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Original article

Subjective sleep quality in cystic fibrosis

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Abstract

Objectives: To evaluate sleep quality in patients with cystic fibrosis (CF).

Methods: The Pittsburgh Sleep Quality Index (PSQI) questionnaire was administered to 37 CF patients with moderate to severe lung disease in a clinically stable state. Sleep studies were performed concurrently. PSQI scores were correlated with results of anthropometric variables, arterial blood gas tensions, lung function variables, and polysomnographic variables. Potential differences in objective measurements between patients with high and low scores on the PSQI were assessed.

Results: Thirty-seven patients with CF were studied, aged 27 ± 8 (mean ± 1 SD) years and forced expiratory volume in 1 s (FEV_1) 36 ± 12% predicted. The mean PSQI was 5.7 ± 4.0. Fourteen of the 37 patients had a high PSQI, i.e. >5. Significant correlations between objective variables and both component scores and total PSQI were as follows: age and 'subjective sleep quality' ($r = 0.4$, $P < 0.05$), age and 'sleep duration' ($r = 0.3$, $P < 0.05$), FEV_1 % predicted and 'subjective sleep quality' ($r = -0.4$, $P < 0.05$), carbon monoxide transferred per litre of lung volume (KCO) % predicted and 'daytime dysfunction' ($r = -0.4$, $P < 0.01$), $PaCO_2$ and 'sleep latency' ($r = 0.4$, $P < 0.01$), arterial carbon dioxide tension ($PaCO_2$) and 'habitual sleep efficiency' ($r = 0.3$, $P < 0.05$), $PaCO_2$ and total PSQI ($r = 0.4$, $P < 0.05$), absolute minimum sleep oxyhemoglobin saturation by pulse oximetry (SpO_2 %) and 'sleep latency' ($r = -0.4$, $P < 0.05$), absolute minimum sleep SpO_2 % and 'sleep duration' ($r = -0.4$, $P < 0.05$), absolute minimum sleep SpO_2 % and total PSQI ($r = -0.4$, $P < 0.05$) and awake transcutaneous CO_2 and 'sleep duration' ($r = 0.45$, $P < 0.05$). Better sleep efficiency ($P < 0.05$) and a greater % of rapid eye movement (REM) sleep ($P < 0.05$) were found in those patients with a PSQI of ≤5.

Conclusions: A number of CF patients reported poor sleep quality. A relationship was shown between subjective sleep quality and physiological variables describing disease severity. Better sleep efficiency and % REM sleep were seen in patients with low PSQI scores. These results suggest a useful role for the PSQI in assessing sleep quality in patients with CF.

Keywords: Subjective sleep quality; Cystic fibrosis; Sleep-disordered breathing

1. Introduction

Cystic fibrosis (CF) is the most common lethal genetic disease in the Caucasian population with one in 20–25 people a carrier of the recessive gene, and approximately one in every 2500 neonates afflicted. The affected gene responsible for CF has been identified as being on the long arm of chromosome 7 [1–3]. The most common cause of both the morbidity and mortality in patients with CF is respiratory disease [4] due to persistent lung infection and inflammation with progression to chronic suppurative lung disease with established bronchiectasis. Malabsorption due to impaired pancreatic glandular function and diabetes are other sequelae commonly associated with CF.

Nocturnal cough, sleep fragmentation and falls in oxyhemoglobin saturation (SpO_2) during sleep have all been reported in patients with CF [5,6]. Spier et al. (1984) reported sleep quality, as measured on polysomnography, to be poor in patients with CF with severe lung disease compared with normal controls [7]. However, there are no data available on subjective sleep quality in these patients and whether abnormalities in sleep quality are specific to those with sleep-disordered breathing. Disturbance of the sleep/wakefulness cycle has been shown to adversely affect daytime function [8,9], lead to reduced immune responses [10–12], and decreased insulin sensitivity [13–15] in normal

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healthy subjects. Therefore, the aim of this study was to evaluate sleep quality in patients with moderate and severe lung disease due to CF.

2. Methods

2.1. General

Adult patients with CF were studied whilst in a stable clinical condition, defined by the criteria of Fuchs et al. (1994) [16]. Any patient with moderate or severe lung disease was invited to participate in a trial involving polysomnography and subjective sleep quality assessment using the Pittsburgh Sleep Quality Index (PSQI). Subjective sleep quality results for patients at a single time point only are reported.

This study was conducted at the Royal Prince Alfred Hospital in Sydney, Australia, and approved by the ethics committee of our institution (protocol number X97-0204). Written informed consent was obtained from all patients.

