Predictive value of clinical features for the obstructive sleep apnoea syndrome

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Predictive value of clinical features for the obstructive sleep apnoea syndrome. P.C. Deegan, W.T. McNicholas. ©ERS Journals Ltd 1996.
ABSTRACT: The advantage of being a National Referral Centre for patients with suspected obstructive sleep apnoea (OSA) was used to seek clinical factors predictive of OSA, and thus determine if the number of polysomnography tests required could be reduced. Patients were mainly primary referrals, from an island population of 3.5 million.

Two hundred and fifty consecutive patients underwent clinical assessment, full polysomnography, and a detailed self-administered questionnaire. This represents one of the largest European studies, so far, utilizing full polysomnography.

Fifty four percent (n=134) had polysomnographic evidence of OSA (apnoea/hypopnoea index (AHI) ≥15 events·h^{-1} sleep). Patients with OSA were more likely to be male, and had a significantly greater prevalence of habitual snoring, sleeping supine, wakening with heartburn, and dozing whilst driving. Alcohol intake, age and body mass index (BMI) were significant independent correlates of AHI. After controlling for BMI and age, waist circumference correlated more closely with AHI than neck circumference among males, while the opposite was true among females.

No single factor was usefully predictive of obstructive sleep apnoea. However, combining clinical features and oximetry data, where appropriate, approximately one third of patients could be confidently designated as having obstructive sleep apnoea or not. The remaining two thirds of patients would still require more detailed sleep studies, such as full polysomnography, to reach a confident diagnosis.
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Obstructive sleep apnoea (OSA) has an estimated prevalence of 0.3–4%, depending on the population studied and criteria used [1, 2]. Diagnosis is currently based on a combination of clinical features and overnight polysomnography (PSG). This approach has its limitations, as polysomnography is expensive, time-consuming and labour intensive. In addition, the number of patients referred for assessment is rising, with the increasing awareness that OSA is common and can lead to adverse medical consequences if left untreated [3, 4]. If it was possible to predict the likelihood of having OSA based on history and physical examination alone, then utilization of sleep laboratory facilities could be improved and the number of unnecessary sleep studies reduced.

Several previous studies have examined the predictive value of clinical features [1, 5–14]. However, there is considerable variabilty in the findings, which may be partly explained by differences in study design. In some reports, the subject numbers were small. The study population was often heterogeneous, with subjects from a number of different ethnic, racial and social groups. Patients were often "preselected" during the referral process, so that those who were more likely to have OSA were assessed and not all patients underwent full

polysomnography. Furthermore, there have been few studies involving a European population in which full polysomnography was performed [9, 11, 14].

We utilized the advantage of being a national referral centre for an island population of 3.5 million, to carry out a detailed prospective assessment of 250 consecutive patients referred to a respiratory sleep laboratory with symptoms suggestive of sleep apnoea. All the patients studied were white Northern European, with free access to a good level of health-care and a good basic level of education. Most were referred from practitioners without any specialist knowledge of respiratory sleep disorders, and all underwent full polysomnography.

Methods

Consecutive patients, aged 18 yrs or over, referred to the respiratory sleep laboratory because of a suspicion of sleep-disordered breathing, were assessed by one investigator at their first visit. Any patient who had been referred with one or more of: excessive daytime somnolence (EDS), snoring or observed apnoeas was, thus, included in the study and underwent full assessment, even if the likelihood of a significant respiratory sleep

disorder was considered low. Any patients referred for the investigation of insomnia were not included.

The upper airway was examined for any abnormality that could contribute to airway narrowing, such as a deviated nasal septum or a small oropharyngeal airway. Blood pressure was measured in the semirecumbent position, in the late morning or early afternoon, with the patient relaxed and the appropriate sized pressure cuff used. Simple spirometry was also performed using a dry wedge spirometer (Vitalograph Ltd, Buckingham, UK) or an automated spirometer (Microlab 3300, Micro Medical, Rochester, UK). All patients then attended for an overnight sleep study, in order to objectively quantify any sleep apnoea.

Questionnaire

On the evening of the sleep study, patients completed a self-administered questionnaire with 23 sections in all (75 questions). These included questions about symptoms normally associated with sleep apnoea, such as snoring, excessive daytime somnolence (EDS), nocturnal choking, observed apnoeas, sleep disruption and restlessness. In addition, a number of questions were related to symptoms of sleep disorders in general (such as hypnagogic hallucinations), the effects of disturbed sleep on daytime mood and performance, and on a number of other factors thought to have a possible relationship with sleep apnoea. Such factors included reduced sexual drive, gastro-oesophageal reflux, predominant sleeping position, nocturia, nasal obstruction and headaches. Finally, there were inquiries about each patient's tobacco and alcohol consumption, about work and exercise habits, and about their past medical and family histories.

