Spinal Cord (1998) 36, 621–628

http://www.stockton-press.co.uk/sc

Obstructive sleep apneas in relation to severity of cervical spinal cord injury

B Klefbeck[1], M Sternhag[2], J Weinberg[3], R Levi[4], C Hultling[4] and J Borg[2]

Departments of [1]Physical Therapy, Karolinska Institute, Stockholm, [2]Clinical Neuroscience, Karolinska Hospital, Stockholm, [3]Neurology, Huddinge Hospital, Huddinge, [4]Spinalis SCI Research Unit, Karolinska Institute, Stockholm, Sweden

Thirty-three subjects (28 men, five women) with complete or incomplete cervical cord injury representing a wide range of neurological impairment were investigated with regard to the prevalence of Obstructive Sleep Apnea (OSA). The relation between OSA and neurological function, respiratory capacity, body mass index and symptoms associated with OSA were studied. Overnight sleep recordings employed combined oximetry and respiratory movement monitoring. Pulmonary function tests included static and dynamic spirometry, maximal static inspiratory and expiratory pressures at the mouth. The subjects answered a questionnaire concerning sleep quality and tiredness. The prevalence of OSA was 15% (5/33) in this non-obese cervical cord injury study population. Nine percent of the subjects (3/33) fulfilled the criteria for obstructive sleep apnea syndrome, but daytime sleepiness or fatigue were also common in subjects without OSA. There was an inverse correlation between oxygen desaturation index and American Spinal Injury Association (ASIA) motor score in the subjects with complete injury, while there was no such correlation in the whole study group. There were significant correlations between maximal inspiratory and expiratory pressures and vital capacity and between ASIA motor score and vital capacity.

Keywords: ASIA motor score; oxygen desaturation; sleep apnea syndrome; sleep; tetraplegia

Introduction

Cervical spinal Cord Injury (CCI) might cause respiratory insufficiency due to respiratory muscle paresis. Lesions above the C5 level are most severe due to paresis of the diaphragm and might cause a reversible or persistent need for mechanical ventilation.[1,2] In CCI patients with respiratory muscle paresis, but without need for assisted ventilation, an increased incidence of Obstructive Sleep Apneas (OSA), when compared to a non-injured population, has been reported.[3–6]

OSA is characterised by sleep-related intermittent upper airway obstruction which may be associated with episodes of oxygen desaturations and sleep fragmentation.[7] In obstructive sleep apnea syndrome this is combined with symptoms such as snoring, excessive daytime sleepiness and cardiovascular sequelae.[8] The definition of respiratory events differs slightly in different studies.[9–12] Young *et al*[10] and Gislason *et al*[11] define the apnea-hypopnea score as either a complete cessation of airflow lasting 10 s or more (apnea) or a reduction in respiratory airflow accompanied by a decrease of 4% or more in oxygen saturation (hypopnea). The apnea-hypopnea score is the average of apneas and hypopneas per hour of sleep. Using this score, Young *et al* found that 9% of women and 24% of men had an apnea-hypopnea score of $\geqslant 5$. When they combined this with daytime hypersomnolence, they estimated the prevalence of obstructive sleep apnea syndrome to 2% among women and 4% among men. In a community-based study by Gislason *et al*[11] the prevalence of obstructive sleep apnea syndrome was estimated at 1.3% among Swedish men.

Patients with cervical cord injuries often complain of sleeping problems and daytime sleepiness.[13] McEvoy *et al*[3] reported that nine of 40 patients with spinal cord injuries at C8 level and above had obstructive apneas and six of the 40 patients complained of daytime sleepiness which was directly related to the frequency of sleep arousals. Cahan *et al*[5] noted that five of six quadriplegic patients with nighttime hypoxia had an increased daytime sleepiness compared to six of ten quadriplegic patients with nonhypoxia.

The pathophysiology of OSA is not fully understood.[7] Obesity, ethanol, male gender, age and conditions that narrow the upper airway are considered strong risk factors in the non-injured population.[14,15] In the CCI population, data concerning relations between sleep-disordered breathing and characteristics of the CCI is limited. An association between time spent below 90% SaO_2 and body mass

Correspondence: B Klefbeck, Department of Physical Therapy, Karolinska Institute, Novum, S-141 57 Huddinge, Sweden

index, level of injury and maximum expiratory pressure[6] as well as between sleep-disordered breathing and neck circumference and supine sleep posture[3] have been reported.

The aims of this study were to further elucidate the prevalence of OSA and obstructive sleep apnea syndrome in subjects with CCI by use of a screening method for OSA and to study the relation to neurological function, respiratory capacity, body mass index and sleeping position.