2.2. Anthropometric and lung function measurements

Anthropometric and lung function data were obtained on the day of the diagnostic sleep study. Measurements of spirometry were performed using a Mass Flow Sensor (Sensormedics Vmax 20 Pulmonary Spirometry instrument, Sensormedics Corporation, Yorba Linda, CA) which was calibrated before each study and compared with normal predicted values of Quanjer et al. (1993) [17]. Lung volumes were determined by body plethysmography (Gould 2800; Gould Electronics, Dayton, OH). Results were compared with normal predicted values of Goldman and Becklake (1959) [18]. Inspiratory muscle pressure at residual volume ($P_I$max) and expiratory muscle pressure at total lung capacity ($P_E$max) were recorded using a hand held pressure gauge and the results were compared with normal predicted values of Wilson et al. (1984) [19]. A gas chromatograph (1085D Series PF/ Dx™ system, Medical Graphics, St. Paul, MN) was used to measure carbon monoxide transferred per litre of lung volume (KCO) and diffusing capacity of carbon monoxide (D_LCO). D_LCO is a measurement of carbon monoxide transfer from inspired gas to pulmonary capillary blood flow. Our laboratory does not use a predicted value for actual D_LCO but uses a normal predicted value for KCO of 5.4 ml CO/mmHg per min per l (BTPS), a value based on mean laboratory values for normal non-smoking healthy adults. Using a single breath technique such as D_LCO in patients with gas trapping carries the potential for underestimating the alveolar volume. This was avoided with the use of predicted values for KCO, which does not rely on the calculation of alveolar volume.

Two patients colonized with *Burkholderia cepacia* were restricted by hospital infection control policy to having spirometry only. They had arterial blood gas (ABG) testing, but no respiratory muscle strength testing, KCO testing or lung volume measurement by plethysmography. One extremely hypoxic patient was unable to undergo KCO testing due to her inability to remain off low flow oxygen (LFO_2) as is required for this testing procedure.

2.3. ABG tensions

Awake ABGs were obtained with the patient seated and breathing room air, usually in the late afternoon prior to the sleep study being performed.

2.4. Questionnaire data

The PSQI was completed on the day of the sleep study. Administration of the questionnaire occurred during a face-to-face interview, with non-directional guidance. The PSQI is a self-rated questionnaire assessing sleep quality and disturbances over a 1 month time interval that has been validated for clinical populations [20].

The PSQI consists of 19 self-rated questions grouped into seven component scores, each weighted equally on a 0–3 scale. The seven components are then summed to yield a global PSQI score, which has a range of 0–21, with higher scores indicating worse sleep quality. The seven components of the PSQI are standardized versions of areas routinely assessed in clinical interviews of patients with sleep/wake complaints including: (1) subjective sleep quality, (2) sleep latency, (3) sleep duration, (4) habitual sleep efficiency, (5) sleep disturbances, (6) use of sleeping medications and (7) daytime dysfunction.

A global PSQI score of >5 is considered to be a sensitive and specific measure of poor sleep quality [20]. A global PSQI score of >5 indicates that a subject is having severe difficulties in at least two areas, or moderate difficulty in more than three areas [20].

2.5. Sleep study recordings

During polysomnography, continuous recordings were made on a computerized system (Sleepwatch™, Compumedics, Melbourne, Australia). Sleep stage was determined from two channels of electroencephalogram (EEG) (C_4/A_1, C_3/A_2), one electromyogram (EMG) channel recording the mentalis/submentalis muscle activity and two ocular channels recording from the left and right outer canthus (LOC/A_2, ROC/A_1). Additionally, a movement sensor was placed on one tibialis anterior to assess for periodic leg movements. Respiratory variables were monitored using abdominal and thoracic impedance bands for chest wall movement and diaphragm EMG electrodes to reflect respiratory effort. Nasal airflow was measured using nasal prongs attached to a pressure transducer (AutoSet™, ResMed Inc., Sydney, Australia). SpO_2 was measured with a finger probe (3700e, Ohmeda, Boulder, CO). The resting awake value of SpO_2 was noted, and SpO_2 recording continued overnight. Transcutaneous carbon dioxide ($TcCO_2$) (TCM3; Radiometer, Copenhagen, Denmark) was also measured continuously overnight. $TcCO_2$ and

SpO_2 were recorded simultaneously on both the Sleepwatch™ system as well as a PC-based data acquisition system (A/D board National instrument AT-MIO-16, sampling at 10 Hz).