Questions were presented in a simple, straightforward fashion, without medical terminology. Most questions requested the patient to quantify symptoms with an ordinal three point scale: "never", "a minor or infrequent problem", or "a major or frequent problem". The frequency of snoring was quantitated on a four-point scale: "never", "occasionally", "most but not all nights", or "every night". The degree of EDS was measured in a number of different circumstances. A number of questions required yes/no replies, while other questions involved quantifying a continuous variable, such as amount of alcohol consumed per week.

We were concerned that patients complete the questionnaire themselves, without direct influence from the technician overseeing the questionnaire. In some cases, patients were unable to answer a particular question as they did not have the information. When this occurred, questions concerning quantitative variables were left blank, and those relating to categorical variables were scored as no/never.

Anthropometry

After the questionnaire had been completed, the technician then measured the patients' height, using a wall-fixed height rule, and weight (with a lever balance scales,

Seca 220, Germany). Body mass index (BMI) was then calculated for each patient (weight in kg/square of height in m). The neck circumference was measured in all patients, at the level of the cricothyroid membrane, and abdominal circumference at functional residual capacity (FRC) and at the level of the umbilicus in 169 patients.

Sleep studies

Overnight sleep studies were carried out using standard polysomnographic techniques [15], usually from midnight to 7 am. An acclimatization night was not performed. However, if a subject had less than 3 h of total sleep, the sleep study was repeated and data from the second study used in the final analysis. Two leads of electroencephalogram (C_4A_1; C_3/A_1), two leads of eye movement, and a submental electromyogram, were continuously recorded from surface electrodes. Respiration was also recorded continuously using a respiratory inductance plethysmograph (Respitrace®: Ambulatory Monitoring Inc., Ardsley, NY, USA), calibrated using the isovolume technique. Apnoeas were defined as absence of movement in the sum channel of the Respitrace lasting at least 10 s, and a hypopnoea defined as a period of 10 s or more when the tidal volume abruptly fell to less than 50% of the average tidal volume for the preceding 30 s, in association with a fall in arterial oxygen saturation ($\%S$a,O_2) of at least 4% and/or an electroencephalographic arousal.

All variables were recorded continuously, either on a polygraph recorder (Model 78D; Grass Instruments, Quincy, MA, USA), or using a computerized system (Medilog SAC 847 system; Oxford instruments, Abingdon, UK). Ear oximetry was used to continuously measure $\%S$a,O_2. In the case of patients studied with the Grass system, a Biox 3700 oximeter was used (Ohmeda, Louisville CO, USA) and the data recorded on a separate chart recorder set at 30 cm·h^{-1}. Where data were acquired using the computerized system, the system's own integral oximeter was used and data were obtained from the automated record.

Sleep studies were scored without knowledge of the data from the questionnaires or physical examinations. Studies using the Grass system were analysed manually by an experienced technician. In the case of the computerized set-up, the study was scanned by one of the investigators and corrected manually where appropriate. An apnoea/hypopnoea index (AHI) of 15 events·h^{-1}, was taken as the minimum diagnostic criterion for clinically significant OSA [6, 16, 17].

Statistics

All patients in the study were divided into two groups according to their AHI, namely OSA (AHI ≥15 events·h^{-1}) and non-OSA (AHI <15 events·h^{-1}). The differences in distribution of multilevel categorical data between the two groups were initially analysed using the r×c chi-squared test for trend, to take account of the ordering used (i.e. 2×3 and 2×4 chi-squared tests). Where a significant

difference was seen between the two groups, further 2×2 chi-squared tests were performed, with a Yates correction, to isolate which groups in the contingency table accounted for the overall effect. These tests involved comparing the numbers answering "yes" to a question, irrespective of grade, *versus* those answering "no" (*i.e.* 1/2 *vs* 0). This procedure established the significance of having a specific factor. The other comparison involved comparing those with the higher grade, 2, against the others (*i.e.* 2 *vs* 0/1), to see if an answer with a higher degree of positivity was more predictive. A Bonferroni correction for multiple comparisons was used, where appropriate. The positive and negative predictive values for any variable showing a significant difference between the two groups were then calculated using Bayes' theorem.

The relationships between AHI and individual quantitative variables were assessed by single factor regression analysis. Stepwise multiple linear regression analysis was then performed, with AHI as the dependent variable. Independent variables entered into the equation included: age, BMI, weekly alcohol consumption, neck circumference, forced expiratory volume in one second (FEV1) and FVC. The distribution of AHI showed a statistically significant deviation from normality by the Shapiro-Wilks test. Therefore, the nonparametric Spearman Rank correlation test was used in single factor regression analyses. When AHI was the dependent variable in the multiple linear regression equation, square root transformation was performed to reduce this deviation from normality.

