Nocturnal hypoxaemia and quality of sleep in patients with chronic obstructive lung disease

WESLEY CORMICK, LESLIE G OLSON, MICHAEL J HENSLEY, NICHOLAS A SAUNDERS

From the Faculty of Medicine, University of Newcastle, and Department of Thoracic Medicine, Royal Newcastle Hospital, Newcastle, New South Wales, Australia

ABSTRACT Fifty patients with chronic obstructive lung disease were questioned about their sleep quality and their responses were compared with those of 40 similarly aged patients without symptomatic lung disease. Patients with chronic obstructive lung disease reported more difficulty in getting to sleep and staying asleep and more daytime sleepiness than the control group. More than twice as many patients (28%) as controls (10%) reported regular use of hypnotics. In a subgroup of 16 patients with chronic obstructive lung disease (mean FEV₁ 0.88 (SD 0.44) l) sleep, breathing, and oxygenation were measured to examine the relationship between night time hypoxaemia and sleep quality. Sleep architecture was disturbed in most patients, arousals occurring from three to 46 times an hour (mean 15 (SD 14)/h). Arterial hypoxaemia during sleep was common and frequently severe. The mean (SD) arterial oxygen saturation (Sao₂) at the onset of sleep was 91% (7%). Nine patients spent at least 40% of cumulative sleeping time at an Sao₂ of less than 90% and six of these patients spent 90% of sleeping time below this level. Only four of 15 patients did not develop arterial desaturation during sleep. The mean minimum Sao₂ during episodes of desaturation was less in rapid eye movement (REM) sleep (72% (17%)) than in non-REM sleep (78% (10%); p < 0.05). The predominant breathing abnormality associated with desaturation was hypoventilation; only one patient had obstructive sleep apnoea. Arousals were related to oxygenation during sleep such that the poorer a patient's arterial oxygenation throughout the night the more disturbed his sleep (arousals/h v Sao₂ at or below which 40% of the total sleep time was spent: r = 0.71, p < 0.01). Hypoxaemia during sleep was related to waking values of Sao₂ and PaCO₂ but not to other daytime measures of lung function.

It is well recognised that sleep is associated with arterial oxygen desaturation in patients with chronic obstructive lung disease. Some physiological consequences of this desaturation, such as pulmonary hypertension, have been well investigated, reports of the consequences of arterial hypoxaemia in terms of quality of sleep and sleep related symptoms, however, are few and conflicting.

We report the perceptions of 50 patients with chronic obstructive lung disease of the quality of their sleep and compare these perceptions with those of a similarly aged population without lung disease but attending the outpatient department of a large urban hospital. In a subgroup of patients with chronic obstructive lung disease quality of sleep, as defined by electroencephalographic criteria, was compared with the degree of hypoxaemia experienced during sleep.

Methods

PATIENTS Fifty consecutive patients with chronic bronchitis or emphysema, or both, who were attending the chest clinic of the Royal Newcastle Hospital were interviewed about their sleep habits. Patients with asthma were excluded from the study. Patients with chronic obstructive lung disease were taking regular inhaled β₂ agonists, inhaled beclomethasone, or oral theophylline preparations (or more than one of these). Forty control patients of similar age but...
Nocturnal hypoxaemia and quality of sleep in patients with chronic obstructive lung disease

without symptomatic lung disease were interviewed during a routine attendance at the hospital's cardio-
logy (n = 4), hypertension (n = 25), or dermatology (n = 11) clinic. Patients attending these clinics were
taking various medications, including thiazide diuretics, β blockers, α methyldopa, clonidine, and peripheral vasodilators.

Sixteen of the patients with chronic obstructive lung disease gave consent for all night studies of
breathing and oxygenation during sleep. Patients were in a stable clinical state at the time of study.

All patients gave informed written consent for the studies, which were approved by the Royal Newcastle
Hospital ethics committee.