Sleep stages were scored in 30 s epochs according to the standard criteria of Rechtschaffen and Kales (1968) [21]. An EEG arousal was defined as an abrupt increase in EEG frequency for ≥3 s that in rapid eye movement (REM) sleep was accompanied by an increase in submental EMG amplitude. Sleep efficiency was defined as the total sleep time (TST) as a percentage of the time available for sleep. Apnea was defined as cessation of airflow for ≥10 s, or a cessation of airflow for <10 s with an oxygen desaturation of ≥3%, or an arousal. Hypopnea was defined as a reduction in amplitude of airflow, or thoracoabdominal wall movement of >50% for ≥10 s, or a reduction in airflow or thoracoabdominal wall movement of >50% for <10 s if it was accompanied by an oxygen desaturation of ≥3%, or an arousal. The number of apneas and hypopneas per hour of non-rapid eye movement (NREM), REM and TST were calculated and reported as the respiratory disturbance index (RDI). Arousal was scored according to the ASDA 3 s definition [22].

The absolute minimum sleep SpO_2 was documented for the entire night but also for REM and NREM sleep. The percentage of TST, REM and NREM sleep time with $SpO_2 \leq 90\%$ was calculated. The minimum average SpO_2 was calculated as the mean of the minimum value for SpO_2 in each 30 s epoch of sleep (TST min.av. SpO_2). The minimum average SpO_2 was calculated for TST, NREM sleep time and REM sleep time.

Respiratory events leading to desaturation and increases in carbon dioxide occur predominantly in REM sleep in the CF population. These changes in carbon dioxide can be represented by the change in $TcCO_2$ from NREM to REM sleep, or the maximum $TcCO_2$ measured during the night compared with a baseline $TcCO_2$ reading. The change in $TcCO_2$ (mmHg) from NREM to REM sleep in our study was calculated for each REM period. Due to the drift often seen in the $TcCO_2$ trace, a line of best fit was drawn between four points on the $TcCO_2$ trace: one at the start and end of each REM period, taking into account the approximately 3 min time delay for the device, and a point 5 min prior to and following each REM period so long as the $TcCO_2$ reading was stable. A perpendicular line was then drawn to the peak $TcCO_2$ reading for that REM period being measured. A weighted average was then obtained for the delta $TcCO_2$ for each subject. $TcCO_2$ awake was the baseline value recorded at least 7 min after probe placement whilst the patient was breathing spontaneously. $TcCO_2$ maximum was the absolute maximum value of $TcCO_2$ recorded with periods of wakefulness and artefact excluded.

Four of the 37 patients included in this review of PSQI in CF had full polysomnography whilst wearing a nasal mask to facilitate measurement of ventilation. This could potentially alter the objective sleep parameters measured on polysomnography. Hence, when discussing the raw results from the polysomnogram and the relationship of these results with the PSQI, the data of 33 patients only are presented.

2.6. Data analysis and statistics

Mean values of each of the seven components of the PSQI and the global PSQI were calculated. To analyze for a relationship between anthropometric variables, ABGs, lung function variables, including respiratory muscle strength, and polysomnographic variables with both global and component scores of the PSQI, linear correlation analyses were performed [23].

Paired *t*-tests were used to test the null hypothesis that the PSQI raw estimates for sleep latency, duration and efficiency were the same as those measured on polysomnography. Unpaired *t*-tests were used to assess for potential differences in anthropometric, pulmonary function, respiratory muscle strength or ABGs between patients categorized as 'good' versus 'poor' sleepers. No adjustment was made for multiple testing. χ^2 analysis was used to assess the effect of gender upon PSQI [23]. Data are reported as the mean ± 1 SD. Statistical significance was assumed at $P < 0.05$.

3. Results

3.1. General

We administered the PSQI to 37 patients with CF, at the time of undergoing sleep studies. These patients were all in a stable clinical condition with forced expiratory volume in 1 s (FEV_1) ranging from 18 to 70% of predicted. The majority of these patients had severe lung disease due to CF with 27 of the 37 patients having an FEV_1 of <40%. The remaining ten patients had FEV_1 % predicted ranging from 40 to 70%. Twelve out of 37 patients were hypercapnic with $PaCO_2 \geq$ 45 mmHg. Four of the 37 patients were hypoxemic on room air with arterial oxygen tension (PaO_2) < 60 mmHg and 29 patients had a PaO_2 between 60 and 80 mmHg.

Anthropometric and daytime pulmonary function data are presented in Table 1. Data are presented with the mean ± 1 SD, and the range, for the entire group as well as with the patients divided into 'good' and 'poor' sleepers as defined by Buysse et al. [20].

