

Original article

Personality, anxiety and mood traits in patients with sleep-related breathing disorders: effect of reduced daytime alertness

Emilia Sforza^{a,*}, Zara de Saint Hilaire^a, Antoine Pelissolo^b, Thierry Rochat^c, Vincent Ibanez^a

^a*Sleep Laboratory, Division of Neurophysiology, Department of Psychiatry, Geneva University Hospital, Hôpital Belle Idée, 2 Chemin du Petit Bel Air, Ch 1225 Chêne Bourg, Geneva, Switzerland*

^b*Department of Psychiatry, Hospital Fernand-Widal, Paris, France*

^c*Division of Pneumology, Geneva University Hospital, Geneva, Switzerland*

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Abstract

Objective: The etiology of depression and personality disorders in patients with sleep-disordered breathing (SDB) is not well defined and it is still unclear if they are directly related to the severity of the disease. In this study we test the hypothesis as to whether daytime sleepiness largely contributes to appearance of mood disorders.

Methods: Sixty patients diagnosed as having snoring ($n = 16$) or OSA ($n = 44$) were examined. Daytime sleepiness was assessed by the administration of the Epworth Sleepiness Scale (ESS) and by the Maintenance Wakefulness Test (MWT). The Hospital Anxiety (HAD-A) and Depression (HAD-D) Scale and the Temperament and Character Inventory (TCI) questionnaires were used for psychopathological evaluation.

Results: The mean HAD-A score was 6.9 ± 0.45 and the average HAD-D score was 4.6 ± 0.48 , with no significant difference between snorers and OSA patients. Anxiety was present in 16% of cases and depression in 7%. The HAD-D score was related to the ESS score ($R = 0.37$, $P = 0.003$), the mean sleep latency at the MWT ($R = -0.34$, $P = 0.04$), and the mean low SaO₂, ESS score alone explaining the 17% of variance in the HAD-D score. Compared to controls, there were no differences in almost all TCI scores, with novelty-seeking temperament score higher in patients. No relationships were found between HAD or TCI scores and apnea density.

Conclusions: We conclude that among patients evaluated for SDB, higher depression scores show an association with reduced daytime alertness, which therefore may have important effects on mood. © 2002 Elsevier Science B.V. All rights reserved.

Keywords: Mood disorders; Anxiety; Personality; Daytime sleepiness; Obstructive sleep apnea

1. Introduction

The Obstructive Sleep Apnea syndrome (OSA) results from the repeated occlusion of the upper airway during sleep, inducing nocturnal hypoxemia and sleep fragmentation. Among diurnal symptoms fatigue, reduced alertness and impaired quality of life have been described, which appear to be a consequence of the combined effects of sleep disruption and apnea recurrence. Over the past few years, the burgeoning interest in psychopathological changes in patients with sleep-disordered breathing (SDB) has resulted in a large increase in the number of published studies on this topic. From the initial studies, mood and personality disorders were described as significantly higher in OSA patients than in the general population, depression occurring in 40% [1] to 56% [2] of the OSA population.

Moreover, the depression score was greater when the frequency of sleep-disordered breathing was higher and the nocturnal hypoxemia more severe [3]. A similar association has also been described with hypochondriasis, conversion-hysteria, psychotic symptoms [4,5], and delirium [6].

In contrast to the above investigations, the results of more recent studies failed to identify apnea density or nocturnal hypoxemia as potential risk factors for depression or personality changes. In a study of 60 OSA patients [7], the author found that not one of these OSA patients fulfilled the criteria of the DSM-III-R for diagnosis of depression. In a study examining 112 patients with OSA [8], depression and mood disturbances were correlated to age and body mass index, severity of sleep apnea and sleep parameters not contributing to highest depression score. Similar results were obtained in an extensive study on 2271 patients examined for SDB [9]. In this group neither the existence of OSA nor the apnea-hypopnea index (AHI) was associated with

* Corresponding author. Tel.: +41-22-3055329; fax: +41-22-3055343.
E-mail address: sforza-emilia@diogenes.hcuge.ch (E. Sforza).

depression and anxiety, women with simple snoring having higher scores.