QUESTONNAIRES
All patients completed a 22 item questionnaire, administered by an assistant, about their usual
sleeping habits over the past six months. Patients did not know the interviewer. They were told that the
purpose of the study was to investigate sleeping habits in patients attending the outpatient department.
Questions related to initiation and maintenance of
sleep (for example, perceptions of sleep latency and
duration, frequency and causes of arousals, use
of hypnotics and sedatives), events during sleep (for example, snoring, restlessness, dreaming) and sleep
related daytime symptoms (for example, early morning headaches, daytime sleepiness, memory,
concentration).

The test-retest reliability of the questionnaire was examined in 15 patients by readministration of the
questionnaire two to six weeks after the initial inter-
view. Concurrent validity of the questionnaire was
assessed by interviewing the bed companions of 16
patients and seeking their response to items in the
questionnaire about which they could be reasonably
expected to have knowledge (for example, snoring,
akawenings).

The 16 subjects undergoing sleep studies also
completed a questionnaire relating to their percep-
tions of the preceding night's sleep.15 This question-
naire was completed on the morning after the second
sleep study, after one night's acclimatisation to the
laboratory, and allowed comparison of the patient's
perceptions with objective sleep recordings.

PULMONARY FUNCTION TESTING
All patients with chronic obstructive lung disease had
spirometric measurements (FEV₁/FVC) made with a
dry bellows spirometer (Vitalograph). In the 16
patients undergoing sleep studies total lung capacity
and its subdivisions were measured plethysmo-
graphically. Arterial blood was sampled by radial
artery puncture under local anaesthesia with the
patient seated. Artery blood gas values were deter-
mined in duplicate by an automated method (Radi-
ometer ABL-2). The ventilatory response to asphyxia
was measured by the method of Hensley and Read,16
and the ventilatory response to hypercapnia was mea-
sured by the rebreathing method of Read17; responses were measured at least in duplicate. Studies
of pulmonary function were performed on a different
day from the sleep studies.

SLEEP STUDIES

Patients were studied on two consecutive nights. They
settled down to sleep at their usual time and were
allowed to sleep through the night until they awoke
spontaneously. No patient was taking hypnotics at
the time of study. The first night was for acclimi-
atisation and no data were analysed from this night.
Sleep was monitored with silver cup electrodes
secured by collodion in standard positions for electro-
encephalography, chin electromyography, and
recording of eye movements by electro-oculo-
graphy.18 Arterial oxygen saturation (SaO₂) was
monitored continuously by a Hewlett Packard
47201A ear oximeter.19 20 Anteroposterior move-
ment of the chest and abdomen was measured by two
pairs of magnetometers (NP 1400Q) secured at the
level of the nipples and umbilicus respectively. The
magnetometers were not calibrated for volume and
hence provided only a qualitative assessment of
ventilation. Air flow at the nose and mouth was
assessed with thermistors (Grass TCT-1R) taped
securely to the face. Electrocardiographic monitoring
was performed continuously.

All signals were recorded on a 12 channel ink pen
recorder (Grass) at a paper speed of 5 mm/second.

ANALYSIS OF SLEEP STUDIES
Sleep was staged in 30 second epochs according to the
criteria of Rechtsaffen and Kales.18 Oxygenation
was assessed by analysing cumulative time—SaO₂
plots to derive (a) the cumulative percentage time
that the patient spent desaturated during sleep (% CT
desat), desaturation being defined as a reduction in
SaO₂ of 4% or more below the stable baseline SaO₂ at
onset of sleep; (b) the percentage cumulative time
spent with an SaO₂ of 85%, 80%, 75%, 70%, and
65% and (c) the SaO₂ at cumulative percentage time
of 90% (SatC₉₀) and 40% (SatC₄₀). SatC₉₀ defined
the SaO₂ at or below which the patient spent 90% of
the total sleep time (TST) and SatC₄₀ defined the
SaO₂ at or below which the patient spent 40% TST.