Logistic regression analysis was then employed to develop a predictive model for the determination of probability of having OSA from a number of clinical variables. In this test, the presence or absence of OSA (as determined by polysomnography) is the dependent variable, and is expressed as one of two probable outcomes (OSA=1, non-OSA=0). Thus, the probability (p) of an individual patient having OSA (AHI $\geq$15 events·h^{-1}), is determined as:

$$p = (e^y) / (1 + e^y)$$

where y=constant (c) + x_1Variable1+x_2Variable2+x_3Variable3...is the predicted value of y for a given set of values for the explanatory variables. We repeated the logistic regression analysis using the minimum AHI criteria quoted in other reports, namely AHI events·h^{-1} $\geq$10 [7, 8, 10, 13, 14, 18] and AHI $\geq$20 events·h^{-1} [12].

These analyses were performed using the software package Statistica for Windows Release 4.5 (Statsoft Inc., 1993). Results are quoted as means with standard deviations in parentheses. A significant value was taken as p-value less than 0.05. Ninety five percent confidence intervals (95% CIs) are provided where appropriate.

Results

A total of 250 patients was studied. The majority of patients had been referred by their family practitioner (66%). Only 14% had been referred by an ear, nose and throat (ENT) specialist and 7% by a respiratory physician.

Of the remainder, 2% had been referred by a neurologist and 11% by a specialist in another area. One hundred and fifty two patients were assessed using the manual Grass system and 98 by the Oxford system. A total of 136 patients (54%) had an AHI $\geq$15 events·h^{-1} and these patients were assigned to the OSA group, while 114 (46%) had an AHI <15 events·h^{-1}, and were assigned to the non-OSA group. There were no significant differences in the proportion of patients tested by the semiautomatic system assigned to the OSA group compared to those assessed by the manual system. Anthropometric, spirometric and PSG data for both groups (OSA and non-OSA) are presented in table 1.

Questionnaire

Variables showing a significant difference between the two groups, OSA and non-OSA, are shown in table 2. With the exception of snoring, grading a "yes" response had no additional benefit. We did not study the highest against the lowest (0 *vs* 2), as this would have excluded a large number of patients from analysis. Both average number of units of alcohol, and weight gain over the last 5 yrs were significantly correlated with AHI (table 3). Two other variables approached significance (p<0.1), namely, falling asleep whilst reading and reduced libido.

The categorical variables shown in table 2 remain significantly different between OSA and non-OSA groups, when reanalysed with a minimum AHI criterion for OSA of either 10 or 20 events·h^{-1}, with the exception of "falls asleep driving" where an AHI of 20 events·h^{-1} was used (NS). Positive predictive values increased (71–90%) and negative predictive values decreased (39–46%) at the lower AHI, whilst positive predictive values decreased (50–70%) and negative predictive values increased (57–71%) at the higher AHI.

Table 1. – Anthropometric, spirometric and polysomnographic data

	Non-OSA		OSA	
Subjects	114	(46%)	136	(54%)
Sex M/F*	82:32		119:17	
Age yrs	45	(13)	50	(11)
BMI kg·m^{-2}	28.1	(5)	29.2	(5.1)
% ideal body weight	115	(25)	118	(20)
FEV1 % pred	98	(19)	94	(18)
FVC % pred	97	(15)	95	(15)
FEV1/FVC %	81	(8)	79	(12)
PEFR % pred	99	(31)	95	(21)
AHI events·h^{-1}†	5	(0–14)	33	(15–96)
Total dips in S_{a,O_2} $\geq$4% n	16	(19)	133	(124)
Lowest S_{a,O_2} %	88	(6)	79	(10)

Data are expressed as mean, and SD in parenthesis. *: p=0.003 (χ^2 test); odds ratio=2.7; 95% CI=1.4–5.3. †: values are median (and range in parenthesis). OSA: obstructive sleep apnoea; M: male; F: female; BMI: body mass index; FEV1: forced expiratory volume in one second; FVC: forced vital capacity; PEFR: peak expiratory flow rate; % pred: percentage of predicted value; AHI: apnoea/hypopnoea index; S_{a,O_2}: arterial oxygen saturation; 95% CI: 95% confidence interval.

Table 2. – Categorical questionnaire data showing significant differences between sleep apnoea and non-sleep apnoea patients

	χ^2 for trend	χ^2 with Yates' correction	OR (95% CI)	Predictive value % Positive	Negative
Observed apnoeas	p<0.001	p=0.049 (1/2 vs 0)*	2 (1.1–3.8)	64	53
Snoring	p<0.005	p=0.004 (3 vs 0–2)*	2.3 (1.4–3.9)	63	56
Weight increase as snoring worsened	p<0.005	p=0.006 (1/2 vs 0)*	2.2 (1.3–3.7)	64	55
Sleeping position	p<0.025	p=0.04 (supine vs others)*	3 (1.2–7.2)	77	47
Awoken with heartburn	p<0.025	p=0.01 (1/2 vs 0)*	2.6 (1.4–4.8)	71	50
Falls asleep driving	p<0.05	p=0.008 (1/2 vs 0)*	2.5 (1.4–4.4)	70	51

OR: odds ratio; 95% CI: 95% confidence interval. *: see results (Questionnaire) for details.

