Case Report

Two case study reports of sleep apnoea in patients with paraplegia

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Study design: Case studies of sleep apnoea occurring in two patients with paraplegia. **Objective:** To raise awareness of sleep apnoea in paraplegia.

Setting: Belfast, Northern Ireland.

Case report: We report two patients with paraplegia, one who was having apparent episodes of loss of consciousness and the other daytime somnolence, who were found to have sleep apnoea. The first patient had been medically investigated extensively and a diagnosis of epilepsy was being considered. A joint consultation with the respective partners in each case revealed periods of night-time apnoea and led to sleep study investigations.

Conclusion: Sleep apnoea is a treatable condition that can occur in patients with paraplegia who are not necessarily obese. Once diagnosed, resolution of symptoms can be rapid and can result in improved quality of life for patients.

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Introduction

A few studies have been written that describe the occurrence of sleep apnoea in persons with paraplegia. ^{1,2} These investigators report that it may be more common in these patients than in the general population. The association of tetraplegia with sleep apnoea, however, is well recognized.³

Sleep apnoea can lead to significant morbidity – daytime sleepiness, nocturnal sweating, enuresis and increased cardiovascular risk.⁴ While obesity is frequently found in patients with sleep apnoea and can prompt the doctor to consider sleep studies, it may not always be present. We present two patients with paraplegia, neither of whom was obese, who were showing signs and symptoms of sleep apnoea. The presence of the partner during the consultation in each case was crucial to the diagnosis being considered.

Case report 1

A 54-year-old male who had sustained a T 7 level incomplete paraplegia 37 years earlier secondary to a spinal osteoblastoma attended the Spinal Injury Review Clinic.

He had suffered a blackout 3 months earlier while driving his car and a further episode when he almost blacked out. He was admitted to a district general hospital for further investigations including Computerized Tomography of the brain and 24-h ambulatory taped electrocardiography, all of which were normal.

His wife, who had accompanied him to the clinic, reported that he snored extremely loudly and had episodes when he stopped breathing at night and during the day he sometimes fell asleep inappropriately. The patient then confirmed that he slept poorly and was usually tired when he woke in the mornings.

He was a nonsmoker, and took occasional alcohol. Medication: Tolterodine 2mg b.d. Body mass index (BMI) 27 kg/m^2 , spirometry revealed FEV₁ 81%, FVC 79%. He was referred for investigations for sleep apnoea. Overnight oximetry showed a typical saw tooth pattern of desaturations and 25 dips/h in SAO₂ in keeping with a diagnosis of sleep apnoea (Figure 1). Continuous positive airways pressure (CPAP) was started at home, and at review the patient reported that he had not slept as well in years and he has had no further episodes of loss of consciousness, which were most likely sudden episodes of sleep.

Case report 2

A 55-year-old male who had sustained a T 4 level complete paraplegia 25 years back secondary to a road traffic accident was reviewed at the Spinal Injury Clinic. He complained of daytime drowsiness, especially during the previous 6 months. His wife who accompanied him

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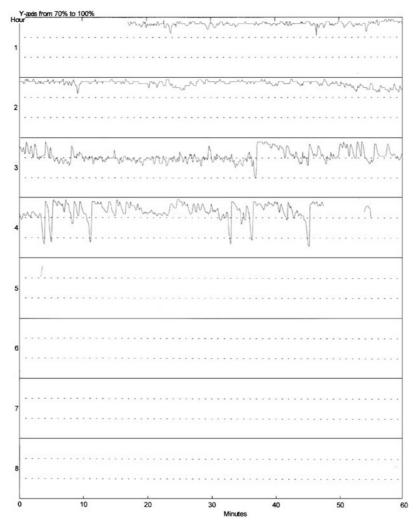


Figure 1 A four hour nocturnal (while sleeping) oxygen saturation (pulse oximetry) tracing for patient 1. The tracing starts top left and finishes bottom right. The scale for the Y-axis is 70-100% SaO₂. The typical saw-tooth pattern of SaO₂ desaturations is shown indicating sleep apnoea – average 25 dips per hour. Normal sleep would show a straight line

to the clinic mentioned that he appeared to stop breathing for periods during the night and would awake abruptly. He was not known to snore.