Records were also analysed in terms of discrete ep-
isodes of desaturation to compare the present results
with the results of previous studies. Each episode of
desaturation, defined as a fall of 4% or more from
the baseline sleeping SaO₂, was examined in terms of
duration, minimum SaO₂ reached, and breathing pat-
tern accompanying the fall in SaO2. Breathing patterns were identified as episodes of apnoea of central, mixed and obstructive types; hypoventilation with or without snoring, characterised by decreased deflections of the thermistor and magnetometer tracings; and periodic breathing, characterised by cyclic variation in magnetometer and thermistor tracings.

**Statistical Methods**

Differences in questionnaire responses between patients with chronic obstructive lung disease and controls were compared by means of \( \chi^2 \) analysis. For comparisons between test and retest results and between information given by patients and bed partners, kappa\textsuperscript{22} and weighted kappa\textsuperscript{23} scores were calculated as measures of agreement; the statistical significance of kappa was tested by Z score conversion. The significance of relationships within and between daytime and night time variables was assessed by Spearman rank correlation.

**Results**

**Perception of Sleep Quality Over Past Six Months**

Fifty patients (41 men, 9 women) with chronic obstructive lung disease were interviewed. Their mean (SD) age was 64 (9) years, FEV\textsubscript{1} 0.81 (0.34)\textsubscript{l}, and FVC 1.95 (0.75)\textsubscript{l}. Of the 40 control subjects, 30 were men and 10 were women; their mean age was 63 (8) years.

Responses to 11 of the 22 questionnaire items are shown in table 1. The 11 items not shown were qualitative in nature (for example, "What usually awakens you at night?"). Patients with chronic obstructive lung disease reported more difficulty getting to sleep and staying asleep, more use of hypnotics, and more daytime sleepiness than the control subjects. No differences were seen between the groups in reported duration of sleep, prevalence of snoring or dreaming, early morning headache, or daytime concentration.

**Test-retest reliability**

Good agreement was found between first and second administration of the questionnaire. The kappa score for each of the 22 items was positive, indicating agreement between the first and second occasion of testing. The degree of agreement (range 0.11–1.00) was greater than could be expected by chance alone (\( p \leq 0.05 \)) on 12 of the 22 items.

**Concurrent validity of the questionnaire**

Agreement was found between the 16 pairs of reports by patients and bed companions on the patient's sleeping habits for the six questionnaire items so tested. Kappa scores ranged from +0.18 to +1.00 and on four of the six items this agreement was significant (\( p \leq 0.05 \)).

**Sleep Studies**

**Patient characteristics**

The general characteristics of the 16 patients studied during sleep are shown in table 2. The patients' ages ranged from 48 to 84 years and all but one were men. Fifteen of the 16 were current cigarette smokers (n = 4) or ex-smokers. Six patients had hypercapnia (PaCO\textsubscript{2} > 45 mm Hg (6 kPa)) and seven had hypoxaemia (PaO\textsubscript{2} < 60 mm Hg (8 kPa)) while awake. The ventilatory responsiveness to asphyxia ranged from 0.10 to 1.8 1 min\textsuperscript{-1}%SaO\textsubscript{2}\textsuperscript{-1} and ventilatory response to hypercapnia ranged from 0.02 to 1.70 1 min\textsuperscript{-1} mm Hg\textsuperscript{-1}.

**Characteristics of sleep**

Only results from the second study night are reported (table 3, fig 1). The control values shown in figure 1 were not obtained in the present study but were taken from work done by others in different environ-

