypoxaemic dipping and other variables was assessed by Pearson's correlation coefficient, except for the discontinuous variables when analysis of variance was used. Differences in proportions were tested by χ^2 analysis with computed confidence intervals[17] or Fisher's exact test (for small numbers).

Results

There were 20 men and two women with spinal lesions from C_5–T_9 (15 cervical, 7 thoracic) and with a mean duration of paralysis of 12 years (range 0·3–46) (table 1). The sex distribution reflects the pattern of incidence of spinal cord injuries and hospitalisation in this age group. The women were 41 and 49 years of age, and were premenopausal and perimenopausal respectively. All except three cases were traumatic in origin. The other causes were epidural abcess, cervical discectomy for cervical spondylosis and repair of thoracic aortic aneurysm.

The resting arterial oxygen saturation during wakefulness averaged 96·9% (range 94%–98%) indicating that none had significant daytime hypoxaemia. Three patients complained specifically of some sleep disturbance caused by the discomfort of the electrodes, but all completed a virtually full night's sleep study. Nursing staff encountered little difficulty handling patients overnight apart from occasional problems with the ear oximeter falling off. Most patients were turned at least once during the night so that no patient spent the whole of the night in a supine position. Commonly a semi-lateral decubitus position was employed.

Sleep architecture

As a group, sleep quality was poor, (table 2). Not only was there an increased amount of wake after sleep onset but also a marked diminution of slow wave sleep. Much of this sleep disruption did not appear to be related to respiratory disturbances.

Table 1 Age, obesity measures and lung function (of 22 patients studied)

	Median	Range
Age (years)	58·5	40–77
Height (m)	1·78	1·64–1.88
Weight (kg)	79	47–102
Body mass index (kg/m^2)	25·3	14·8–33·3
Neck circumference (cm)	40·3	37–50
Neck circumference (% predicted)	98.3	90·1–125
FEV_1 (litres)	1·99	0·9–3·3
FVC (litres)	2·47	1·11–3·84

Table 2 Sleep architecture (of 22 patients studied)

	Median	Range
Length of sleep study (hours)	7·2	5·5–9·5
Sleep time (hours)	6·4	2·6–8·7
Wake after sleep onset (minutes)	36	9–192
Sleep efficiency (%)	91	43–96
Slow wave sleep (%)	3·5	0–40
REM sleep %	21	0–38
>4% SaO_2 dip rate (per hour)	5·9	0·4–48·1
Apnoea index (per hour)	3·2	0·0–37·7

Table 3 Amount of hypoxaemia during sleep (expressed as the > 4% SaO_2 dip rate per hour)

> 4% SaO_2 dip rate per hour	Number of patients
0–5	10
> 5–10	2
> 10–15	2*
> 15–20	3
> 20	5*

*Two patients had hypoxaemia recorded during sleep without obstructive apnoeas.

Obstructive sleep apnoea and hypoxia

The amount of hypoxaemia during sleep, expressed as the number if dips of SaO_2 more than 4% below the preceding 10 minute average (> 4% SaO_2 dip rate) per hour is shown in table 3. All but two of the subjects with dip rates in excess of five per hour had mainly obstructive apnoeas as the cause. These two patients had hypoxaemia during sleep without apnoeas but periods of central hypoventilation, mainly during REM sleep. One subject subsequently developed clinical left ventricular failure. The other was noted to be sweaty overnight–an indication of autonomic dysreflexia, a condition that causes peripheral vasoconstriction.[18] This possibly affected the finger oximeter used for recording in his case.

Of those with obstructive apnoeas, six patients (out of 22, 27%) had clearly abnormal rates (above 15 per hour). Gross obstructive sleep apnoea, where sleep and breathing cannot occur together, was present in three patients with dip rates of 30, 31 and 48 per hour respectively. The worst was a 42 year old male patient. Periods of central apnoea did occur, but always in association with obstructive sleep apnoea, as a consequence of the hyperventilation overshoot following obstruction.