Table 3. – Single factor regression analysis expressing the relationship between quantifiable variables and AHI

	Spearman Rank correlation test r	p-value
Age yrs	0.22	<0.001
BMI $kg \cdot m^{-2}$	0.18	0.004
% ideal body weight	0.15	0.02
Weight gain over last 5 yrs kg	0.20	0.002
Waist circumference cm	0.35	<0.001
Neck circumference cm	0.27	<0.001
FEV_1 % pred	-0.26	0.01
FVC % pred	-0.16	0.04
Units of alcohol per week*	0.28	<0.001

*: one unit=8.0 g of absolute alcohol. For abbreviations see legend to table 1.

The tendency to doze in a number of other circumstances, such as watching television, attending meetings, out socially or eating, occurred equally in both groups. No significant differences were detected with questions related to the effects of EDS, nocturnal sleep disruption, abnormal motor activity during sleep, personality changes, nocturia, nocturnal enuresis, nasal symptoms and headaches.

More patients with OSA were male (odds ratio (OR)=2.7; 95% CI 1.4–5.3). Female patients with OSA were more likely to be postmenopausal (OR=3.2; 95% CI 1.5–7). There was a past history of myocardial infarction (MI) in 5.3% of OSA and 2.7% of non-OSA patients, but this difference was not significant (OR for OSA in those with history of MI=2; 95% CI 0.5–4). Both groups had similar proportions of subjects on antihypertensive medication (16.5 vs 17.1%). Asthma, chronic obstructive pulmonary disease (COPD), angina, cerebrovascular accidents and treated hypothyroidism occurred to the same extent in both groups. Furthermore, there were no differences in family history of obesity, snoring, hypertension, ischaemic heart disease or hypothyroidism.

Physical examination

Examination of the upper airway was not helpful in predicting the likelihood of OSA. Redundant pharyngeal tissue and/or a small oropharyngeal airway was detected in 30% of OSA patients and in 24% of non-OSA patients, a deviated nasal septum (15 vs 13%), enlarged tonsils (5 vs 9%) and glossomegaly (2.2 vs 0.9%). None of these differences was statistically significant on chi-squared testing.

There was a strong correlation between anthropometric measurements (BMI, % predicted body weight, neck and abdominal circumferences) and AHI (table 3). Among patients not on antihypertensive medication, the proportions with a systolic blood pressure (BP) ≥160 mmHg (14.9% with OSA vs 17.8% without OSA) were not significantly different between the two groups. However, the proportion with a diastolic BP ≥ 95 mmHg was higher (20.8 vs 9.1%) in patients with OSA (p=0.05; OR=2.6; 95% CI 1.1–2.5). There was no correlation between AHI and either systolic BP (r=0.08; p=0.25) or diastolic BP (r=0.08; p=0.26).

Oximetry

Simple overnight oximetry is often used as a screening test for OSA and, therefore, we looked at the oximetry data in isolation. Oximetry tracings performed in association with the Grass system were analysed manually before the polygraph data were scored. In the case of the Oxford system, the automated oximetry record was used. The end-points studied were absolute number of ≥4% dips in $\%Sa,O_2$ overnight and the lowest $\%Sa,O_2$ during the night. Both correlated significantly with AHI (r=0.69; (p<0.001) for number of dips and (r=-0.65; p<0.001) for the lowest $\%Sa,O_2$. The ORs and predictive values for these data are shown in table 4.

Stepwise multiple linear regression

The square root transformed AHI was used as the dependent variable. The total r^2 was 0.189 and the variables showing a significant relationship were alcohol consumption (r^2=0.074, p<0.001), BMI (r^2=0.06, p<0.001) and age (r^2=0.048, p<0.001). These factors remained significant when male patients alone were considered, although BMI was now slightly more significant than

Table 4. – Summary of oximetry data: the end-points studied were absolute number of ≥4% dips in Sa,o_2 overnight and the lowest %Sa,o_2 during the night

		OR (95% CI)	Predictive value % Positive	Negative
Total desaturations n	≥30	21 (10–45)	83	81
Nadir of O_2 saturation %	<90	3.6 (1.6–9.8)	69	86
	<85	6.3 (1.6–24.2)	78	67
	<80	10.7 (2.3–24.9)	89	55

OR: odds ratio; 95% CI: 95% confidence interval; Sa,o_2: arterial oxygen saturation.

alcohol consumption (r^2=0.073 *vs* r^2=0.063, p<0.001 for both). However, among female patients, age (r^2=0.122; p=0.026) and neck circumference (r^2=0.097; p=0.036) were the only significant factors.