<table>
<thead>
<tr>
<th>Item</th>
<th>Patients (n = 50)</th>
<th>Controls (n = 40)</th>
<th>( \chi^2 )</th>
<th>( p )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Q1</td>
<td>Trouble falling asleep</td>
<td>18 (36)</td>
<td>6 (15)</td>
<td>5.01</td>
</tr>
<tr>
<td>Q2</td>
<td>Use of hypnotics regularly</td>
<td>14 (28)</td>
<td>4 (10)</td>
<td>4.50</td>
</tr>
<tr>
<td>Q3</td>
<td>Fall asleep minutes of lights out</td>
<td>19 (38)</td>
<td>25 (63)</td>
<td>5.78</td>
</tr>
<tr>
<td>Q4</td>
<td>Duration of sleep &lt; 6 hours</td>
<td>20 (40)</td>
<td>12 (30)</td>
<td>0.97</td>
</tr>
<tr>
<td>Q5</td>
<td>Snoring</td>
<td>24 (59)</td>
<td>22 (63)</td>
<td>0.66</td>
</tr>
<tr>
<td>Q9</td>
<td>Restlessness</td>
<td>21 (42)</td>
<td>7 (23)</td>
<td>7.78</td>
</tr>
<tr>
<td>Q10</td>
<td>Dreaming infrequent</td>
<td>26 (52)</td>
<td>13 (33)</td>
<td>3.61</td>
</tr>
<tr>
<td>Q12</td>
<td>Awakenings &gt; 2/night</td>
<td>38 (76)</td>
<td>21 (53)</td>
<td>4.43</td>
</tr>
<tr>
<td>Q16</td>
<td>Daytime sleepiness</td>
<td>36 (72)</td>
<td>12 (30)</td>
<td>15.75</td>
</tr>
<tr>
<td>Q19</td>
<td>Early morning headaches</td>
<td>14 (28)</td>
<td>5 (13)</td>
<td>3.21</td>
</tr>
<tr>
<td>Q21</td>
<td>Impaired daytime concentration</td>
<td>16 (32)</td>
<td>11 (28)</td>
<td>0.21</td>
</tr>
</tbody>
</table>

* Nine patients and five controls did not know whether they snored.
ments.\textsuperscript{24,25} The wide range of values of total sleep period (TSP) among patients in the present study despite approximately equal total sleep duration indicates that some patients had great difficulty falling asleep whereas others fell asleep easily. The percentage of REM sleep varied from zero to 30-4% TSP but on average the REM sleep period was about 20% of the time spent asleep. Sleep architecture was disturbed in most patients; the number of sleep state changes ranged from 7 to 46 per hour TST and arousals to wakefulness from 3 to 46 per hour TST.

Table 2. Characteristics of the 16 patients with chronic obstructive lung disease taking part in sleep studies

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Mean</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>64.8</td>
<td>9.7</td>
</tr>
<tr>
<td>FEV(_1) (l)</td>
<td>0.88</td>
<td>0.44</td>
</tr>
<tr>
<td>FVC (l)</td>
<td>2.06</td>
<td>0.77</td>
</tr>
<tr>
<td>TLC (% predicted)</td>
<td>117</td>
<td>24</td>
</tr>
<tr>
<td>RV (% predicted)</td>
<td>197</td>
<td>64</td>
</tr>
<tr>
<td>%SaO(_2)</td>
<td>89</td>
<td>7</td>
</tr>
<tr>
<td>PaO(_2) (mm Hg)</td>
<td>64</td>
<td>17</td>
</tr>
<tr>
<td>PaCO(_2) (mm Hg)</td>
<td>43</td>
<td>8</td>
</tr>
<tr>
<td>(dV_i/dSao_2 ) (l min(^{-1}) %Sao(_2)(^{-1}))</td>
<td>0.65</td>
<td>0.61</td>
</tr>
<tr>
<td>(dV_i/dPaco_2 ) (l min(^{-1}) mm Hg(^{-1}))</td>
<td>0.61</td>
<td>0.62</td>
</tr>
</tbody>
</table>

FVC—forced vital capacity; TLC—total lung capacity; RV—residual volume; %SaO\(_2\)—% arterial oxygen saturation (calculated); PaO\(_2\)—arterial oxygen tension; PaCO\(_2\)—arterial carbon dioxide tension; \(dV_i/dSao_2\)—ventilatory response to asphyxia; \(dV_i/dPaco_2\)—ventilatory response to hypercapnia.

Conversion: Traditional to SI units—Blood gas tensions: 1 mm Hg \(\approx\) 0.133 kPa.