When we consider the incidence of mood disorders in patients with OSA, the important question is whether the incidence of these psychopathological changes is related to the disease itself or whether it is the result of other variables such as sleepiness [10] and impaired general health status [11] related to sleep fragmentation and apnea. We know that some of the symptoms most commonly reported by OSA patients such as fatigue, loss of interest, decreased libido, poor concentration, and cognitive impairment overlap with those described in patients with depression, and they are dependent on daytime sleepiness. Moreover, the reduced quality of life [11] present in SDB patients may affect their general health perception and their functional and emotional well-being inducing, in turn, personality changes such as aggressiveness, irritability, anxiety or depression, all expressing adaptation of the patients to their worsening life condition [12]. Unfortunately, until now the role of daytime sleepiness on OSA psychopathology has not been examined, and it remains unknown if some of the so-called psychopathological traits described in OSA patients are related to misinterpretation of sleepiness and personality changes occurring when reduced alertness, reduced vigor and impaired health status become manifest.

The present study used the Hospital Anxiety and Depression Scale (HAD) [13] and the Temperament and Character Inventory (TCI) [14] questionnaire to quantify the incidence of mood disorders and personality traits in a population of patients referred to a sleep clinic for investigation of snoring and possible OSA. The primary aim was to test the hypothesis as to whether diurnal sleepiness, more than the severity of the disease, greatly contributed to the association between SDB, personality changes and mood disorders.

2. Material and methods

2.1. Patients

Sleep data were selected from patients referred to our sleep laboratory over the period December 1999 to July 2000 for probable SDB. A structured interview was conducted prior to the polysomnography, and data relevant to this investigation were medication (with attention to psychoactive drugs), substance abuse, alcohol intake, history of neurological disorders, head injuries, and psychiatric diseases. Questions about psychiatric diseases were constructed to assess previous or current depression, psychosis or anxiety based on the Structural Clinical Interview for DSM-III [15]. Criteria for inclusion in the study were: (1) a clinical history of heavy snoring, associated with or without reported apneas by a witness; (2) no other sleep disorders including insomnia, periodic leg movements or hypersomnia; and (3) absence of current organic mental disorder or psychiatric disease including depression,

psychosis or agitation. Of the original sample of 75 patients, 60 patients met the selection criteria.

The patients were informed that some of the collected data would be used for research purposes and they gave written informed consent.

2.2. Nocturnal sleep studies

All patients underwent polysomnography including three electroencephalograms (C3-A2, C4-A1, PZ-O2), right and left electro-oculograms and electromyograms of chin muscles and tibialis anterior muscles. Nasal and oral flow were recorded with a thermistor, and thoracic and abdominal respiratory movements with a strain gauge. Arterial oxygen saturation (SaO_2) was continuously measured with a finger oximeter. During the night, lights-out time was set at 22:00–23:00 h and lights-on time at 07:00 h.

Sleep was scored using the criteria of Rechtschaffen and Kales [16] for epochs of 30 s. Calculation of sleep parameters included total sleep time (TST), wake time after sleep onset (WASO), sleep efficiency (SE: total sleep time/total recording time \times 100), and percentage of stages 1, 2, 3, 4 and rapid-eye-movement sleep (REM) with reference to sleep period time. Stages 3 and 4 were collectively regarded as slow wave sleep (SWS). Sleep latency was calculated as the time elapsed from the start of the recording to three epochs of stage 1, or one epoch of stage 2. REM latency was calculated from the first epoch of stage 2 to the first epoch of stage REM. As indices of sleep fragmentation, we defined the number of awakenings as well as the number of sleep state transitions. An awakening was defined as a shift in alpha frequency lasting 1 min or more.

Respiratory events were scored using standard criteria. Hypopneas were defined as 50% or greater reduction in airflow lasting at least 10 s and associated with an arousal from sleep, or 4% oxyhemoglobin desaturation. Apneas were defined as a total cessation of airflow lasting at least 10 s. Apnea was further defined as obstructive when airflow cessation was associated with persistent respiratory effort. The apnea + hypopnea index (AHI) was defined as the number of apneas + hypopneas per hour of sleep. As indices of nocturnal hypoxemia we considered the mean low SaO_2 (mean of the low SaO_2 recorded after each respiratory event), the minimal value recorded during sleep (minimal SaO_2) and the mean value during sleep (mean SaO_2).