3.2. PSQI

The mean PSQI in the CF patient group described in this study was 5.7 ± 4.0 (mean ± 1 SD) (Table 2). Fourteen out of the 37 patients (i.e. 38%) reported themselves to be 'poor' sleepers, i.e. to have a PSQI of >5. This group of 14 patients had PSQI scores ranging from 6 to 19, with a mean score of 9.7 ± 3.7. Table 2 contains the mean results for each component score of the PSQI and the global PSQI for all 37 patients, as well as the mean results when divided into 'good' and 'poor' sleepers as defined by Buysse et al. [20].

Pearson's correlation coefficients between global and

Table 1
Anthropometric data, blood gas tensions, and spirometry values in the 37 patients studied[a]

	PSQI ≤ 5 ($n = 23$)		*PSQI* > 5 ($n = 14$)		All patients ($n = 23 + 14 = 37$)	
	Mean ± 1 SD	Range	Mean ± 1 SD	Range	Mean ± 1 SD	Range
Sex (M, F)	14 M, 9 F	–	6 M, 8 F	–	20 M, 17 F[†]	–
Age (years)	26 ± 7	18–39	29 ± 9	18–49	27 ± 8[†]	18–49
BMI (kg/m^2)	19 ± 2	16–24	20 ± 3	16–24	20 ± 2	16–24
FEV_1 % predicted	38 ± 12	18–70	33 ± 10	19–52	36 ± 12[†]	18–70
FVC % predicted	61 ± 22	17–95	51 ± 16	31–77	57 ± 20	17–95
TLC % predicted	106 ± 17*	85–148	102 ± 17	60–128	104.6 ± 17.1*	60–148
RV % predicted	236 ± 50*	130–350	230 ± 59	148–339	233.5 ± 52.7*	130–350
$P_I$max % predicted	123 ± 31*	77–204	118 ± 48	61–241	120.7 ± 38.4*	61–241
$P_E$max % predicted	101 ± 37*	27–186	92 ± 24	53–128	97.8 ± 32.4*	27–186
KCO % predicted	115 ± 17*	87–150	103 ± 24**	48–149	110.3 ± 20.6*,**,[†]	48–150
PaO_2 evening (mmHg)	69 ± 8	54–87	66 ± 9	42–78	68 ± 9	42–87
$PaCO_2$ evening (mmHg)	43 ± 4	35–50	45 ± 6	35–58	43 ± 5[†]	35–58

[a] M, male; F, female; BMI, body mass index; FEV_1, forced expiratory volume in 1 s; FVC, forced vital capacity; TLC, total lung capacity; RV, residual volume; $P_I$max, maximal inspiratory pressure; $P_E$max, maximal expiratory pressure; KCO, carbon monoxide transferred per litre of lung volume; LFO_2, low flow oxygen. *Two patients did not undergo these test procedures due to infection control policy. **One patient did not undergo this test procedure due to severe hypoxia. [†]Significant correlations with component scores of PSQI, $P < 0.05$.

component scores were calculated. The three components that contributed most to the global PSQI were subjective sleep quality ($r = 0.8$, $P < 0.001$), habitual sleep efficiency ($r = 0.8$, $P < 0.001$), and sleep disturbances ($r = 0.8$, $P < 0.001$). The component that contributed the least to the global PSQI was use of sleeping medications ($r = 0.5$, $P < 0.01$). Very few patients in either group reported regular use of medications, prescribed or 'over the counter', to aid initiating or maintaining sleep. The areas that appeared to contribute most to the higher PSQI scores for the patients that reported themselves as 'poor' sleepers were subjective sleep quality, sleep latency and sleep disturbance. The mean value for each of these three components of the PSQI was 2 (Table 2).

3.3. Polysomnography results

Sleep architecture in these 33 patients who had polysomnography for diagnostic purposes (four excluded, see Section 2) was normal [24], with 20.7 ± 6.5% of TST spent in REM sleep (Table 3), and 19 ± 9% of slow wave sleep. The arousal index was also within normal limits [22] with 13.3 ± 5.2 events per hour (Table 3). Sleep efficiency was 87.1 ± 6.3%. Six patients had a sleep efficiency of between 70 and 80%, with the remainder of the group >80% (Table 4). The respiratory disturbance was greatest in REM sleep with a mean REM RDI of 11.9 ± 11.4, compared with a NREM RDI of 0.7 ± 1.2. Ten out of 33 patients had a REM RDI of >15 events per hour (Table 3). The group mean TST average minimum SpO_2 was 90.1 ± 4.4% and the mean absolute minimum SpO_2 was 82.5 ± 8.9% (Table 3). The group mean baseline awake $TcCO_2$ was 46 ± 7 mmHg, the change in $TcCO_2$ from NREM to REM sleep was 2.1 ± 1.6 mmHg and the maximum sleep $TcCO_2$ was 57 ± 15 mmHg.