Subjects

The criteria for participation were traumatic tetraplegia, one year or more post injury, motor injury level C4-T1 with complete or incomplete tetraplegia (A-D according to the American Spinal Injury Association (ASIA) impairment scale)[16,17] 20–60 years of age, no pulmonary or cardiovascular disorders. Subjects with ventilatory treatment due to hypoventilation were excluded.

The subjects were invited to participate in the study as a complement to an annual routine follow up (May 1995 to November 1995) offered to all spinal cord injured subjects living in the Stockholm region.[13] Of thirty-seven consecutive subjects, 33 subjects gave informed consent. There were 28 men and five women. Mean age was 37.9 (range 22–59) years and mean duration of injury was 13.9 (range 1–33) years. Individual data in groups of complete and incomplete injury are given in Table 1.

Methods

Neurological examination for determination of completeness according to the ASIA impairment, sensory and motor scoring scales[16,17] was performed.

Overnight sleep recording was performed in a single room in hospital. The sleeping position was repeatedly observed. However, the majority of the subjects slept all night in the same position because they were unable to turn. A simplified sleep apnea investigation consisting of combined ear oximetry (Ohmeda Biox 3740, Health Care, Louisville, USA) and respiratory and body movement monitoring (Static Charge Sensitive Bed) as described by Svanborg *et al*[8] was performed. The body movement monitoring and oximetry data were collected by use of a pen-recorder (Graftec Thermal Array-corder WR 770, Graphitic Corporation, Yokohama, Japan) using slow paper speed (10 mm/min) to clearly demonstrate changes in oxygen saturation (SaO_2) and respiration movement patterns. Oxygen Desaturation Index (ODI) was defined as the average number of oxygen desaturations of 4% or more per sleeping hour.[8] Periods of obstructive apneas form a typical diamond-shaped pattern in the body movement monitoring (periodic respiration pattern), usually accompanied by repetitive oxygen desaturations.[8] The recordings were analyzed manually. The number of significant desaturations and total time with periodic respiration in minutes were calculated. The nadir SaO_2 value (the lowest value during the night) was also noted. Sleeping time was esimated from the patterns in the movement recording. Total sleeping time had to be 4 h or more, otherwise the examination was repeated. Criterion for pathologic recording, ie indicating OSA, was the combination of ODI $\geqslant 6$ and $>45\%$ periodic respiration time out of the total estimated sleeping time.[8] If the subjects were unable to sleep in hospital, they were offered to sleep at home with the ApnoLog system (C-A Tegner Inc, Stockholm, Sweden) including pulse oximetry (Biox 3760), respiration and body movements (PVDF

Table 1 Characteristics of the subjects, nos 1–17 complete injuries, nos 18–33 incomplete injuries

	Subject	*Gender*	*Age (years)*	*Time (years)*	*Level of injury (motor)*	*(sens)*
	1	Male	26	1	C4	C3
	2	Male	46	28	C7	C7
	3	Male	59	30	C5/C6	C3
	4	Female	25	9	C5	C4
	5	Male	36	20	C5	C6
	6	Female	26	4	C6	C6
	7	Male	25	6	C6	C5
	8	Male	29	1	C6/C5	C5
	9	Male	46	32	C7	C6
	10	Male	28	7	C6	C5
	11	Male	30	27	Th1	C7
	12	Male	32	15	Th1	C7
	13	Male	24	3	C6	C5
	14	Male	23	4	C6	C4
	15	Male	22	2	C5	C4
	16	Female	51	18	C5	C3
	17	Male	28	8	C4	C4
	Mean		*32.7*	*12.6*		
	SD		*11.0*	*11.0*		
	18	Male	30	11	C7	C7
	19	Male	24	2	C6	C6
	20	Male	43	25	C5	C4
	21	Female	39	4	C5	C4
	22	Male	34	15	C6	C4
	23	Male	45	3	C8	C7
	24	Male	54	33	*	C3
	25	Male	53	4	C5	C3
	26	Male	39	16	C6	C7
	27	Male	43	24	C5	C5
	28	Male	53	10	C7	C7
	29	Male	50	33	C7	C7
	30	Male	47	2	C6	C4
	31	Male	58	33	C4	C4
	32	Female	30	2	C5	Th5
	33	Male	53	27	C5	Th7
	Mean		*43.4*	*15.3*		
	SD		*10.1*	*12.2*		
Total	*Mean*		*37.9*	*13.9*		
	SD		*11.8*	*11.5*		

Time (years) = time elapsed since injury, motor = motor level of injury, sens = sensory level of injury, * = not possible to assess with ASIA, SD = standard deviation

pressure sensor mattress).[18] To make certain both methods were correlated, four subjects performed recordings both at home and in hospital. The results of the two methods were in good accordance even in estimating sleeping time.