There was no significant correlation between the degree of hypoxic dipping and age, level of spinal cord lesion, duration of paralysis, obesity index, neck circumference or lung function.

Table 4 Comparison of patients with hypoxaemia and obstructive sleep apnoea (dip rate more than five/hr) and those with essentially normal sleep (dip rate less than five/hr)

	> 4% SaO_2 dip rate > 5/hour	> 4% SaO_2 dip rate < 5/hour)
Numbers	10 men*	8 men, 2 women
Cord lesion	C5–T9	C5–T8
	6 cervical	8 cervical
	4 thoracic	2 thoracic
	Median (range)	Median (range)
Age (years)	64 (42–77)	52·5 (40–67)
Duration of paralysis (years)	2 (0·3–46)	8 (0·3–24)
Obesity index (kg/m^2)	25·4 (14·8–29·6)	23·7 (18·7–29·4)
Neck circumference (cm)	41 (38–46)	40 (37–50)
Vital capacity (litres)	2·21 (1·40–3·84)	2·70 (1·1–3·38)
Clinical features	number (mean dose)	number (mean dose)
Smoking	2	1
Alcohol	1	1
Medication		
Dantrolene	4 (187·5 mg)	2 (150 mg)
Baclofen	6 (35 mg)	1 (40 mg)
Diazepam	2 (4·5 mg)	3 (8·3 mg)
Lorazepam	1 (2 mg)	1 (2 mg)
Amitriptyline	1 (50 mg)	1 (100 mg)
Carbamazepine	1 (600 mg)	2 (500 mg)

*Two patients without obstructive apnoea excluded–see text.
Number = number of patients in each group.
(Mean dose) = mean daily dose of medication.

The only assessment of symptoms was an enquiry about daytime sleepiness. None complained specifically of this, although feeling fatigued is common, particularly among patients with higher spinal cord lesions. Four patients in the obstructive sleep apnoea group had been noted by nursing staff to have levels of snoring that disturbed the ward.

The clinical features of those with > 4% SaO_2 dip rates of less than five (essentially normal sleep breathing pattern) and those with more than five per hour dip rate associated mainly with obstructive apnoeas are shown in table 4. Baclofen was used by six patients in the group with dip rates of more than five per hour compared with one patient in the group with rates of less than five per hour ($p = 0{\cdot}06$, Fisher's exact test).

Two patients in the OSA group were taking antihypertensive medication. Another, with the worst OSA, was on phenobarbitone and phenytoin for epilepsy only. Interestingly one patient in the normal group with a > 4% SaO_2 dip rate of zero was on high dose clomipramine following treatment of severe depression.

Discussion

This study demonstrated obstructive sleep apnoea resulting in > 4% SaO_2 dip rate of more than five per hour in 10 out of 22 unselected spinal cord inpatients over the age of 40 years. This is far more common than in the normal population as studied in a large general practice based survey of 1001 men aged 35–65 years.[6] Overnight oximetry performed in 893 of these men demonstrated > 4% SaO_2 dip rates of more than five per hour in 45 subjects. Of these, 31 participated in a full sleep study, performed in hospital, which showed OSA only when supine in 18 and clinically obvious severe sleep apnoea in three. This gives a prevalence of occult sleep apnoea of between 2% and 4%, with clinical sleep apnoea occurring in 0·3%. Our present study recorded OSA to some degree in 45% of 22 inpatients.

The factors determining inpatient status may have had an influence on this assessment of prevalence. Approximately half of the patients were examined during their first admission. Two thirds of the remainder were elective urological cases and most of the remainder had pressure sore problems. The effect of a strange night-time environment is likely to be small as they had either been in this hospital for at least two months or were previously familiar with ward procedures. Most studies were carried out in the patient's usual bed on the ward. Only one sleep study was carried out on each patient. While there may be night-to-night variation in amount of OSA, the degree of severity of the syndrome in most of the patients was far too much to be caused by a particularly bad night in a normal person. These considerations are unlikely to be sufficient to detract from the definite increase in incidence over the normal population.