Both neck and abdominal circumference measurements showed a significant correlation with AHI (table 3). Both measurements were entered, along with age and BMI, into a stepwise multiple linear regression with square root of AHI as the dependent variable. In male subjects where abdominal girth was measured (n=140), only this measurement was found to significantly correlate with AHI (r^2=0.125; p<0.001), and neck circumference was not a significant independent correlate. In the case of female patients (n=29), neck circumference (r^2=0.13; p=0.03) and age (r^2=0.1; p=0.047) were the significant factors.

Logistic regression

The likelihood of a patient having an AHI ≥15 events·h[-1], based on a number of independent clinical variables, was determined using the equation outlined in the statistics section, and the parameter estimates shown in table 5. The probability value for each patient (ranging 0–1) was then plotted against the observed AHI for that patient (fig. 1). From figure 1 it may be seen that patients with a probability of 0.26 or less (7.4% of total patients) all had an AHI <15 events·h[-1], with the majority having an AHI <8 events·h[-1], and they could be confidently designated as not having clinically significant OSA, without the need for polysomnography.

However, there was no corresponding probability level, above which all patients had an AHI ≥15 events·h[-1]. Thus, further studies would be required to identify patients with OSA. A second logistic regression equation was developed to establish the usefulness of next performing simple overnight oximetry (table 5b). It was found that all patients with a probability of 0.2 or less on oximetry had an AHI <15 events·h[-1] (3% of all patients), whilst all patients with a probability of 0.86 or greater (24% of all patients) had an AHI ≥15 events·h[-1]. Most patients with a probability ≥0.86 on oximetry, had probability scores of greater than 0.54 based on clinical factors (22% of all patients). Thus, an upper cut-off of 0.54 was taken,

Table 5. – Parameter estimates for logistic regression relationships, both for : a) clinical features; and b) oximetry data

Variable	Parameter estimates (x)
a) Clinical features	
Sex (male=1, female=0)	-0.541
Age yrs	0.046**
Snores every night (yes=1, no=0)	0.282
Observed apnoeas (yes=1, no=0)	0.69*
Dozes whilst driving (yes=1, no=0)	0.624
Alcohol consumption (units·week[-1])	0.038*
BMI (≥30=1, <30=0)	0.707*
Constant (c)	-2.689**
b) Oximetry data	
Number of dips ≥4% (<30=0, 30–59=1, 60–89=2, ≥90=3)	1.108***
Lowest %Sa,o_2 (≥90=0, 85–89=1, 80–84=2, <80=3)	0.806***
Constant (c)	-2.044***

*: p,0.05; **: p<0.01; ***; p<0.001. For abbreviations see legend to table 1. p-values refer to the level of significance of each variable in the logistic regression equation.

with all patients above this (48% of all patients) next having oximetry. Performing oximetry on patients with a probability of 0.27–0.53 (44% of patients) would have significantly increased the number of indeterminate oximetry studies (probability of 0.2–0.85) without identifying many more patients with probability factors of <0.2 or ≥0.86. Thus, these patients should go straight onto more detailed sleep studies, which in this report involved PSG.

Thus, using clinical features and oximetry data alone, nearly one third of patients (7.4+22+3=32.4%) of patients could be confidently categorized as OSA or non-OSA without the need for full polysomnography. This result correlates with a number of other studies that have shown
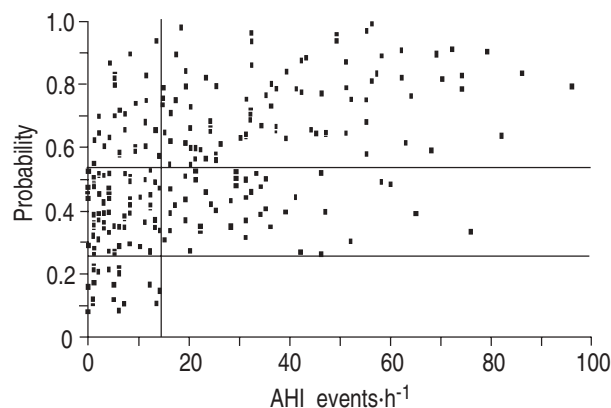

Fig. 1. – Relationship between probability values for obstructive sleep apnoea (OSA), calculated using the logistic regression model, and the apnoea/hypopnoea index (AHI), for each patient. The vertical line represents the reference criterion for OSA (*i.e.* AHI ≥15 events·h[-1] to the right and <15 events·h[-1] to the left). Points above the upper horizontal line represent patients who should have overnight oximetry. Points between the two horizontal lines denote those patients who should go straight to polysomnography. Points below the lower horizontal line indicate patients who could be designated non-OSA at the initial visit (see Results).