Relation between perceived quality of sleep and electroencephalogram

No significant differences were observed between patients’ perceptions and objective measures of sleep latency (mean (SD) 19 (15) \(\pm\) 30 (11) min), total sleep period (283 (111) \(\pm\) 316 (95) min), or number of arousals lasting longer than five minutes (3.2 (1.9) \(\pm\) 5.3 (3.1), Wilcoxon matched pairs test). When all EEG records of arousals were included in the analysis (table 3), subjective and objective reports differed significantly (\(p < 0.01\)).

Oxygenation during sleep

Table 4 shows the severity of arterial oxygen desaturation during the second night of study. Oximetry data were not available for one patient owing to malfunctioning of the equipment. Four patients did not develop desaturation during sleep; these patients had awake baseline SaO\(_2\) levels of 95%, 95%, 95%, and 90%. The remaining 11 patients spent 4–98% of sleeping time with an SaO\(_2\) more than 4% less than the baseline level (%CT\(_{\text{desa}}\)). Nine patients spent 40% of their sleeping time with an SaO\(_2\) below 90% (Sat\(_{\text{CT40}}\)) and six patients spent 90% of their sleeping time below this level (Sat\(_{\text{CT90}}\)). As a group, patients spent 31.2% (42.3%) TST (mean (SD)) at or below 85% SaO\(_2\), 26.1% (39.9%) TST at or below 80% SaO\(_2\), 19.0% (34.6%) TST at or below 75% SaO\(_2\), 15.4% (32.0%) TST at or below 70% SaO\(_2\), and 12.7% (29.0%) TST at or below 65% SaO\(_2\). The large
Table 3 Characteristics of the sleep of 16 patients with chronic obstructive lung disease

<table>
<thead>
<tr>
<th>Sleep variable</th>
<th>Mean (SD) Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>TSP (min)</td>
<td>316 (95) 135–461</td>
</tr>
<tr>
<td>TST (min)</td>
<td>208 (88) 50–323</td>
</tr>
<tr>
<td>Wakefulness (%)*</td>
<td>35 (19) 13–65</td>
</tr>
<tr>
<td>Non-REM sleep (%)*</td>
<td>52 (14) 30–73</td>
</tr>
<tr>
<td>REM sleep (%)*</td>
<td>13 (9) 0–30</td>
</tr>
<tr>
<td>Sleep state change/h TST</td>
<td>17 (12) 7–54</td>
</tr>
<tr>
<td>Total No of arousals (&gt; 15 seconds)</td>
<td>37 (25) 15–103</td>
</tr>
<tr>
<td>No of arousals/h TST</td>
<td>15 (14) 3–46</td>
</tr>
<tr>
<td>Mean duration of uninterrupted sleep periods (min)</td>
<td>8 (5) 1–18</td>
</tr>
</tbody>
</table>

TSP—total sleep period, from onset of sleep to last awakening, including time spent in all intervening awakenings; TST—total sleep time (total sleep period minus intervening wakefulness); REM—rapid eye movement.
*Percentage of total sleep period.

The large standard deviations show that the cumulative time spent at any given Sao2 varied widely among patients. Indeed, nine patients did not spend any of their sleeping time at an Sao2 less than 80%.

Three of the 16 patients had multiple episodes of apnoea of more than 10 seconds' duration during sleep but in only one were the criteria of sleep apnoea achieved.26 Apnoea in this patient occurred almost exclusively in REM sleep. The three patients in whom apnoea were observed were all snorers and all their episodes of apnoea were obstructive in type. Most of the episodes of worsening hypoxaemia observed in the 11 patients who developed desaturation were associated with hypoventilation, with or without snoring. One patient’s dominant breathing pattern was cyclical ventilation. In the 11 patients who developed desaturation during sleep arterial oxygenation was worse during REM sleep than during non-REM sleep (mean (SD) minimum Sao2 during episodes of desaturation (Sao2 72-3% (17-0%) v 78-2% (10-2%); p < 0.05, paired Student’s t test).