Subjects were classified as OSA patients if they had an AHI more than 10 or snorers if they had an AHI less than 10 during the nocturnal polysomnography.

2.3. Daytime sleepiness: subjective and objective evaluation

The patients rated their subjective daytime sleepiness by administration of the Epworth Sleepiness Scale (ESS) [17,18], an eight-item questionnaire that asked the patients to answer each question from 0 (not at all likely to fall asleep) to 3 (very likely to fall asleep), yielding a score of 0 (minimum) to 24 (maximum). The ESS, measuring the

subjective sleep propensity, was completed by patients on the day of polysomnography as a part of standard clinical protocol.

In 37 patients objective daytime alertness was assessed with the Maintenance of Wakefulness Test (MWT) according to standard criteria [19] performed on the day following the nocturnal recording. The test was performed by asking the patients to sit in a quiet, dark room and to try to stay awake for five sessions scheduled at 09:00, 11:00, 13:00, 15:00 and 17:00 h. All tests were terminated 15 min after sleep onset or after 40 min without sleep. Sleep latency was defined by the appearance of one 30 s epoch of stage 1 according to standard criteria [20].

2.4. Anxiety, depression and personality questionnaires

Anxiety, depression and personality traits were measured by means of the Hospital Anxiety and Depression Scale (HAD) [13] and the Temperament and Character Inventory (TCI) [14] questionnaires, selected from those most commonly used which met validity, reliability and sensitivity to psychopathological conditions.

The HAD is a self-report rating scale consisting of two subscales each containing seven items on a 4-point Likert scale (range 0–3). Its anxiety and depression score is strongly correlated with the Beck Depression Inventory and the Spielberger State Trait Anxiety Inventory [21].

The TCI used in this study is a French version [22,23] of the original self-administered, true-false questionnaire developed by Cloninger [14]. This questionnaire, containing 226 items, assesses four primary dimensions of the temperament including Novelty-seeking (NS), i.e. the tendency to

respond actively to novel stimuli; Harm Avoidance (HA), reflecting passive behavior and fatigue; Reward Dependence (RD), reflecting sentimentality and dependence; and Persistence (P), defining perseverance despite frustration and fatigue. Three dimensions of character are also assessed, the Self-directedness (SD), the Cooperativeness (C) and the Self-transcendence (ST). The TCI scores of the patient group were compared with a control population including 96 healthy adults (80 men and 16 women) aged 27–72 years (mean age 50.0 ± 1.22 yr.) randomly selected and age- and sex-matched from an epidemiological sample of 602 individuals [22,23]. All normal controls were drug-free and had no personal histories of psychiatric diseases, snoring and/ or other sleep disorders.

2.5. Statistical analyses

Results in the text and in Tables 1–3 are presented as means \pm SEM.

Controls, snorers and OSA patients were compared using the Mann–Whitney *U*-test with Bonferroni correction for multiple comparison, and significance was taken as a *P* value <0.001 .

Bivariate correlation analysis using Pearson's correlation coefficient was used to find the variables correlated with HAD and TCI scores; multivariate regression analysis was done to define the contribution of anthropometric, diurnal and nocturnal variables in explaining HAD and TCI scores. All statistical analyses were performed with the SPSS statistical software package (SPSS 9.0 for Windows, SPSS Inc, Chicago).

Table 1

Anthropometric, clinical and polysomnographic data of the patient group (means \pm (SEM)). The differences between snorers and OSA patients are reported^a