He was a nonsmoker, and took occasional alcohol. Medication: Oxybutynin 5 mg t.i.d. (trial of stopping this medication provided no relief of symptoms).

BMI 26 kg/m^2 , FEV₁ 93%, FVC 98%. He was referred for investigations for sleep apnoea. Overnight oximetry revealed a typical saw tooth pattern and 15.6 dips/h in keeping with a diagnosis of sleep apnoea.

CPAP was tried at home; however, he did not persist with this treatment as he found it too uncomfortable to wear.

Discussion

Sleep apnoea can be either obstructive or central and is arbitrarily defined as cessation of breathing for longer than $10 \,\mathrm{s}$ with > 35 episodes at night. A less restricting definition such as 'sleep disruption syndrome sufficient to cause daytime symptoms' is now often preferred.³

Hypoxia and hypercapnia usually accompany the periods of obstructed breathing and evidence suggests that the main stimulus to arousal from sleep is the actual inspiratory effort made in response to asphyxia rather than the asphyxia $per se.^{6}$ Obstructive sleep apnoea (OSA) is more common than central and affects at least 1% of the general population especially men aged >40years. It is quite possible that our two patients represent members of that cohort of people who are more likely to have OSA and that the paraplegia is an incidental, noncontributory finding. It can be diagnosed by sleep studies measuring oxygen saturation, arterial BP and airflow limitation. Most patients with this have central obesity as the main cause and have an average BMT $> 30 \text{ kg/m}^2$, although neck circumference is a better predictor of OSA severity.

Alcohol and sedatives can aggravate sleep apnoea.

Stopping sedatives and weight reduction can be effective treatments for mild symptoms; however, nasal CPAP is the most rapid and effective treatment.⁸

Sleep apnoea in tetraplegic patients is well documented and, although it is usually obstructive in type, it can also be mixed.³ The prevalence of sleep-disordered breathing in patients with tetraplegia is estimated as twice that of the normal population.⁹

Sleep apnoea in paraplegic patients is poorly documented. One retrospective population-based study of 584 patients with spinal cord injury revealed that 3.7% of paraplegic patients had sleep apnoea and they were not able to specify if the apnoea was obstructive or central. The true prevalence is likely to be higher as this was a retrospective study and considered only previously diagnosed cases.¹⁰ Both of our patients had BMI $<30 \text{ kg/m}^2$. Overnight oximetry only was used to diagnose sleep apnoea in our patients and therefore we are unable to specify if the apnoea was obstructive, central or mixed in type. We consider, however, that the cause of sleep apnoea in patient 1 was most likely to be obstructive as he was a snorer and this could have been confounded by the intercostal muscle weakness and hypoventilation resulting from the spinal injury. Other possible mechanisms whereby paraplegia might lead to obstructive sleep apnoea include neck muscle hypertrophy as a compensatory response to intercostal muscle weakness and a supine sleeping position.^{11,12}

Patient 2, who was not known to snore, may have had central sleep apnoea secondary to weakness of the respiratory muscles. However, repeat sleep lab testing perhaps with a different, more comfortable mask would be essential to rule out that CPAP was not helpful and thus obstructive type apnoea is unlikely.

Conclusion

This case report highlights three important points.

Sleep apnoea can occur in patients with paraplegia.

Spouses or partners of patients with paraplegia presenting with episodes of loss of consciousness or daytime somnolence should be carefully questioned regarding any problems that the patient might have with snoring or episodes of cessation of breathing. Driving for patients with paraplegia is an important aspect of mobility, independence and quality of life. Episodes of loss of consciousness and daytime somnolence threaten this independence. The diagnosis and treatment of sleep apnoea in patients with paraplegia could significantly improve their quality of life.

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