Table 6. – Comparison of logistic regression analysis at different threshold values for the AHI

| | | AHI criterion events·h^{-1} | | |
		$\geq$10	$\geq$15	$\geq$20
With OSA %		64	54	46
Clinical features	Upper limit	$\geq$0.57	$\geq$0.54	$\geq$0.54
	Lower limit	<0.28	<0.26	<0.26
Oximetry	Upper limit	$\geq$0.90	$\geq$0.86	=1.00
	Lower limit	<0.19	<0.20	<0.20
Clinical features alone	Non-OSA %	1.4	7.4	1.4
Clinical features and oximetry	OSA %	31.3	22	20.2
	Non-OSA %	6.1	3.3	6.7
	Indeterminate %	36.2	22.8	41.1
Direct to PSG %		25	44.4	30
For full PSG %		61.2	67.2	71.1

AHI: apnoea-hypopnoea index; OSA: obstructive sleep apnoea; %: percentage of all patients; PSG: polysomnography.

that use of predictive factors can reduce the number of patients undergoing PSG by about one third [6, 7, 11]. In addition, when a cut-off AHI of 10 events·h^{-1} was used there was a greater proportion correctly categorized without PSG (39%), whilst the proportion was less when a cut-off AHI of 20 events·h^{-1} was used (29%) (table 6).

Discussion

The present study shows that individual symptoms and signs of OSA are of little diagnostic help among patients referred to a respiratory sleep clinic, as none are close to 100% predictive of having or not having OSA. However, when a number of these factors are combined in a mathematical model, the predictive accuracy is increased, and when augmented by overnight oximetry data, they can be used to accurately detect the presence or absence of clinically significant OSA, without the need for PSG, in about one third of cases.

Previous studies have reported that neck circumference is a better predictor of obstructive event frequency than BMI among patients suspected of sleep apnoea [9, 18–20]. However, in the present report, BMI was a significant independent correlate with AHI on multiple linear regression analysis, while neck circumference was not. Furthermore, in male patients allowing for age, abdominal girth was a closer correlate with AHI than neck circumference and BMI. In contrast, neck circumference was the best correlate of AHI in female patients, suggesting a possible sex difference in fat distribution and its effect on AHI.

Self-reported snoring has been shown to have a strong association with sleep apnoea in most reports [5, 7, 8, 10, 13, 14]. However, in the present report, snoring was only significant if it occurred every night. A tendency to doze whilst driving proved to be the only circumstance in which EDS was more common among OSA than non-OSA patients, a finding supported by the recent study of KUMP et al. [12], although such an association was not observed by FLEMONS et al. [13]. In studies where EDS was not specified in a range of circumstances, no difference between the two groups has been found [6, 7,

14], with one exception [10]. This lack of difference is explained by the fact that habitual snorers without OSA are also more likely to have EDS than subjects with little or no sleep-disordered breathing [8], and by the fact that self-reported sleepiness is not an objective measure, and may underestimate the physiological state of sleepiness [21].

The tendency for alcohol to worsen both snoring and OSA is well-recognized [22, 23], with a significant relationship between dips in $\%S_{a,O_2}$ and alcohol consumption [1]. In the present study, the amount of alcohol consumed per week was a significant independent correlate of AHI in the group as a whole, but this relationship only remained significant in male patients when sex was taken into account.

Most patients slept in the lateral position. However, a minority of patients stated that they slept predominantly on their back (12.7%) and were found to be at increased risk of having OSA (table 2). Sleeping supine predisposes to obstruction of the upper airway by dorsal movement of the tongue and soft palate. It was also observed that awakening from sleep with symptoms of heartburn was a risk factor for OSA (table 2). Patients with OSA tended to be more obese with thicker waists, which would predispose to gastric acid reflux. In addition, intra-abdominal pressure is increased, and intrathoracic pressure is decreased, during inspiratory efforts against an obstructed upper airway, further increasing the tendency to gastro-oesophageal reflux.

Information was not obtained from bed partners, since the questionnaire was administered on the evening of the sleep study, and it was not usually feasible to have the bed partner present because of time, travel and social difficulties. In addition, the initial referral was usually precipitated by the bed partner, who was perturbed by the loud snoring and observed apnoeas. The patients themselves were usually made aware, by their bed partner, of the severity of these problems by the time they presented for a sleep study. Furthermore, there is only marginal improvement in the prediction of apnoea frequency from a room-mate administered questionnaire over that from a self-administered questionnaire [12].