	Total group (<i>n</i> = 60)	Snorers (<i>n</i> = 16)	OSA (<i>n</i> = 44)	<i>P</i> ^b
Age years	50.6 (1.6)	43.2 (2.8)	53.3 (1.7)	0.001
BMI kg/m ²	30.4 (0.7)	30.3 (1.2)	30.5 (0.9)	Ns
AHI n/h	34.4 (3.5)	5.4 (0.8)	45.0 (3.7)	0.001
Minimal SaO ₂ %	74.4 (1.3)	83.5 (1.3)	71.2 (1.5)	0.001
Mean low SaO ₂ %	86.3 (0.8)	90.2 (0.8)	84.9 (0.9)	0.001
Mean SaO ₂ %	92.2 (0.4)	94.1 (0.4)	91.4 (0.4)	0.001
St. 1 %	21.9 (1.5)	17.6 (1.7)	23.5 (1.9)	Ns
St. 2 %	51.6 (1.2)	49.7 (1.4)	52.3 (1.6)	Ns
St. 3–4 %	8.4 (0.9)	11.5 (2.1)	7.3 (1.0)	Ns
St. REM %	17.8 (0.8)	21.2 (1.4)	16.5 (0.9)	Ns
TTS min	444.6 (9.9)	463.0 (19.2)	437.9 (11.5)	Ns
REM latency min	97.2 (6.6)	92.4 (9.7)	99.0 (8.4)	Ns
WASO min	125.1 (9.9)	106.9 (17.3)	131.7 (11.9)	Ns
SE %	78.3 (1.7)	81.3 (3.2)	77.2 (2.0)	Ns
Awakenings <i>n</i>	19.1 (2.0)	15.7 (1.6)	20.3 (2.6)	Ns
Sleep stage transition <i>n</i>	268.1 (13.2)	220.8 (15.9)	285.3 (16.4)	Ns
ESS	10.9 (0.5)	11.8 (1.0)	10.7 (0.6)	Ns
MWT min	19.7 (2.0)	17.1 (3.5)	20.5 (2.3)	Ns

^a AI, apnea index; AHI, apnea plus hypopnea index; BMI, body mass index; TTS, total sleep time; WASO, wake after sleep onset; SE, sleep efficiency; MWT, mean sleep latency at the Maintenance Wakefulness Test; ESS, Epworth Sleepiness Scale.

^b Mann–Whitney *U*-test.

Table 2

Pearson's correlation coefficients of relation between HAD scores for anxiety (HAD-A) and depression (HAD-D), and diurnal and nocturnal variables

	HAD-A		HAD-D	
	R	P	R	P
Age years	0.093	ns	-0.100	ns
BMI kg/m ²	0.093	ns	-0.100	ns
ESS	0.174	ns	0.371	0.003
MWT min	-0.195	ns	-0.337	0.04
AHI n/h	-0.084	ns	-0.253	0.06
Mean low SaO ₂ %	0.046	ns	0.293	0.03
SE %	0.010	ns	0.127	ns
Stage 1 %	0.031	ns	-0.044	ns
Stage 2 %	-0.047	ns	-0.108	ns
Stages 3-4 %	0.068	ns	0.139	0.04
Stage REM %	-0.046	ns	0.039	ns
REM latency min	-0.092	ns	0.175	ns

3. Results

3.1. Patient characteristics

Clinical, anthropometric and nocturnal respiratory findings of the patient group are listed in Table 1. The patients, aged 50.6 ± 1.6 years with a mean body mass index (BMI) of 30.4 ± 0.7 kg/m², had a mean AHI of 34.4 ± 3.5 (range:0.7–92). Of the original sample of 60, 44 had OSA while 16 were snorers. A wide range in severity of nocturnal hypoxemia was present, with a minimal SaO₂ of 74.4 ± 1.3 % (range 48–91%), and a mean low SaO₂ during apneas of 92.2 ± 0.4 %. Total sleep time, sleep efficiency and percentage of sleep spent in different sleep stages indicate disturbed sleep with a high number of awakenings and sleep state transitions, a low sleep efficiency (SE), as well as a high WASO (mean = 125.1 ± 9.9 min). The sleep architecture showed a large amount of light sleep and a low percentage of slow wave and REM sleep. Although a trend exists for OSA to have lower SE and a higher number of awakenings and sleep stage transitions, there was no significant difference between patient subgroups.

Table 3

Mean scores for each TCI temperament and character dimension in patients and controls (means \pm SEM)

	Patients N = 60	Controls N = 96	P ^a
Novelty seeking	19.2 (0.75)	14.2 (0.57)	0.001
Harm avoidance	14.8 (0.99)	15.8 (0.66)	NS
Reward dependence	15.2 (0.52)	13.5 (0.43)	NS
Persistence	4.8 (0.23)	4.5 (0.19)	NS
Self directedness	32.3 (0.82)	32.8 (0.59)	NS
Cooperativeness	34.0 (0.70)	30.7 (0.70)	NS
Self Transcendence	14.5 (0.70)	12.9 (0.58)	NS

^a Mann-Whitney U-test.