3.4. Relationship of anthropometric, ABG, lung function and polysomnographic variables with component scores and global score of the PSQI

3.4.1. Age

In the CF population studied, age was positively corre-

Table 2
Component scores of PSQI, mean scores and ranges, plus global PSQI score ($n = 37$)

	PSQI ≤ 5 ($n = 23$) ('good' sleepers)		*PSQI* > 5 ($n = 14$) ('poor' sleepers)		All patients ($n = 23 + 14 = 37$)	
	Mean ± 1 SD	Range	Mean ± 1 SD	Range	Mean ± 1 SD	Range
Subjective sleep quality	0.3 ± 0.4	0–1	2.0 ± 1.2	0–3	0.9 ± 1.2	0–3
Sleep latency	0.7 ± 0.8	0–2	2.0 ± 0.8	1–3	1.2 ± 1.0	0–3
Sleep duration	0.3 ± 0.4	0–1	0.9 ± 0.9	0–3	0.5 ± 0.7	0–3
Habitual sleep efficiency	0.2 ± 0.4	0–1	1.4 ± 1.2	0–3	0.6 ± 1.0	0–3
Sleep disturbances	1.1 ± 0.4	0–2	2.0 ± 0.6	1–3	1.4 ± 0.7	0–3
Sleep medication	0.04 ± 0.2	0–1	0.1 ± 0.5	0–2	0.1 ± 0.4	0–2
Daytime dysfunction	0.7 ± 0.5	0–1	1.4 ± 0.9	0–3	1.0 ± 0.7	0–3
Global PSQI	3.3 ± 1.3	1–5	9.7 ± 3.7	6–19	5.7 ± 4.0	1–19

Table 3
Sleep variables as measured on polysomnography ($n = 33$)[a]

	Arousal index (events per hour)	REM sleep as % of TST	NREM RDI (events per hour)	REM RDI (events per hour)	TST RDI (events per hour)	TST min.av. SpO_2 (%)	Absolute minimum sleep SpO_2 (%)
Mean	13.3	20.7	0.7	11.9	3.1	90.1	82.5*
1 SD	5.2	6.5	1.2	11.4	3.4	4.4	8.9

[a] REM, rapid eye movement; TST, total sleep time; NREM, non-REM; RDI, respiratory disturbance index; TST min.av. SpO_2, TST minimum average SpO_2.
*Significant correlation between absolute minimum sleep SpO_2 and global PSQI ($r = -0.4$, $P < 0.02$).

lated with the component scores of subjective sleep quality ($r = 0.4$, $P < 0.05$) and sleep duration ($r = 0.3$, $P < 0.05$). As PSQI component scores increase from 0 to 3 with worsening sleep quality, these results infer that older patients have poorer sleep quality and shorter sleep duration than younger patients. There was no relationship between age and global PSQI score in this group.

3.4.2. Gender

Gender was positively correlated with the sleep disturbance component of the PSQI ($r = 0.4$, $P < 0.05$), where 'male' was ascribed the value of 1, and 'female' the value of 2. This result infers that female patients report greater sleep disturbance than male patients. No other significant correlations were found between gender and the global score for PSQI or its component scores.

3.4.3. Respiratory function variables, including respiratory muscle strength, and ABG tensions

FEV_1 % predicted was negatively correlated with subjective sleep quality ($r = -0.4$, $P < 0.05$). KCO % predicted was negatively correlated with the daytime dysfunction component of the PSQI ($r = -0.4$, $P < 0.01$) (Table 1). These results imply that the lower the FEV_1 % predicted and KCO % predicted, the worse the subjective sleep quality and daytime dysfunction, respectively. No other significant correlations were found between lung function or respiratory muscle strength variables and the global score for PSQI or its component scores.

ABG tensions showed a significant relationship between $PaCO_2$ and PSQI scores for sleep latency ($r = 0.4$, $P < 0.01$), habitual sleep efficiency ($r = 0.3$, $P < 0.05$) and global PSQI ($r = 0.4$, $P < 0.05$) (Fig. 1, Table 1). The higher the $PaCO_2$ therefore, the greater (i.e. the worse) the score for sleep latency, habitual sleep efficiency and global PSQI, inferring poorer sleep quality. In addition, there was a trend toward a significant relationship between PaO_2 and both subjective sleep quality and sleep latency ($r = -0.3$, $P < 0.1$ for both variables), meaning the greater the PaO_2, the lower (i.e. the better) the score for those component variables.