A pulmonary function test with static and dynamic spirometry was performed and Maximal Inspiratory Pressure (MIP) and Maximal Expiratory Pressure (MEP) at the mouth (Precision Medical Ltd, UK)[19–21] were measured. Body mass index (kg/m^2) was calculated from the subject's weight in kg and length in meters.[22] For the Swedish population, body mass index between 20.1 and 25.0 in men and 18.7 and 23.8 in women is considered normal.[22]

A detailed drug report including present use of antispastic and sedative drugs was obtained.

At the time of the sleep recordings, a questionnaire concerning sleep and tiredness during the last three months was distributed. The Basic Nordic Sleep Questionnaire[23,24] was used together with three questions from a Sleep and Health Questionnaire developed by Kump *et al* (Likert scale 1–6).[25] In Kump's Questionnaire, the subject was asked to quantify the extent of her/his sleepiness, energy level, or performance impairment due to sleepiness.

Regression analyses were used to describe the relation between ODI, periodic respiration, ASIA motor score and pulmonary function data. Pearson's or Spearman's correlation coefficient was used to identify the relationship between variables. To analyse the answers from the questionnaire, they were subjectively divided into two groups (normal and pathologic) and a non-parametric test, Mann–Whitney, was used. $P<0.05$ was considered significant.

Results

Sleep respiratory recordings

Thirty-one of the subjects performed the sleep recordings during one night. One patient had to do the sleep recording an extra night due to too short sleeping time during the first night. One further subject had sleeping difficulties in hospital and performed a sleep recording at home.

Mean sleeping time was 6.4 (SD 1.0) h and ranged 4.0–8.2 h. Further individual sleep recording data are presented in Table 2. Mean awake SaO_2 was 96.2% (SD 1.2) and ranged 94–98. Mean nadir SaO_2 was 88.4% (SD 6.4) and ranged 69–95.

Median ODI was 1 and ODI ranged 0–29. Median periodic respiration was 4% and ranged 0–89%. When only subjects with ODI > 0 ($n=17$) were considered, there was a statistically significant correlation between ODI and nadir SaO_2 ($r_s=0.82$, $P<0.05$). When all subjects with ODI > 0 and periodic respiration > 45% were considered there were statistically significant correlations between ODI and periodic respiration ($r_s=0.50$, $P<0.05$), as well as in the two subgroups with complete and incomplete injuries ($r_s=0.67$, $P<0.05$ and $r_s=0.68$, $P<0.05$). Five of the 33 subjects (15%), all men, had ODI > 6 and periodic breathing > 45% (values thus considered as pathological).[8] Thus five of the 28 men (18%) fulfilled the diagnostic criteria for OSA. Five additional subjects had pathological amounts of periodic breathing of clearly obstructive type (21–71%). A sixth subject had ODI = 5 and periodic respiration of 19%. These six subjects are regarded as borderline cases.[8]

ASIA motor score and sensory score and ODI/sleep respiratory recordings

Mean ASIA motor score was 36.6 (SD 26.9) and ranged 0–90. Mean ASIA sensory score was for light touch 33.7 (SD 26.7) and ranged 8–110 and for pin prick 40.0 (SD 29.3) and ranged 8–110. Individual data are given in Table 2. There was no significant correlation between sleep respiratory pathology and motor or sensory scores. Of the five subjects with OSA, two had ASIA motor scores of 0 and 9, while the other three subjects had ASIA motor scores of 43, 68 and 72. Two had complete injury and three had incomplete injury.

In the 17 subjects with complete injury (cf. Table 1) there was a significant ($r=0.48$, $P<0.05$) correlation between ODI and ASIA motor score.

Vital capacity, MIP, MEP and ODI/sleep respiratory recordings

Spirometric pulmonary function tests were performed in 27 patients, and MIP and MEP were performed in 30 patients.

Mean Vital Capacity (VC) was 3.39 (SD 1.0) and ranged 1.21–4.85 litre. Mean forced expiratory volume during 1 s was 2.95 (SD 0.8) and ranged 1.15–3.90 litre. Mean MIP was 76.7 (SD 28.2) and ranged 17–131 cm H_2O. Mean MEP was 70.4 (SD 31.7) and ranged 19–149 cm H_2O. Individual data are given in Table 3. There were no significant correlations between ODI or periodic respiration and any of the lung function tests. There were significant correlations between MIP and VC ($r=0.59$, $P<0.01$), MEP and VC ($r=0.66$, $P<0.001$) and between ASIA motor score and VC ($r=0.60$, $P<0.01$).