3.2. Daytime sleepiness evaluation

During the day, the mean ESS score was 10.9, with pathological subjective sleepiness (ESS > 11) present in 29 patients. The mean sleep latency at the MWT was 19.7 ± 2.0 min. with a sleep latency shorter than 10 min in 11 patients. No difference between snorers and OSA patients was seen when subjective sleep propensity and daytime alertness were considered (Table 1).

3.3. HAD and TCI questionnaires

The mean score of the HAD questionnaire was within normal range both for the anxiety subscale (HAD-A) (mean = 6.9 ± 0.45 , range 0–14) and for the depression subscale (HAD-D) (mean = 4.58 ± 0.48 , range 0–14). Applying the criteria [21] for detecting anxiety and depression, i.e., 8 for probable cases and 11 for clear cases, the prevalence of these psychopathological changes was low. The incidence of probable cases was 18% for anxiety and 8% for depression, whereas the prevalence of clear cases was respectively 16% and 7%. No significant differences between snorers and OSA patients were seen on both HAD-A (6.89 ± 0.92 vs 7.0 ± 0.51) and HAD-D (5.2 ± 0.74 vs 4.4 ± 0.59) scales. A trend to higher scores was seen in the female group (HAD-A: 8.75 ± 0.96 , HAD-D: 5.92 ± 1.1) compared to males (HAD-A: 6.48 ± 0.49 , HAD-D: 4.25 ± 0.52), but the differences did not reach statistical significance. Table 2 reports the correlation coefficients of the diurnal and nocturnal parameters, and HAD-A and HAD-D scores. The HAD-D score was related to the ESS score ($R = 0.37$, $P = 0.003$), to the mean sleep latency at the MWT ($R = -0.34$, $P = 0.04$) (Fig. 1) and to the mean low SaO₂. There was a near significant negative relation with the AHI ($R = -0.25$, $P = 0.06$), all other potential nocturnal predictors not included. Stepwise regression analysis showed that the ESS score alone explained the 17% of variance in the depression score.

Table 3 shows the means and SEM for the four temperament and the three character dimensions of the TCI in the whole sample of patients and in the control group. The average values of 60 patients were within normal ranges for almost all the temperament and dimension scores. No significant differences were found for the HA, RD, and P dimension of temperament and for SS and ST characters between patients and controls. Cooperativeness was higher in the patient group but the difference did not reach statistical significance. Comparison between groups showed that the only significant difference was present for all subscales of novelty-seeking temperament, showing higher values in the patient group. In order to assess whether the presence of OSA predisposes to personality changes, the 44 patients with OSA were compared to 16 patients with simple snoring. As shown in Fig. 2, no differences were seen between snorers and apneic patients for the four temperament and the three character dimension scores. Correlation of TCI scores

