



## Treatment of sleep apnoea in spinal cord injured patients

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Little is known about sleep disorders in spinal cord injured (SCI) patients. Three SCI patients who reported severe daytime sleepiness and sleep complaints were evaluated with nocturnal polysomnography and oxygen saturation with pulsoximeter on several occasions at home. In addition respiratory registration was performed during overnight stay in the hospital. Two patients who had sleep apnoea episodes with reduced oxygen saturation during sleep were treated with continuous positive airway pressure with good results on sleep architecture, oxygen saturation, and daytime sleepiness. One obese patient was advised to lose weight and to reduce smoking and alcohol consumption, and following this advice his sleep related problems were reduced with no further treatment necessary. It is suggested that SCI patients who complain about sleep related disorders should be appropriately screened, with inclusion of nocturnal polysomnography, oxygen saturation, and respiratory registration and, depending on the screening results, appropriate advice/treatment applied.

**Keywords:** spinal cord injury; tetraplegia; sleep; sleep apnoea; nocturnal sleep registration; oxygen saturation

### Introduction

In recent years there has been growing interest in the respiratory events occurring during sleep. These include the diagnosis of sleep apnoea<sup>1</sup> which is associated with symptoms of irregular snoring, daytime sleepiness, lack of concentration, personality changes, and other symptoms.<sup>2</sup> Spinal cord injured (SCI) patients commonly complain about difficulty in sleeping.<sup>3</sup> They have restless sleep, complain of spasms, difficulty in initiating and maintaining sleep, some snore, often awaking in the early hours of the morning and are unable to fall asleep again, and during daytime they are tired and sleepy. SCI patients often sleep in the supine position, they have a tendency to obesity, are commonly using sedative medication, and all these factors may aggravate the quality of sleep. Many SCI patients use a wheelchair and their daytime activities start late in the morning.<sup>3</sup> In spite of these risk factors in the SCI patient population there is at present no evidence that SCI individuals are more prone to have sleep apnoea beyond that anticipated from their age and body mass.

There is a general agreement that sleep apnoeic events increase with age and are more common in men than in women.<sup>4-7</sup> Further there is an association with lifestyle factors such as obesity, and tobacco and alcohol consumption.<sup>8,9</sup> Obstructive sleep apnoea may require treatment with continuous positive airway

pressure (CPAP) via a nasal mask. It relieves the obstruction and gives an undisturbed sleep. Daytime sleepiness disappears after a few nights of treatment.<sup>10-12</sup>

### Methods

Three patients with SCI who reported sleep disturbances and daytime tiredness were studied.

The level of the spinal cord lesion was determined as the most caudally normally functioning spinal cord segment at injury and the functional level, the Frankel class<sup>13</sup> at the latest follow-up.

Nocturnal recordings were performed at least three times in each patient: first in the patient's home (untreated), secondly in the hospital (untreated) and finally at control registration after advice or during treatment with CPAP (in home/hospital). The home registrations were polysomnographies (PSG) with simultaneous registration of: electroencephalogram (C3/4-A1/2), surface chin electromyogram, eye movements (actigraph), nasal flow (thermistor), electrocardiogram and sound (microphone). The oxygen saturation (SaO<sub>2</sub>) was measured with finger pulse oximetry.

The in-hospital registration was an overnight respiratory recording of oxygen saturation, transcutaneous oxygen and dioxide tensions, nasal/oral ventilatory flow, chest wall and abdominal respiratory movements.

Obstructive apnoea is defined as more than 10 s cessation of nasal/oral flow with continued activity of

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the respiratory muscles. Flow cessation is due to total pharynx obstruction. Every apnoea is terminated by an arousal during which the patient respire and snores violently. After a few inspirations a new apnoea starts and the pattern is repeated. The typical patient with severe sleep apnoea has 400–500 apnoeas with arousals per night.<sup>4,14</sup>

## Case reports

### Patient 1

A 47-year-old man, 19 years after a diving accident, with a neurological level of C6 incomplete, T2 complete, and a functional level corresponding to Frankel class D, complained of severe daytime sleepiness, even in the middle of a conversation. He slept in the supine position, and had the impression of waking up about once every hour because of bad dreams, respiratory problems, bladder emptying feeling, etc. He took medication for spasticity (baclofen 25 mg  $\times$  3, diazepam 5 mg  $\times$  3). He reported snoring for 20–30 years, and smoked 20 or more cigarettes daily. At the time of the initial PSG his weight was approximately 110 kg.