The lack of difference in frequency of anatomical abnormalities between the two groups might not have occurred

if more specific methods of assessment were used, such as pharyngoscopy by an ENT specialist, or upper airway imaging. However, it was the aim of the present study to only employ methods of assessment that could be easily used in a respiratory outpatient clinic. The present report does, however, highlight the observation that there is no easily detectable anatomical abnormality on clinical examination in more than half of the patients with OSA.

Previous studies examining the value of performing overnight oximetry alone, have found it of limited value in the investigation of patients suspected of having OSA [24, 25]. We have found that by using specific measurements, namely total number of dips $\geq 4\%$ and nadir of $\%Sa,O_2$, a subgroup of patients can be correctly designated as OSA or non-OSA. It must be emphasized that all patients had slept for a minimum of 3 h and, thus, oximetry data were sufficiently representative. However, in circumstances where there is some doubt that the patient had slept, it may not be correct to conclude that such patients do not have OSA and these patients may still need full PSG.

It could be argued that oximetry could also be used to screen patients in the intermediate group of $0.26 \leq p < 0.54$. Most of the patients who had conclusive evidence of OSA on their oximetry ($p \geq 0.86$) had probability scores above 0.54 based on their clinical features. This effectively identified 89% of patients with $p > 0.86$ on oximetry. In order to detect the remaining 11% (amounting to 2% of all patients), oximetry would need to be performed in the other 44%, with the majority of these still requiring PSG. Performing oximetry in all patients not excluded at the first visit would involve 93 overnight oximetry assessments and 65 PSGs (*i.e.* 158 studies) per 100 patients, whilst following the guidelines in the present study would involve 48 overnight oximetry assessments and 67 PSGs (*i.e.* 115 studies) per 100 patients. Thus, 45 extra oximetry assessments would be required to save two PSGs.

The confounding effect of possible "prescreening" by the referring physician [7] was minimized in the present report as the majority of patients were referred by their family practitioner, who would have had no particular knowledge of sleep-disordered breathing and would have automatically referred the patient for a specialist opinion. Only 21% of patients were referred by a practitioner expected to have any specific knowledge of sleep-disordered breathing. All patients who were referred underwent a full sleep study, and thus there was no prescreening of patients at the initial visit, *i.e.* no patient with one of the cardinal features of snoring, EDS or observed apnoeas was excluded. Thus, all patients were essentially primary referrals without any prior attempt to treat them for OSA. It could be argued that the patients "preselected" themselves by seeking medical attention. However, the present report was intended as a study of patients referred to a respiratory sleep centre and is not a screening study of the population as a whole.

The limitations of defining OSA on the basis of an AHI criterion alone must be recognized, since it is the usual clinical practice to view PSG data in combination with the patient's symptoms and other clinical factors. In the present report, the AHI was treated as a continuous variable where possible, *i.e.* single and multiple factor regression analyses. However, other statistical tests required AHI to be expressed as a categorical variable, namely the chi-squared and logistic regression analyses. We chose an AHI of 15 events·h^{-1} as the threshold of clinical significance, since this is an internationally accepted reference level [6, 16, 17], and it is also a useful limit in order to detect patients who need treatment with nasal continuous positive airway pressure (nCPAP). Thus, we effectively compared patients with moderate to severe OSA with patients with mild or no OSA. Nevertheless, the principal conclusions are unchanged when either an AHI of 10 or 20 events·h^{-1} is used as the minimum criterion for OSA.

Patients with an AHI of <15 events·h^{-1} could still have troublesome symptoms, as in the upper airway resistance (UAR) syndrome [26]. However, all such patients with a probability of equal to or greater than 0.26 (table 5b) would eventually have full PSG. Patients with disrupted sleep architecture and frequent arousals, but with an AHI <15 events·h^{-1}, could still be detected and treated appropriately (nCPAP, ENT surgery for socially disruptive snoring, *etc.*).

The present report represents one of the largest European studies of patients referred to a respiratory sleep centre, and in which all patients underwent full sleep studies. RAUSCHER *et al.* [11] also performed a study of similar scale, but in a more select study population of patients referred for investigation of snoring. Snoring did not enter their model and most subjects were self-referrals. Also patients with an AHI of 10–20 events·h^{-1} were excluded from their model, in order to have a clear dichotomy between the two groups.

In conclusion, individual clinical features are not helpful in predicting obstructive sleep apnoea among patients presenting to a respiratory sleep clinic. However, in the present report, the combination of clinical features was able to correctly define 7.4% as not having obstructive sleep apnoea, and when combined with overnight oximetry data, a further 25.3% could be correctly identified as either having or not having OSA. The number of patients requiring full polysomnography could possibly be reduced further if oximetry was combined with other less sophisticated means of assessment, such as video recording or a static charge sensitive mattress. Finally, it remains for us to evaluate the prediction rules, derived above, in a prospective fashion on a newly constituted sample.