Body mass index, age and ODI/sleep respiratory recordings

Mean Body Mass Index (BMI) was 21.2 (SD 3.8) and ranged 13.6–30.0 kg/m^2. It should be noted that none of the subjects was morbidly obese (BMI > 30). Individual data are given in Table 4. Three men had BMI > 25 and one female had BMI = 30.0, none of these had ODI > 6. The age of the subjects ranged from 22–59 years. There were no significant correlations between ODI or periodic respiration and BMI, age or time elapsed since injury.

Sleeping position and ODI/sleep respiratory recordings
Twenty-seven of 33 subjects slept in the supine position and only five of them alternated between the supine and lateral positions or between the prone and supine positions. Six subjects spent all night in the lateral position. Individual data are given in Table 4. Of five subjects with OSA, four spent all night in the supine position and the fifth subject spent part of the night in the supine position. Of the six subjects with borderline results, four slept all night in the supine position, one altered between the lateral and supine position and the sixth subject slept in the lateral position. None of the 22 subjects slept in the elevated position.

Table 2 Individual sleep breathing data and motor and sensory scores, subjects nos 1–17 complete injuries, subject nos 18–33 incomplete injures

	Subject	*Awake SaO_2 (%)*	*Nadir SaO_2 (%)*	*ODI*	*Periodic respiration (%)*	*Motor score*	*Sensory score light t*	*pin p*
	1	95	77	29	89	0	12	12
	2	97	88	2	0	38	30	30
	3	97	81	11	77	9	18	18
	4	98	92	0	4	8	14	15
	5	96	86	1	4	19	20	22
	6	95	92	0	4	18	18	12
	7	97	88	1	30	16	14	14
	8	97	95	0	3	14	16	16
	9	96	93	0	0	40	29	29
	10	97	91	1	11	19	22	22
	11	95	93	0	0	46	26	25
	12	96	92	0	0	48	33	33
	13	95	90	0	3	23	17	20
	14	95	93	0	0	34	22	22
	15	94	87	1	20	15	17	21
	16	98	95	0	0	11	8	8
	17	94	75	5	19	4	10	10
	Mean	*96*	*88.7*			*21.3*	*19.2*	*19.4*
	Median			*0*	*4*			
	SD	*1.3*	*6.0*			*14.7*	*7.1*	*7.2*
	18	96	92	0	0	25	28	28
	19	96	92	1	4	44	25	59
	20	96	90	0	16	12	36	52
	21	98	92	0	30	24	16	62
	22	97	91	1	9	17	47	62
	23	94	80	23	74	43	83	82
	24	98	79	7	48	72	12	–
	25	96	69	22	79	68	60	60
	26	96	80	5	71	68	74	81
	27*	96	93	0	0	76	110	110
	28	95	93	0	0	90	71	108
	29	98	89	2	21	30	34	35
	30	97	90	1	24	87	86	74
	31	95	91	0	5	–	13	43
	32	97	94	0	0	88	76	80
	33	98	93	1	4	65	14	14
	Mean	*96.4*	*88*			*53.9*	*49.1*	*63.3*
	Median			*1*	*12.5*			
	SD	*0.2*	*7.1*			*27.4*	*31.2*	*27.1*
Total	*Mean*	*96.2*	*88.4*			*36.6*	*33.7*	*40.0*
	Median			*1*	*4*			
	SD	*1.2*	*6.4*			*26.9*	*26.7*	*29.3*

Awake SaO_2 = oxygen saturation at start of the registration, Nadir SaO_2 = lowest registered saturation, ODI = total number of desaturations divided by the sleeping time in hours, light t = light touch, pin p = pin prick, * = home sleep recording, – = missing value, SD = standard deviation

Drugs and ODI/sleep respiratory recordings
Eleven subjects used baclofen (70–125 mg) and diazepam (1.25–5 mg) for reducing spacticity. One subject with OSA and three borderline cases used baclofen. A routine clinical evaluation revealed no history of alcohol or psychotropic drug abuse. Individual data are presented in Table 4.