The initial PSG at home showed a marked respiratory disturbance with a mean of 73 apnoeas/hypopnoeas per hour. The SaO<sub>2</sub> dropped to 78% (mean) during every apnoea. The sleep pattern was totally disturbed and there was no deep sleep (slow wave sleep). Because of obesity he was primarily advised to lose weight, and no further treatment was found to be indicated.

Six months after the initial PSG he was admitted for one night in hospital (respiratory) registration. The SaO<sub>2</sub> was 80–90%, and there were 16 apnoeas/hypopnoeas per hour of 10–60 s duration. The respiration was paradoxical all through the night. The registration showed mixed central-obstructive apnoeas.

Meanwhile, the patient lost 33 kg in weight and reduced his intake of alcohol (which was previously 10–15 beers/day) and his smoking. Afterwards he reported that he was no longer tired during the day. He could concentrate and only seldom had bad dreams. The control PSG at home (2 years after the initial PSG) showed normal sleep architecture with normal SaO<sub>2</sub> 94–97%.

### Patient 2

This patient was 54 years old, 6 years after a traffic accident, and with a neurological level C6 incomplete, and Frankel class D. In later years he had experienced increasing problems with tiredness during the day; he began to take a nap during the day, started snoring, had sleep difficulties, and headache. He was smoking 10–15 cigarettes per day, but seldom took alcohol. He had a nasal septum operation 6 months prior to the initial PSG, because of breathing difficulties. His medications included baclofen, and frusemide with potassium.

The first PSG showed severe sleep apnoea with a mean of 83 apnoeas per hour during each of which the SaO<sub>2</sub> decreased to 83%. Every apnoea disturbed sleep and precluded deep sleep.

Five months later in-hospital respiratory registration was performed showing typical obstructive sleep apnoea with SaO<sub>2</sub> down to 70%. There were 164 apnoeic episodes longer than 10 s. Treatment with nasal CPAP during the night was started, and his daytime tiredness and sleeping difficulties disappeared.

Control PSG (with CPAP equipment), 18 months after the initial measurement, showed normal sleep architecture with undisturbed normal sleep during the whole night. Regular snoring was observed in shorter periods of the night. The SaO<sub>2</sub> was normal 97–98% during the whole night.

Twenty three months after the initial registration new respiratory registration (with CPAP equipment) in hospital was performed, showing no signs of apnoea or oxygen desaturation.

### Patient 3

This patient was a 56-year-old man 37 years after a traffic accident, with a T2 complete spinal cord lesion, Frankel class A, and also avulsion of the right brachial plexus. In later years he had increasing problems with concentration, and reported air-hunger during night-time. He had been snoring for about 20 years, and for about 5 years he had apnoeas with increasing tiredness and decreasing work ability during daytime. He had no lung disease, but took methadone for pain affecting his right arm. He smoked 0–3 cheroots daily, weighed about 90 kg, and was 186 cm tall.

The first PSG showed an apnoea pattern during 61% of the night. For the remainder of the night he had regular snoring. In periods with regular snoring sleep was undisturbed. In periods with apnoeas sleep was totally disturbed with 64 apnoeas per hour. For the whole night there was a mean of 39 apnoeas per hour. The SaO<sub>2</sub> dropped to 80% during every apnoea.

Three months later the in-hospital registration showed obstructive apnoeas and mixed central-obstructive apnoeas. The patient started CPAP treatment and his tiredness during the daytime disappeared. In addition he lost 10 kg in weight.

Because of arm pain a control PSG (with CPAP equipment), 13 months after initial PSG, showed only half a night's sleep. The sleep was undisturbed without snoring. For some unknown reason rapid eye movement (REM) sleep was not seen. The oxygen saturation was normal 96–98%.

## Discussion

Paradoxical respiration was secondary to cervical SCI. All three subjects slept in a supine position and used a wheelchair in their daily life. Patient 1 with severe overweight had improvement in his symptoms as he lost weight, which is in accordance with previous experience.<sup>15</sup> After treatment with CPAP the two other patients gained a normal sleep pattern, normal oxygen saturation, were no longer tired during the day, felt they slept well and had no more problems in concentrating. Two of the patients used antispasm medication (baclofen/diazepam) which may affect the tone and strength of all muscles, and contribute to oropharyngeal airway occlusion, which may prolong apnoea.<sup>16</sup> Cahan and co-authors<sup>17</sup> have found that six out of 16 stable male tetraplegic patients (C4–T5) had SaO<sub>2</sub> profiles outside of normal range compared with a control group of 12 age-matched healthy male subjects. They used 24-h pulse oximetry to evaluate the SaO<sub>2</sub> profiles. They concluded that tetraplegic patients who are hypoxic during sleep are better identified by monitoring SaO<sub>2</sub> or sleep studies, rather than by their



clinical history. Our experience likewise indicates that after taking a clinical history, a nocturnal sleep registration, including measurement of oxygen saturation, should be performed to permit prescription of optimal treatment. In another study Braun *et al*<sup>18</sup> screened 11 stable patients with cervical and thoracic spinal cord lesions with nocturnal oximetry for an average of 2.6 h. The stage of sleep was not determined. They concluded that young tetraplegic patients do not have severe oxygen desaturation during sleep, but older patients and patients with some diaphragmatic dysfunction, should be screened for sleep desaturation.