RELATIONSHIP BETWEEN WAKING PULMONARY FUNCTION AND SLEEPING HYPOXÆMIA

No relationship was found between nocturnal hypoxæmia and severity of lung disease (as judged by spirometry and lung volumes) or ventilatory responsiveness. A relationship was found, however, between waking Sao2 and sleeping hypoxæmia, whether this was expressed as a continuously distributed variable, SatCT40 (r = 0.85, p < 0.01) or as mean minimum Sao2 during episodes of desaturation (r = 0.82, p < 0.01)—fig 2). An inverse relationship existed between

---

**Fig 2** Relation between nocturnal hypoxæmia and "awake" arterial oxygen saturation (Sao2—left and middle panels) and arterial carbon dioxide tension (Paco2—right panel). Each point represents one patient. 1 mm Hg ≈ 0.133 kPa.
Nocturnal hypoxaemia and quality of sleep in patients with chronic obstructive lung disease

The relationship between arousals and nocturnal hypoxaemia was examined in two ways. Firstly, the number of arousals/hour TST was related to \%CT_{desat}, Sat_{CT90}, Sat_{CT40}, number of episodes of desaturation/hour TSP, and mean minimum \% Sao2 during desaturation. Arousals were related inversely to Sat_{CT90} (r = 0.63, p < 0.05) and Sat_{CT40} (r = 0.71, p < 0.01) (fig.3) but not to the other variables. Secondly, the relationship between intervening wakefulness and number of hypoxaemic episodes was examined; no significant relationship was found (fig.4). Thus, while arousals were related to overall oxygenation during sleep such that those with the lowest arterial oxygenation had the most frequent arousals, no relationship existed between arousals (or intervening wakefulness) and discrete episodes of desaturation.

Relationship between sleeping hypoxaemia and perception of sleep
In the 16 patients who underwent sleep studies, questionnaire responses were related to measurements of severity of hypoxaemia during sleep, \( \chi^2 \) analysis being performed after patients had been assigned to two groups according to the median values of \%CT_{desat}, Sat_{CT90}, Sat_{CT40}, number of episodes of desaturation/hour TST, and mean minimum \% Sao2 during desaturation. No significant relationships were observed.

Graph 3
Relation between arousals from sleep and nocturnal hypoxaemia expressed as saturation at cumulative time 90\% (Sat_{CT90}—left panel) and saturation at cumulative time 40\% (Sat_{CT40}—right panel) (see under “Methods”).

Graph 4
Relation between mean duration of episodes of uninterrupted sleep and discrete episodes of arterial desaturation (\( \text{Sao}_2 \geq 4\% \) less than baseline \( \text{Sao}_2 \)). Each point is one subject.

waking PaCO\(_2\) and sleeping oxygenation (p < 0.01). Thus those patients with a lower \% Sao2 and a higher PaCO\(_2\) when awake spent their sleeping time at a lower level of arterial oxygenation and had greater arterial hypoxaemia during episodes of disordered breathing.
Discussion

The results of this study show that patients with chronic obstructive lung disease report poorer quality sleep than other patients of similar age attending a large urban outpatient department. In patients with chronic obstructive lung disease nocturnal hypoxaemia is common and frequently severe, and is related to arousal from sleep. Hypoxaemia during sleep is most severe in those patients with lower Sao2 and higher PaCO2 while awake during the day, but it is not related to other routine measures of daytime pulmonary function. It does not appear possible to predict those at risk of severe arterial desaturation during sleep from patients' own reports of sleep related symptoms.

There are problems associated with the use of questionnaires to examine sleeping habits. Most studies reporting perceptions of sleep have not provided information relevant to the validity or reliability of the questionnaire. The questionnaire used in the present study had acceptable test-retest reliability and there was good agreement between patients and bed partners on the limited number of items so tested. Too much emphasis should not, however, be placed on this latter point since some of the patient's information about sleeping habits almost certainly came from the bed partner (for example, snoring). Comparison of immediate perceptions of sleep quality with objective variables defined by EEG showed that our patients' perceptions were reasonably accurate when considered as a group. This finding supports the results of Johns,27 who studied sleep latency in normal subjects. He found no difference between group mean values of sleep latency derived from objective measurements (that is, EEG) and from subjective reports of the night's sleep. Johns27 also showed a significant correlation between objective measurements of sleep latency in the laboratory and habitual sleep latency at home as reported by questionnaire.