Questionnaire, snoring and ODI/sleep respiratory recordings
Three of the five subjects with ODI > 6 and seven of the 28 subjects with ODI < 6 reported excessive tiredness and sleep disturbances. Three of 33 (9%) subjects therefore fulfilled the criteria for obstructive sleep apnea syndrome. Three subjects with ODI > 6

Table 3 ODI and data from pulmonary function tests, subjects nos 1–17 complete injuries, subjects nos 18–33 incomplete injuries

	Subject	*ODI*	*VC (litre)*	*VC pred*	*$FEV_{1,0}$ (litre/s)*	*$FIV_{1,0}$ (litre/s)*	*FRC (litre)*	*MIP (cm H_20)*	*MEP (cm H_20)*
	1	29	1.21	20	1.20	–	3.96	48	19
	2	2	3.78	75	3.75	3.52	5.24	68	63
	3	11	–		–	–	–	70	39
	4	0	1.22	29	1.15	1.02	1.86	42	25
	5	1	3.00	53	2.46	2.62	3.10	51	38
	6	0	2.92	74	2.67	2.68	2.70	80	51
	7	1	3.93	64	3.64	3.31	4.06	58	32
	8	0	2.92	51	2.70	2.42	–	74	63
	9	0	–		–	–	–	–	–
	10	1	3.81	66	3.23	3.20	4.18	71	71
	11	0	–		–	–	–	131	109
	12	0	4.21	75	–	–	2.52	129	103
	13	0	4.42	72	3.75	–	3.88	107	105
	14	0	–		–	–	–	56	58
	15	1	2.66	47	2.43	2.42	3.58	62	32
	16	0	1.28	36	1.19	–	–	–	–
	17	5	2.20	37	–	–	–	41	48
	Mean		*2.89*	*53.8*	*2.56*	*2.64*	*3.5*	*72.5*	*57.1*
	Median	*0*							
	SD		*1.1*	*18.9*	*1.0*	*0.8*	*1.0*	*28.6*	*29.2*
	18	0	–		–	–	–	66	67
	19	1	4.13	67	3.68	3.91	3.85	123	97
	20	0	3.74	68	3.23	3.01	3.48	114	74
	21	0	3.11	92	2.86	2.86	3.29	47	44
	22	1	3.31	66	3.10	2.99	3.94	88	65
	23	23	4.85	96	3.80	4.47	3.94	85	80
	24	7	3.96	81	3.25	3.66	3.41	–	–
	25	22	4.57	93	3.90	3.83	4.07	101	104
	26	4	4.06	84	3.32	3.69	2.52	100	108
	27	0	3.83	72	3.40	3.18	3.76	55	78
	28	0	3.34	74	2.69	2.94	2.66	73	149
	29	2	3.32	62	3.11	2.97	4.29	92	75
	30	1	–		–	–	–	109	118
	31	0	3.05	79	2.04	1.54	2.87	17	30
	32	0	3.95	92	3.32	3.75	2.36	81	78
	33	1	4.69	107	3.85	3.65	4.31	63	88
	Mean		*3.85*	*80.9*	*3.3*	*3.32*	*3.48*	*80.9*	*83.7*
	Median	*1*							
	SD		*0.6*	*13.5*	*0.5*	*0.7*	*0.7*	*28.2*	*29.2*
Total	*Mean*		3.39	67.9	2.95	3.07	3.49	76.7	70.4
	Median	*1*							
	SD		*1.0*	*21.2*	*0.8*	*0.8*	*0.8*	*28.2*	*31.7*

ODI = total number of desaturations divided by the sleeping time in hours, VC = Vital capacity, pred = percent of predicted normal, $FEV_{1,0}$ = Forced expiratory volume during 1 s, $FIV_{1,0}$ = Forced inspiratory volume during 1 s, FRC = Functional residual capacity, MIP = Maximal inspiratory pressure, MEP = Maximal expiratory pressure, – = missing values, SD = standard deviation

and four subjects with ODI < 6 reported of snoring. Individual data of symptoms and snoring are presented in Table 4. There were no statistically significant correlations between ODI and data from the questionnaire.