3.4.4. Polysomnography

There was a negative correlation between global PSQI and absolute minimum sleep SpO_2 % ($r = -0.4$, $P < 0.02$) (Table 3). In addition, the absolute minimum sleep SpO_2 % was negatively correlated with the sleep latency ($r = -0.4$, $P < 0.05$) and sleep duration ($r = -0.4$, $P < 0.05$) components of the PSQI, implying that the lower the minimum sleep SpO_2, the worse the overall sleep quality. The baseline awake value of $TcCO_2$ was positively correlated with the sleep duration component of the PSQI ($r = 0.45$, $P < 0.05$), i.e. the higher the $TcCO_2$, the worse the sleep duration. Baseline awake $TcCO_2$, the change in $TcCO_2$ from NREM to REM sleep, and the maximum sleep $TcCO_2$ were not correlated with the global PSQI or other components of the PSQI.

Paired *t*-tests showed that there was no difference in sleep efficiency between that measured on polysomnography and the raw value estimated in the PSQI, but that the estimated values for sleep latency and duration are greater than the values measured in the sleep laboratory ($t = 11.348$ and 104.2, respectively, $P < 0.05$ and $P < 0.0001$) (Table 4).

3.5. 'Good' versus 'poor' sleepers

Patients were categorized as 'good' or 'poor' sleepers

Table 4
Sleep efficiency, duration and latency, both measured and subjectively estimated ($n = 33$)

	Raw estimate for PSQI			Measured on sleep study		
	Sleep efficiency	Sleep duration	Sleep latency	Sleep efficiency	Sleep duration	Sleep latency
Mean	85.7	456.3[a]	22.7[a]	87.1	352.1	11.4
1 SD	11.2	87.9	23.7	6.3	34.9	9.4

[a] Raw estimate of sleep duration and sleep latency significantly different to measured sleep duration and latency on polysomnography, $t = 11.3$ and 104.2, respectively, $P < 0.05$ and $P < 0.0001$.

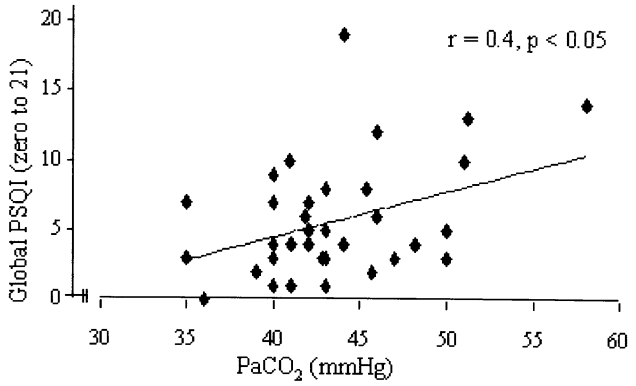

Fig. 1. Relationship between $PaCO_2$ (mmHg) and global PSQI.

based on their PSQI score. Those patients reporting a PSQI of ≤5 were categorized as 'good' sleepers, while those with a PSQI score of >5 were classed as 'poor' sleepers [20]. Using unpaired *t*-tests, there were no significant differences in anthropometric, pulmonary function, respiratory muscle strength or ABGs between the two groups. Although a greater proportion of female patients studied were 'poor' sleepers (eight out of 17) than male patients (six out of 20) (Table 1), this was not significant on χ^2 analysis for observed frequency of gender amongst 'good' versus 'poor' sleepers.

Assessing for differences in sleep architecture between the patients when grouped according to their PSQI score, there was better sleep efficiency ($P < 0.05$) and a greater % of REM sleep ($P < 0.05$) in those patients who subjectively reported themselves to be 'good' sleepers (Fig. 2). No differences were noted between the two patient groups for sleep latency, sleep duration, variables reflecting sleep oxygenation, $TcCO_2$, respiratory disturbance indices or arousal index (events per hour).