References

1. Stradling JR, Crosby JH. Predictors and prevalence of obstructive sleep apnoea and snoring in 1,001 middle-aged men. *Thorax* 1991; 46: 85–90.
2. Young T, Palta M, Dempsey J, Skatrud J, Weber S, Badr S. The occurrence of sleep-disordered breathing among middle-aged adults. *N Engl J Med* 1993; 328: 1230–1235.

3. He J, Kryger M, Zorick F. Mortality and apnea index in obstructive sleep apnea. *Chest* 1988; 94: 9–14.

4. Partinen M, Guilleminault C. Daytime sleepiness and vascular morbidity at seven-year follow-up in obstructive sleep apnea patients. *Chest* 1990; 97: 27–32.

5. Kapuniai LE, Andrew DJ, Crowell DH, Pearce JW. Identifying sleep apnea from self-reports. *Sleep* 1988; 11: 430–436.

6. Crocker BD, Olson LG, Saunders NA, *et al*. Estimation of the probability of disturbed breathing during sleep before a sleep study. *Am Rev Respir Dis* 1990; 142: 14–18.

7. Viner S, Szalai JP, Hoffstein V. Are history and physical examination a good screening test for sleep apnea? *Ann Intern Med* 1991; 115: 356–359.

8. Bliwise DL, Nekich JC, Dement WC. Relative validity of self-reported snoring as a symptom of sleep apnea in a sleep clinic population. *Chest* 1991; 99: 600–608.

9. Davies RJ, Ali NJ, Stradling JR. Neck circumference and other clinical features in the diagnosis of the obstructive sleep apnoea syndrome. *Thorax* 1992; 47: 101–105.

10. Hoffstein V, Szalai JP. Predictive value of clinical features in diagnosing obstructive sleep apnea. *Sleep* 1993; 16: 118–122.

11. Rauscher H, Popp W, Zwick H. Model for investigating snorers with suspected sleep apnoea. *Thorax* 1993; 48: 275–279.

12. Kump K, Whalen C, Tishler PV, *et al*. Assessment of the validity and utility of a sleep-symptom questionnaire. *Am J Respir Crit Care Med* 1994; 150: 735–741.

13. Flemons WW, Whitelaw WW, Brant R, Remmers JE. Liklihood ratios for a sleep apnea clinical prediction rule. *Am J Respir Crit Care Med* 1994; 150: 1279–1285.

14. Dealberto M-J, Ferber C, Garma L, Lemoine P, Alpérovitch A. Factors related to sleep apnea syndrome in sleep clinic patients. *Chest* 1994; 105: 1753–1758.

15. Rechtschaffen A, Kales A. A manual of standardized terminology, techniques and scoring system for sleep stages in human subjects. Washington, DC, National Institutes of Health, 1968; Publication No. 204.

16. Gould GA, Whyte KF, Rhind GB, *et al*. The sleep hypopnea syndrome. *Am Rev Respir Dis* 1988; 137: 895–898.

17. Douglas NJ, Thomas S, Jan MA. Clinical value of polysomnography. *Lancet* 1992; 339: 347–350.

18. Hoffstein V, Mateika S. Differences in abdominal and neck circumferences in patients with and without obstructive sleep apnoea. *Eur Respir J* 1992; 5: 377–381.

19. Katz I, Stradling J, Slutsky AS, Zamel N, Hoffstein V. Do patients with obstructive sleep apnea have thick necks? *Am Rev Respir Dis* 1990; 141: 1228–1231.

20. Davies RJ, Stradling JR. The relationship between neck circumference, radiographic pharyngeal anatomy and the obstructive sleep apnoea syndrome. *Eur Respir J* 1990; 3: 509–514.

21. Thorpy MJ. The clinical use of the Multiple Sleep Latency Test: the Standards of Practice Committee of the American Sleep Disorders Association. *Sleep* 1992; 15: 268–276. (Erratum, *Sleep* 1992; 15: 381).

22. Scrima L, Broudy M, Nay KN, Cohn MA. Increased severity of obstructive sleep apnea after bedtime alcohol ingestion. *Sleep* 1982; 5: 318–328.

23. Issa FG, Sullivan CE. Alcohol, snoring and sleep apnea. *J Neurol Neurosurg Psychiatry* 1982; 5: 353–359.

24. Cooper BG, Veale D, Griffiths CJ, Gibson GJ. Value of nocturnal oxygen saturation as a screening test for sleep apnoea. *Thorax* 1991; 46: 586–588.

25. Williams AJ, Yu G, Santiago S, Stein M. Screening for sleep apnea using pulse oximetry and a clinical score. *Chest* 1991; 100: 631–635.

26. Guilleminault C, Stoohs R, Clerk A, Cetel M, Maistros P. A cause of excessive daytime sleepiness: the upper airway resistance syndrome. *Chest* 1993; 104: 781–787.