Discussion

The study group can be considered as unselected and representative for the cervical cord injured subjects in the Stockholm area as the subjects were recruited

Table 4 ODI in relation to BMI, sleeping position, symptoms, snoring and used muscle relaxant, subjects nos 1–17 complete injuries, subjects nos 18–33 incomplete injuries

	Subject	*ODI*	*BMI* (kg/m^2)	*Sleeping position*	*Symptom*	*Snoring*	*Muscle relaxant*	*Dosage*
	1	29	14.9	sup	+	+		
	2	2	20.1	prone/sup				
	3	11	22.5	sup	+	+		
	4	0	15.5	lat				
	5	1	23.4	sup			bac	125 mg
	6	0	23.7	lat				
	7	1	16.2	sup			bac	75 mg
	8	0	19.1	sup			bac	30 mg
	9	0	22.0	lat	+	+	bac	75 mg
	10	1	14.8	sup				
	11	0	21.6	lat	+			
	12	0	17.6	lat				
	13	0	19.9	sup				
	14	0	18.6	sup			bac	75 mg
	15	1	20.1	sup	+			
	16	0	16.9	sup				
	17	5	25.2	lat/sup			bac	100 mg
	Mean		*19.5*					
	Median	*0*						
	SD		*3.2*					
	18	0	23.0	sup				
	19	1	20.5	sup				
	20	0	24.6	sup			dia	5 mg
	21	0	19.6	sup				
	22	1	21.6	sup	+		bac + dia	70 + 1.25 mg
	23	23	22.5	sup	+	+		
	24	7	23.1	lat/sup				
	25	22	21.1	sup			bac	75 mg
	26	4	23.4	sup		+		
	27	0	23.5	sup				
	28	0	27.7	lat/sup				
	29	2	13.6	lat		+		
	30	1	25.6	sup	+		bac	125 mg
	31	0	23.0	lat/sup	+	+	dia	5 mg
	32	0	30.0	sup	+			
	33	1	24.9	sup				
	Mean		*23.0*					
	Median	*1*						
	SD		*3.6*					
Total	*Mean*		*21.2*					
	Median	*1*						
	SD		*3.8*					

ODI = total number of desaturations divided by the sleeping time in hours, BMI = Body Mass Index, sup = supine, lat = lateral, + = symptom present as excessive tiredness and sleep disturbances or snoring, bac = baclofen, dia = diazepam, Dosage = dosage of muscle relaxant, SD = standard deviation

consecutively from the yearly routine follow-up. There were 28 men (85%) in this study, as compared to 81% men in an inventory of the spinal cord injured population in the same region in Sweden.[13] Subjects included had complete or incomplete injuries at different cervical levels representing a wide spectrum of neurological and respiratory dysfunction.

In the present study the prevalence of cases who fulfilled the laboratory criteria for OSA was 15% (5/33), that is within the same order as in a community based study by Young *et al*[10] of non-injured subjects where the prevalence of OSA was 9% in women and 24% in men. However, when comparing this data two points must be considered. First, Young's as well as other studies of non injured populations include obese subjects known to have an increased risk for OSA.[12,14,26,27] In our study there were no morbidly obese subjects. Four subjects had BMI above normal range, corresponding to overweight for three men and obesity for one woman,[22] and those four all had normal nocturnal respiration. In a population-based survey of non-injured subjects by Kripke *et al*[27] the prevalence of ODI $\geqslant$20 in subjects with BMI $<$29 was 3%, while the corresponding prevalence in our study was 9%. Thus, the prevalence of OSA seems to be higher in non-obese CCI-subjects than in non-obese, non-injured subjects. Further, in our study there were six additional border-line cases with high amounts of obstructive breathing but virtually normal oximetry. Some of these might have more than 10 s cessation of airflow and thus fulfil the criteria for OSA according to Young *et al*.[10]

There was a significant negative correlation between the degree of obstructive respiration and ASIA motor score in the subpopulation of CCI subjects with more severe injuries. Previous studies also indicate that the prevalence of OSA increases with more severe injuries (cf. further discussion below).

There was no correlation between ODI and data from the questionnaire. Ten subjects reported symptoms in accordance with obstructive sleep apnea syndrome. Three of them had OSA and reported sleep disturbances and excessive daytime tiredness/sleepiness and snoring and thus fulfilled the criteria for obstructive sleep apnea syndrome, thus the estimated prevalence of obstructive sleep apnea syndrome in the study population was 9% (3/33), that is higher than the estimated 1.3% in a Swedish prevalence study[11] of non injured subjects. Seven subjects reported sleep disturbances and excessive tiredness, but had normal sleep recordings, indicating that other causes of disturbed sleep are common in this group.