4. Discussion

This is the first study in which the PSQI has been used in patients with CF. The PSQI provided useful clinical information regarding sleep in patients with CF, and took very little time to administer and score. Importantly we found that many patients with CF had poor sleep quality as defined by a global PSQI score of >5. The areas contributing most to perceived 'poor' sleep quality were subjective sleep quality, sleep latency and sleep disturbance. The exploratory results from our study suggested that poorer sleep quality was associated with more severely impaired lung function and worse gas exchange. In particular, two lung function variables, FEV_1 % predicted and KCO % predicted, were negatively correlated with component scores of PSQI (subjective sleep quality and daytime dysfunction, respectively) suggesting an association between severity of lung disease, and poor sleep quality and perceived daytime dysfunction. Also, an increased $PaCO_2$, a known poor prognostic factor in CF [4,25], correlated positively with worsening of sleep latency, habitual sleep efficiency and global PSQI. Moreover, there was a relationship between absolute minimum sleep SpO_2 and the global PSQI score, and also two of its components. In summary, the PSQI score appeared to be related to measures of lung function and gas exchange.

When patients were grouped according to severity of reported sleep quality, the patients with low PSQI scores, i.e. better subjective sleep quality, showed significantly better sleep efficiency and a greater percentage of REM sleep on polysomnography than those with higher global scores. This result supports the usefulness of the PSQI in reflecting recorded sleep architecture. Spier et al. (1984) reported sleep quality on polysomnography to be poor in patients with CF with very severe lung disease (FEV_1 25 ± 9% predicted) compared with normal controls. Their

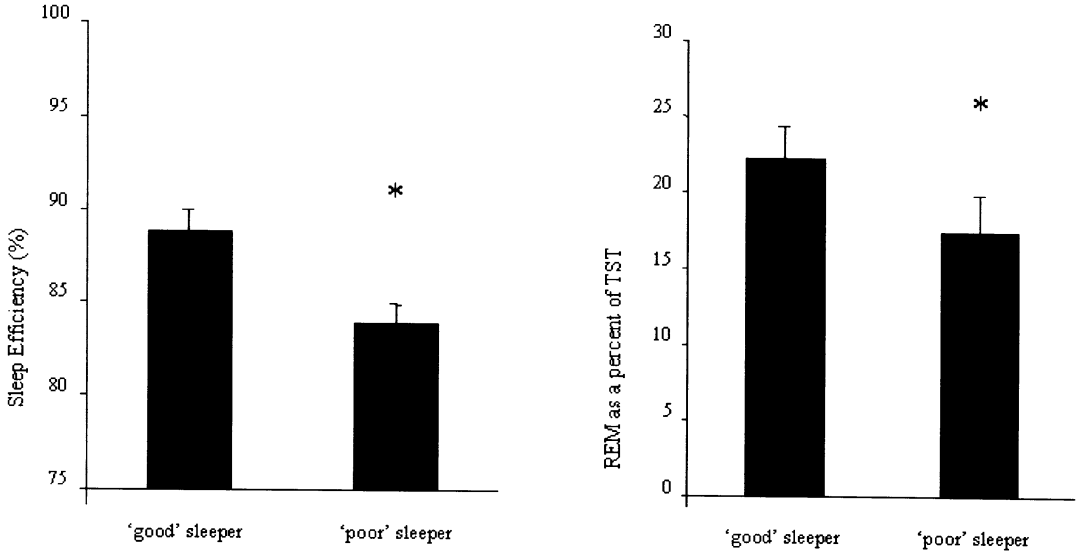

Fig. 2. Sleep efficiency and REM as a percent of TST: differences between patients when grouped according to PSQI score, with 'good' sleepers having a PSQI score of ≤5 and 'poor' sleepers scoring >5. *$P < 0.05$.

sleep efficiency was 58% (compared with 81% in controls), and they spent less time in REM sleep [7]. This is in contrast to our findings in which both subjective and objective measures of sleep efficiency were >85% and the amount of REM sleep was normal. These differences in results may in part reflect the fact that our patient group contained more patients with less severe lung disease. In addition, advances in CF treatment since 1984 could also indirectly explain our better results.

In this study of patients with CF and moderate to severe lung disease, a significant correlation between reported and measured sleep efficiency was found, although the estimated values for sleep latency and duration were greater than the values measured in the sleep laboratory. PSQI scores reflect sleep quality over the past month, while polysomnography looks only at the particular night/s studied. It is, therefore, important to note that these patients with CF were studied in a hospital sleep laboratory, and were told to go to bed at a time of their preference. This was often later than the time that they reported as their usual bedtime. This perhaps helps to explain why the measured sleep latency and sleep duration were shorter than subjectively reported as being representative of the previous month. In analyzing the validity of the PSQI, Buysse et al. (1989) looked at the relationship between polysomnographic data and reported sleep quality from the PSQI in a range of patients with sleep complaints as well as a normal control group [20]. The patients of Buysse et al. were not selected primarily on the basis of lung disease or significant nocturnal hypoxemia. They found no difference between the PSQI raw estimate of sleep latency and sleep laboratory findings for sleep latency, but PSQI estimates of the past month's usual sleep duration and efficiency were greater than those obtained during polysomnography. Previous studies in normal controls and patients with insomnia have assessed for similarities between reported and measured sleep quality, sleep efficiency and sleep duration, and found that although subjective estimates and objective measures often differ in actual amount, they are often strongly and positively correlated [26–29].