Review of published reports

There are few reports on sleep apnoea in

patients with spinal cord damage although their reduced respiratory function is well documented and allusion made to its significance during sleep.[19] Recently Bonekat *et al*[20] described four patients with spinal cord injuries presenting with sleep complaints who underwent nocturnal polysomnography. Disordered breathing was diagnosed in all, three of whom were cervical patients over 45 years of age. All were taking medication with potential sedative effects (two were taking baclofen). Braun *et al*[21] monitored oxygen saturation, using ear oximetry, in 11 patients with spinal cord injuries, four of whom were more than 40 years old. Sleep was recorded by an observing technician. The maximal drop in oxygen saturation recorded correlated significantly with age but not with level of spinal cord lesion or per cent predicted vital capacity. In two patients (a 42 year old man with a C3/4 lesion and unilateral diaphragm dysfunction and a 57 year old man with a T3/T4 lesion) oxygen saturation fell below 90%. A case report[22] of a patient with a long-standing cervical cord lesion who had a respiratory arrest following elective surgery highlighted the existence of unrecognised sleep apnoea syndrome.

Effect of spinal cord injury

Several factors may be relevant to this observed prevalence of OSA in older patients with spinal cord injuries. Impaired ventilatory muscle function obviously produces reduced respiratory reserve, increasing the hypoxic consequence of a period of apnoea, but other factors must operate which affect the aetiology of that airflow obstruction. Patency of the oropharyngeal airway depends on the tone in upper airway muscles and coordination of that activity with respiratory muscle action. Reduction in muscle tone can compromise that patency but the mechanism of occlusion is also related to gravity.[3 7] This is reflected in the importance of sleep posture in the general population; many have OSA only in the supine position. Many spinal patients require assistance to turn and are unable to make the usual postural changes during sleep. Although a completely supine position is infrequently employed, being in one position for several hours may still be relevant to the disturbances of breathing during sleep.

The tone and strength of all muscles is affected by antispasm medication administered orally, such as diazepam, baclofen and dantrolene. This could be directly relevant to the mechanism of oropharyngeal airway occlusion although such drugs also have central effects. Baclofen, in particular, acts as a γ-aminobutyric acid agonist, that is, an inhibitory neurotransmittor, possibly relevant to sleep induced changes, for example, depression of monosynaptic and polysynaptic reflexes in the tonic phase of REM sleep.[23] Use of antispasm medication, particularly baclofen, was greater in the group of patients with sleep apnoea, although the difference in baclofen usage did not reach statistical significance.

There is some suggestion that the "sensory deprivation" produced by a spinal cord lesion itself affects the pattern of sleep in these patients with less time being spent in the deeper stages of sleep.[24] The amount of daytime motor activity is considered to influence sleep make-up as well.[24] Alterations in the ascending activity from the spinal cord could also have an effect on ascending reticular activity and central autonomic functioning so as to affect the control of respiration.[23]

The reduced ventilatory function due to muscle involvement below the level of the spinal lesion may be further reduced by sleep related changes in respiratory muscle activity. In non-REM sleep there is a reduced drive to the diaphragm of the order of 1–2 litres per minute; in REM sleep intercostal and accessory muscle activity is reduced.[3] Effective use of accessory muscles is also dependent on posture (less when supine) and mainly voluntary as far as is known. This is relevant with high cervical cord lesions; the accessory muscle contribution is probably reduced during sleep, exposing the diaphragm to fatigue effects.[19]

The present study has found obstructive sleep apnoea to be a common problem in older spinal cord patients. This may well affect their ability to participate effectively in rehabilitation, let alone find employment and cope with the demands of a disabled lifestyle. The degree of OSA suggests a need for further studies to evaluate its effect on psychological function and to assess methods of treatment.

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