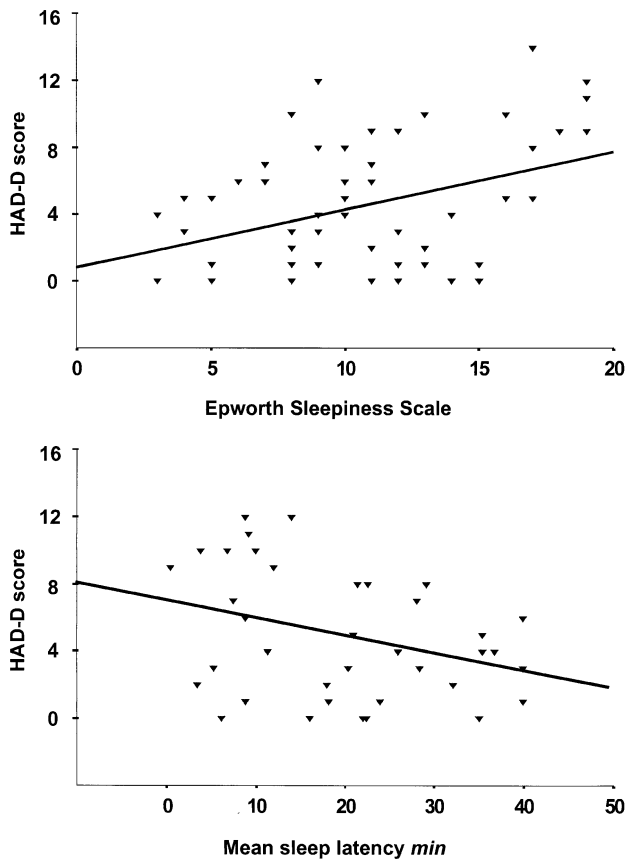


Fig. 1. Scatterplots showing relationship between the HAD-D score, the Epworth Sleepiness Scale and the mean sleep latency at the Maintenance Wakefulness Test.

with the diurnal and nocturnal variables indicated for NS a near significant relation with the ESS score ($R = 0.25$, $P = 0.054$). No relationship was found in our study sample between dimension and temperament scorers and nocturnal variables (sleep parameters, AHI and oxygen desaturation) in either the OSA or the snorer group.

4. Discussion

The purpose of this study was to investigate the relation between daytime alertness and anxiety, depression and personality changes in patients with SDB. The most interesting finding of the study was that patients with a greater degree of subjective sleep propensity and daytime sleepiness were found to be at increased risk for depression, with a higher HAD-D score present in patients having a higher ESS score and reduced sleep latency at the MWT. Secondly, the incidence of anxiety and depression was significantly lower compared to previous reports, affecting 7–16% of the whole group and the search for personality traits was almost entirely fruitless. These findings support the hypothesis that some of the psychopathological changes described in patients with OSA are likely to reflect the reduced alertness and the reduced vigor and quality of life related to breathing disorders.

Several previous investigations examining the covariation between depression score of patients with SDB and nocturnal variables (AI, AHI and nocturnal hypoxemia) found little [24,25] or no evidence of a linear relation [8,9], suggesting that the presence of OSA and its severity

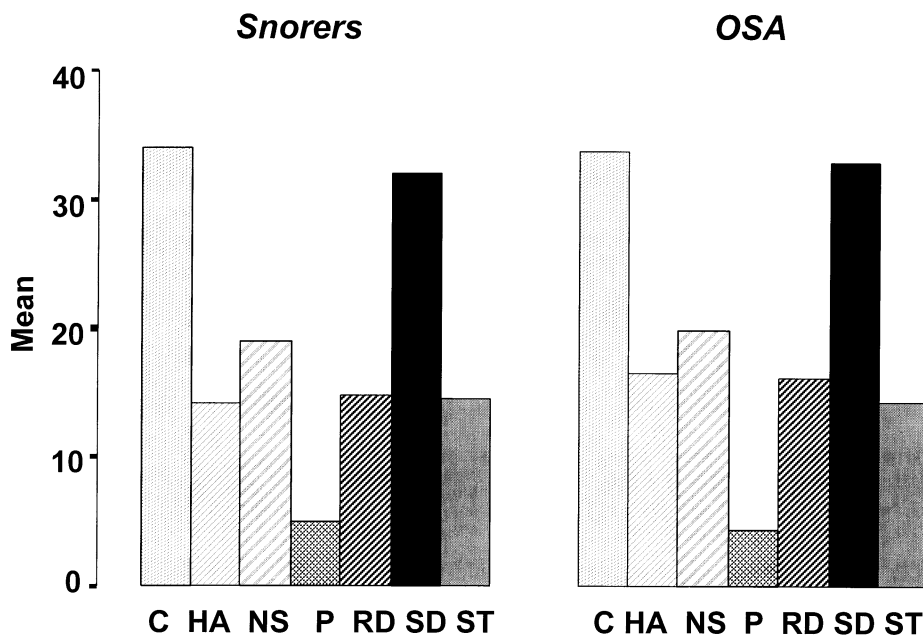


Fig. 2. Histograms of the four temperament (Novelty-seeking (NS), Harm Avoidance (HA), Reward Dependence (RD), Persistence (P)), and three dimension (Self directedness (SD), Cooperativeness (C), Self transcendence (ST)) of the TCI questionnaire in OSA patients and snorers. The Fig. depicts the absence of difference between snorers and OSA patients.