Dancey et al. (1998) presented, in abstract form only, the results of a study examining daytime sleepiness in CF using the Epworth Sleepiness Scale (ESS) and performing the Multiple Sleep Latency Test (MSLT) in addition to overnight polysomnography [30]. Like Spier et al. (1984) [7], these authors reported poor sleep quality on polysomnography in CF, with a decreased proportion of REM sleep and lower sleep efficiency than age-matched healthy controls. The patient group studied by Dancey et al. (1998) had very severe CF lung disease (FEV_1 28 ± 7% predicted) with nocturnal oxyhemoglobin desaturation, but they were unable to demonstrate any significant difference between CF and age-matched controls with regard to the ESS or MSLT [30]. By contrast, the present study did show a significant proportion of patients to be 'poor' sleepers using the PSQI.

We found that age correlated significantly with both the subjective sleep quality and sleep duration component scores of the PSQI. The relationship inferred from the result in this CF population is that with increasing age, reported sleep quality worsens and sleep duration decreases. This finding is in contrast to that of Buysse et al. (1989), who reported that subjective sleep quality improves with age in normal healthy controls [20]. As CF is a disease associated with progressive deterioration in lung function, and the relationship between numerous physiological variables describing lung disease severity and reported sleep quality has been demonstrated, the finding that older patients with CF report poorer sleep quality is perhaps intuitive. In addition, cough has been reported as coinciding with arousals in CF [5,7]. Cough and excessive mucus secretions may therefore contribute to sleep disruption. Accumulation of secretions overnight and nocturnal cough are likely to worsen with progressive disease, and hence age, in CF.

The primary analysis of the validity of the PSQI by Buysse et al. (1989) was to assess the degree to which the PSQI detected differences between their groups that were clinically recognized as different from the perspective of sleep quality [20]. They found that a cutoff score of 5 correctly differentiated 'good' from 'poor' sleepers. Interestingly, in the Buysse study (1989), there were no significant correlations between global PSQI score and polysomnographic results. When we used this cutoff in our patients with moderate to severe lung disease, there were no differences between 'good' and 'poor' sleepers with regard to lung function and ABG tensions. Rather, differences were found on polysomnography, in particular in sleep efficiency and % REM sleep time. When a cutoff of 7 was substituted, the same differences in polysomnographic variables were seen (% REM sleep and sleep efficiency), although age and $PaCO_2$ became significantly different between the groups. With the use of linear correlations, there was a relationship between markers of the severity of lung disease, such as FEV_1 % predicted and levels of CO_2 tension, with both component scores and global scores for PSQI. A significant correlation was also shown to exist between the absolute minimum sleep SpO_2 and the global PSQI as well as two of its components. To put it simply, the linear correlations suggest that subjective sleep quality is related to some markers of disease severity and objective polysomnographic measures of sleep quality.

Unlike the CF population, patients with chronic obstructive pulmonary disease (COPD) have been studied in considerable detail with regard to sleep quality. Both systematic inquiry [31] and objective assessment with polysomnography [32–34] have shown that patients with COPD sleep poorly in comparison to their age-matched controls. Interestingly, in contrast to our findings in CF, results of a questionnaire administered to 50 patients with severe COPD did not show any significant relationship between patient perception of sleep and measured sleep hypoxemia, although the patients with COPD did report more difficulty getting to sleep and more daytime sleepiness than their control group [31].

In conclusion, this study describes subjective sleep quality in a group of 37 adult patients with moderate to severe lung disease due to CF. Thirty-eight percent of these patients described themselves as having poor sleep quality. Relationships between self-reported 'poor' sleep and polysomnographic variables of sleep efficiency and % REM sleep time were found. Moreover, a relationship was shown between components of subjective sleep quality as measured by the PSQI and physiological variables describing disease severity, including FEV_1 % predicted, KCO % predicted, awake $TcCO_2$, daytime $PaCO_2$ and absolute minimum sleep SpO_2. Although these are exploratory results, and there is a need for confirmatory testing of the PSQI in this patient population, this initial study suggests a useful role for the PSQI in assessing sleep quality in patients with CF.

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