Our choice of control subjects can be criticised on several grounds. Although free from breathlessness, control subjects may have had mild left ventricular failure and others were taking antihypertensive medication that is known to cause sedation. Moreover, drugs taken by the patients with chronic obstructive lung disease may have interfered with their sleep. These drawbacks are unlikely to negate our findings. The possible inclusion of controls with left ventricular failure should have minimised the possibility of finding differences in sleep behaviour between patients with lung disease and controls. Patients with chronic obstructive lung disease were sleepier during the day than controls, despite the use of potentially sedating antihypertensive medication by some controls. Miles and Dement28 have recently reviewed studies of the perception of sleep in elderly people. Sleep complaints are frequent in this population, the most consistent finding being an increased number of night time awakenings. Other complaints reported frequently by the elderly include difficulty in falling asleep, increased use of hypnotics, increased early morning waking, and decreased daytime function. The perception of sleep by our control subjects is very similar. The greater prevalence of difficulty in getting to sleep, restlessness with more frequent awakenings, and daytime sleepiness in patients with chronic obstructive lung disease cannot be attributed to age alone.

Laboratory studies support the perceptions of the elderly that their sleep is more disturbed than the sleep of younger groups of people. Total sleep time and slow wave sleep decrease with age and arousals are more frequent throughout the night.28,29 Sleep appears to be even more disturbed in patients with chronic obstructive lung disease.10-12 Our findings are in general agreement with these studies, although our patients had more intervening wakefulness and less non-REM sleep than previously reported. These objective data from several centres mirror the patients' perceptions reported here that sleep is difficult to initiate and maintain.

The cause of disturbed sleep in patients with chronic obstructive lung disease remains unclear and is likely to be multifactorial. We have shown an association between arousals and severity of sleeping hypoxaemia. This finding is at variance with the results of Calverley and coworkers11 but supports the findings of Fleetham and coworkers,10 who showed a statistical association between arousals and episodes of desaturation. The picture is far from clear, however. Calverley and coworkers11 found that patients with the greatest number of episodes of hypoxaemia had the least intervening wakefulness but showed that oxygen administration improved sleep, which is in agreement with the findings of Kearley and associates.13 By contrast, although Fleetham and coworkers10 found an association between episodes of hypoxaemia and arousals, they found no effect of oxygen administration on sleep and a more recent study confirms this, although there was a trend towards longer sleep time with oxygen treatment.14 Comparisons between studies are made difficult by differences in the way hypoxaemia during sleep is expressed.30 We found an association between arousals and hypoxaemia only when Sao2 was expressed as a continuous variable. We emphasise that we have not established a causal relationship between hypoxaemia and sleep fragmentation in our patients. Indeed, review of individual records showed that some patients had multiple arousals with minimal
Nocturnal hypoxaemia and quality of sleep in patients with chronic obstructive lung disease

This work was supported by grants from the National Health and Medical Research Council, Australia. Mrs Joy Peate provided expert secretarial assistance. Ms Lisa Benson provided valuable technical assistance in the conduct of the studies and analysis of records. We thank Drs K Murree Allen, M Saltos, R Mills, and M Deacon for referring us patients for study, and we especially thank the patients who so willingly agreed to participate.

References

17 Read DJC. A clinical method for assessing the venti-


29 Brezinova V. The number and duration of the episodes of the various EEG stages of sleep in young and older people. Electroencephalography and Clinical Neurophysiology 1975;39:273–8.


Nocturnal hypoxaemia and quality of sleep in patients with chronic obstructive lung disease.

W Cormick, L G Olson, M J Hensley and N A Saunders

Thorax 1986 41: 846-854
doi: 10.1136/thx.41.11.846

Updated information and services can be found at:
http://thorax.bmj.com/content/41/11/846

These include:

Email alerting service
Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

Notes

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/