From Bonekat *et al*<sup>3</sup> it appears that patients who have a high cervical transection have a substantial increase in the amount of light sleep, and there is an absolute and relative reduction in the amount of deep and rapid eye movement sleep; we also found this in our study.

Although our case reports include three SCI patients only, we find the results of the advice/treatment very encouraging. Therefore we suggest that SCI patients who complain about sleep related disorders should be appropriately screened. Such a screening should include nocturnal polysomnography, with measurement of oxygen saturation, and respiratory registration. Depending on the screening results, appropriate advice/treatment should be applied, as for instance nasal CPAP during sleep, with considerable improvement in the patients' quality of life.

## References

- 1 Guilleminaut C, Tilkian A, Dement WC. The sleep apnea syndromes. *Annual Rev Med* 1976; **27**: 465–484.
- 2 Guilleminaut C. Clinical features and evaluation of obstructive sleep apnea. In: Kryger MH, Roth T, Dement WC (eds). *Principles and Practice of Sleep Medicine*. WB Saunders Company: Philadelphia, 1989, pp 552–558.
- 3 Bonekat HW, Andersen G, Squires J. Obstructive disordered breathing during sleep in patients with spinal cord injury. *Paraplegia* 1990; **28**: 392–398.
- 4 Douglas NJ, Polo O. Pathogenesis of obstructive sleep apnoea/hypopnoea syndrome. *Lancet* 1994; **344**: 653–655.
- 5 Bixler EO *et al*. Sleep apneic activity in a normal population. *Res Commun Chem Pathol Pharmacol* 1982; **36**: 1441–152.
- 6 Block AJ, Boysen PG, Wynne JW, Hunt LA. Sleep apnea, hypopnea, and oxygen desaturation in normal subjects. *N Engl J Med* 1979; **300**: 513–517.
- 7 Young T *et al*. The occurrence of sleep-disordered breathing among middle-aged adults. *N Engl J Med* 1993; **328**: 1230–1235.
- 8 Jennum P, Sjøel A. Epidemiology of snoring and obstructive sleep apnoea in a Danish population, age 30–60. *J Sleep Res* 1992; **1**: 240–244.
- 9 Kauffman F *et al*. The relation between snoring and smoking, body mass index, age, alcohol consumption and respiratory symptoms. *Eur Resp J* 1989; **2**: 599–603.
- 10 Hoffstein V, Viner S, Mateika S, Conway J. Treatment of obstructive sleep apnoea with nasal continuous positive airway pressure. *Am Rev Respir Dis* 1992; **145**: 841–845.
- 11 Polo O, Berthon-Jones M, Douglas NJ, Sulivan CE. Management of obstructive sleep apnoea/hypopnoea syndrome. *Lancet* 1994; **344**: 656–660.
- 12 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**: 862–865.
- 13 Frankel HL *et al*. The value of postural reduction in the initial management of closed injuries in the spine with paraplegia and tetraplegia. *Paraplegia* 1969; **7**: 179–192.
- 14 American Sleep Disorders Association. Obstructive sleep apnea syndrome. In: *The International Classification of Sleep Disorders*, 1992, pp 52–58.
- 15 Nahmias J, Kirschner M, Karetzky MS. Weight loss and OSA and pulmonary function in obesity. *New Jersey Med* 1993; **90**: 48–53.
- 16 Short DJ, Stradling JR, Williams SJ. Prevalence of sleep apnoea in patients over 40 years of age with spinal cord lesions. *J Neurol Neurosurg Psychiatry* 1992; **55**: 1032–1036.
- 17 Cahan C *et al*. Arterial oxygen saturation over time and sleep studies in quadriplegic patients. *Paraplegia* 1993; **31**: 172–179.
- 18 Braun SR, Giovannoni R, Levin AB, Harvey RF. Oxygen saturation during sleep in patients with spinal cord injury. *Am J Phys Med* 1982; **61**: 302–308.