The observed prevalence of OSA in the present study was lower than reported in previous studies of CCI subjects.[3–6] This might be due to different methods and patient selection. The method used for screening of OSA and the criteria used for classification of OSA in the present study are well established and only slightly different from those in some of the previous studies[3–5] and should have only marginal, if any, significance for the differing results. However, previous studies have included subjects with a higher mean age and/or more severe injury. The observed prevalence of OSA was 22% in a study by McEvoy *et al*[3] in CCI-subjects with Frankel grades A–C and a mean vital capacity of 54.3% of predicted normal, 45% in a study by Short *et al*[4] in CCI-subjects who had a mean age of 58.5 years, a median vital capacity of 2.47 litres and Frankel grades A–C and 38% in a study by Cahan *et al*[5] in subjects with a mean age of 48 years. Flavell[6] reported severe nocturnal desaturations in 30% in subjects with injury at C4–C6 level with Frankel grades A–B and a mean vital capacity of 45.7% of predicted normal. Thus, we suggest that higher age and more severe injury might explain the higher prevalence of OSA observed in these previous studies. This is also in agreement with our observation of an inverse correlation between ODI and ASIA motor score in the subjects with complete injury in the present study and thus in accordance with previous findings where the level of injury or vital capacity was correlated to OSA.[5,6]

It has been shown that normal subjects may exhibit OSA after topical oropharyngeal anesthesia[28] and that the temperature sensitivity is selectively impaired in the oropharynx of patients with obstructive sleep apnea syndrome.[29] The CCI subjects had no sensory involvement of the oropharynx and there was no significant correlation between ODI or periodic breathing and degree of sensory loss, as defined in the ASIA sensory score. This indicates that the sensory disturbance due to spinal cord injury has no significance for OSA.

There was a significant correlation between ASIA motor score and VC, as expected, as well as between VC and MEP and between VC and MIP in the present study. The significant correlation seen between VC and MIP is in contrast to the study of Roth *et al*,[30] who did not find any correlation between these parameters in 52 patients with complete C4-Th6 spinal cord injury. The discrepancy might be explained by a greater range of motor functions in the present study, which included subjects with both complete and incomplete injuries.

Of the subjects with OSA four of five spent all night supine, one subject was supine part of the night, and had ODI = 7. However, the majority[22,33] of the tetraplegic subjects in the present study had difficulties in changing sleeping position during the night and thus spent all night in the supine position. Previous studies of non-injured subjects indicate that the supine position increases the risk for OSA[31] while the lateral position reduces the risk.[32] Even if our data does not permit any conclusion, it is in accordance with these findings.

Eleven of 33 subjects in the present study were using muscle relaxants but only one of them was classified as OSA and three as borderline cases. In the study of Short *et al*[4] six of 22 patients with more than five desaturations per hour were using baclofen. It has

been shown that a single dose of 25 mg baclofen alters the sleep architecture and produces a small reduction in mean sleep oxygen saturation although it does not significantly increase sleep-disordered breathing.[33] Thus, baclofen might be a risk factor for OSA but our data indicates that moderate doses are not a major risk factor.

Conclusion

The prevalence of OSA in CCI subjects with a wide range of motor loss (ASIA motor score 0–90) and respiratory impairment (VC 1.21–4.85 litre) was 15% and higher than in a corresponding, non-obese, non-injured population. There was an inverse relation between ODI and ASIA motor score in the subgroup with complete injury. The prevalence of obstructive sleep apnea syndrome was 9%, which is higher than in the estimated prevalence in Swedish non-injured subjects. Symptoms associated with OSA were not related to the results of the respiratory recordings and are thus not appropriate for predicting OSA in this patient group.

Acknowledgements

This study was supported by grants from The Spinalis Foundation, Karolinska Institute and the Swedish Association for Traffic and Polio Disabled. Eva Svanborg MD, PhD is acknowledged for her valuable comments.