is not the primary cause of psychopathological changes in SDB patients. In a statistical analysis of variables associated with depression, we found no association between AHI and depression score, with the correlation coefficient of mean low SaO₂ quite small. A much stronger relationship was found between HAD-D score and sleepiness, using both subjective (ESS) and objective (MWT) measures of sleepiness. Assuming that diurnal impairment is crucial to psychopathological changes, some hypotheses can be advanced to explain how diurnal sleepiness influences the psychological profile in patients with SDB. We know that patients with SDB may present to the physician with complaints of poor sleep quality, fatigue, loss of interest, lack of energy and reduced ability in tasks requiring concentration, attention and dexterity [24]. Moreover, their health status score is lower when assessed by the Functional Outcomes of sleep questionnaires [25], the Calgary Quality of Life Index [26] or with the Short-form 36 Health Survey [27]. Such difficulties may be so severe that job performances and family life may be affected, leading in turn to emotional disturbances and personality changes. Interestingly, Chervin [28] recently demonstrated that OSA patients reported, as their primary complaint, more frequently fatigue, tiredness and lack of energy than sleepiness, 36–44% of these patients describing the lack of energy as the worst problem affecting the ability to accomplish what they wanted. Thus we can expect that as a consequence of sleep fragmentation and sleep disruption related to SDB, reduced alertness, lower executive functioning and altered perception of functional and emotional well-being would be present, with resultant mood disorders.

The analysis of the seven personality dimensions of the TCI questionnaire reveals no significant differences between patients and controls for almost all personality and character traits, novelty-seeking being the only dimension showing a significantly higher score in patients compared to controls. Might the significantly higher NS temperament be explained by the presence of sleep-disordered breathing? This seems unlikely, however, in that there is no significant correlation between the NS score and AHI, nocturnal hypoxemia and measures of diurnal sleepiness. Since the temperament dimension is supposed to be co-inherited and stable throughout life [14,22,23], and novelty-seeking temperament score is higher in people with dependence on smoking [15] or alcohol [29], we suggest that the higher NS temperament in patients with SDB may simply reflect a genetic personality trait favoring obesity, tobacco and alcohol intake, all factors increasing co-morbidity for OSA [10].

We cannot exclude the possibility that some limitations of our study design have introduced methodological bias. A first consideration is the use of questionnaires, all of which may introduce subjective bias in the estimation of mood disorders and sleepiness. It is possible that using subjective estimation we have classified as 'sleepy' patients who would have been classified as non-sleepy when other ques-

tionnaires are employed. However, this seems unlikely since an association between HAD-D score and sleepiness is still present when objective measure of alertness, i.e. MWT, is used. A second consideration is that in our sample depression, as estimated by the HAD-D score, was less common than previously reported. Mood disorders affect 7% of our sample, which is consistent with the community lifetime depression prevalence rates (8–18%) [30]. These results diverge significantly with previous studies showing an incidence of depression ranging from 20 to 46% [1,2,4,31,32] of the study samples. This discrepancy may derive from several factors affecting selection of populations and methodology. First, in the earliest reports [1,2,31], patients had a very high mean AHI, a score far superior to that in our group, perhaps creating a selection bias toward severely affected subjects. Moreover, we applied criteria intended to select a broad cross-section of patients with SDB, excluding those whose underlying depression or psychosis could confound interpretation. Finally, in our sample no significant differences were seen between snorers and OSA patients, when indices of sleepiness are considered. Although our sample may not fully represent the general population of patients with snoring in whom some cases of upper airway resistance syndrome may have been included, an hypothesis for the higher ESS score is that in these patients the negative intrathoracic pressure swings developed during snoring play a dominant part in sleep restoration and sleepiness [33].

In conclusion, our results support the hypothesis that some of the so-called personality and mood disorders described in patients with SDB may be essentially related to misinterpretation of daytime sleepiness. The reduced alertness process, and the lower quality of life, vigor and energy may substantially account for the mood disorders described in patients with SDB. Whether this hypothesis has an impact in clinical practice will require further investigation in a larger group of patients, considering the effect of efficacious therapies.

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