References

1 Carter RE. Respiratory aspects of spinal cord injury management. *Paraplegia* 1987; **25:** 262–266.
2 Fuhrer MJ *et al*. Postdischarge outcomes for ventilator-dependent quadriplegics. *Arch Phys Med Rehabil* 1987; **68:** 353–356.
3 McEvoy DR *et al*. Sleep apnoea in patients with quadriplegia. *Thorax* 1995; **50:** 613–619.
4 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.
5 Cahan C *et al*. Arterial oxygen saturation over time and sleep studies in quadriplegic patients. *Paraplegia* 1993; **31:** 172–179.
6 Flavell H *et al*. Hypoxia episodes during sleep in high tetraplegia. *Arch Phys Med Rehabil* 1992; **73:** 623–627.
7 Strohl KP, Redline S. Recognition of obstructive sleep apnea. *Am J Respir Crit Care Med* 1996; **154:** 279–289.
8 Svanborg E, Larsson H, Carlsson-Nordlander B, Pirskanen R. A limited diagnostic investigation for obstructive sleep apnea syndrome. Oximetry and static charge sensitive bed. *Chest* 1990; **98:** 1341–1345.
9 Lavie P. Incidence of sleep apnea in a presumably healthy working population: A significant relationship with excessive daytime sleepiness. *Sleep* 1983; **6:** 312–318.
10 Young T *et al*. The occurance of sleep-disordered breathing among middle-aged adults. *N Engl J Med* 1993; **328:** 1230–1235.
11 Gislason T *et al*. Prevalence of sleep apnea syndrome among Swedish men: an epidemiological study. *J Clin Epidemiol* 1998; **41:** 571–576.
12 Strandling JR, Crosby JH. Predictors and prevalence of obstructive sleep apnoea and snoring in 1001 middle aged men. *Thorax* 1991; **46:** 85–90.
13 Levi R, Hultling C, Nash MS, Seiger Å. The Stockholm spinal injury study: 1. Medical problems in regional SCI population. *Paraplegia* 1995; **33:** 308–315.
14 Block A, Boysen PG, Wynne JW, Hunt LA. Sleep apnea, hypopnea and oxygen desaturation in normal subjects. *N Engl J Med* 1979; **300:** 513–517.
15 Deegan PC, McNicholas WT. Pathophysiology of obstructive sleep apnoea. *Eur Respir J* 1995; **8:** 1161–1178.
16 Ditunno JF, Young W, Donovan WH, Creasey G. The international standards booklet for neurological and functional classification of spinal cord injury. *Paraplegia* 1994; **32:** 70–80.
17 El Masry WS *et al*. Validation of the American Spinal Injury Association (ASIA) Motor Score and the National Acute Spinal Cord Injury Study (NASCIS) Motor Score. *Spine* 1996; **21:** 614–619.
18 Svanborg E, Pàal T, Möller J. Apno-Log - a portable monitoring system for home recordings of obstructive sleep apneas. *Sleep Res* 1992; **1:** 225.
19 Black LF, Hyatt RE. Maximal static respiratory pressures in generalized neuromuscular disease. *Am Rev Respir Dis* 1971; **103:** 641–650.
20 Black LF, Hyatt RE. Maximal respiratory pressures: Normal values and relationship to age and sex. *Am Rev Respir Dis* 1969; **99:** 696–702.
21 Hamnegård C-H *et al*. Portable measurement of maximum mouth pressures. *Eur Respir J* 1994; **7:** 398–401.
22 Kuskowska WA, Rössner S. Body mass distribution of a representative adult population in Sweden. *Diab Research Clinical Practice* 1990; **10:** S37–S41.
23 Gisalson T *et al*. Basic Nordic Sleep Questionnaire. Scandinavian Society for Sleep Research. 1988.
24 Biering-Sörensen F, Biering-Sörensen M, Hilden J. Reproducibility of Nordic Sleep Questionnaire in spinal cord injured. *Paraplegia* 1994; **32:** 780–786.
25 Kump K *et al*. Assessment of the validity and utility of a sleep-symptom questionnaire. *Am J Respir Crit Care Med* 1994; **150:** 735–741.
26 Vgontzas AN *et al*. Sleep apnea and sleep disruption in obese patients. *Arch Intern Med* 1994; **154:** 1705–1711.
27 Kripke DF *et al*. Prevalence of sleep-disordered breathing in ages 40–64 years: A population-based survey. *Sleep* 1997; **20:** 65–76.
28 McNicholas W *et al*. Upper airway obstruction during sleep in normal subjects after selective topical oropharyngeal anesthesia. *Am Rev Respir Dis* 1987; **135:** 1316–1319.
29 Larsson H *et al*. Temperature thresholds in the oropharynx of patients with obstructive sleep apnea syndrome. *Am Rev Respir Dis* 1992; **146:** 1246–1249.
30 Roth EJ *et al*. Pulmonary function testing in spinal cord injury: correlation with vital capacity. *Paraplegia* 1995; **33:** 451–457.
31 Svanborg E *et al*. Hur vanlig är positionskänslighet vid obstruktivt sömnapnésyndrom? *Abstract Läkarstämman, Stockholm* 1995.
32 McKenzie Neill A, Angus SM, Sajkov D, McEvoy RD. Effects of sleep posture on upper airway stability in patients with obstructive sleep apnea. *Am J Respir Crit Care Med* 1997; **155:** 199–204.
33 Finnimore AJ, Roebuck M, Sajkov D, McEvoy RD. The effects of the GABA agonist, baclofen, on sleep and breathing. *Eur Respir J* 1995; **8:** 230–234.