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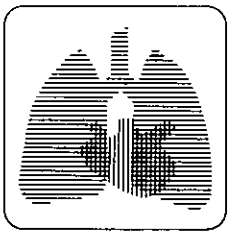

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The Epworth Sleepiness Scale (ESS) is a simple questionnaire measuring the general level of daytime sleepiness, called here the average sleep propensity. This is a measure of the probability of falling asleep in a variety of situations. The conceptual basis of the ESS involves a four-process model of sleep and wakefulness. The sleep propensity at any particular time is a function of the ratio of the total sleep drive to the total wake drive with which it competes. ESS scores significantly distinguished patients with primary snoring from those with obstructive sleep apnea syndrome (OSAS), and ESS scores increased with the severity of OSAS. Multiple regression analysis showed that ESS scores were more closely related to the frequency of apneas than to the degree of hypoxemia in OSAS. ESS scores give a useful measure of average sleep propensity, comparable to the results of all-day tests such as the multiple sleep latency test. *(Chest 1993; 103:30-36)*

ASP = average sleep propensity; BMI = body mass index (kg/m^2); ESS = Epworth Sleepiness Scale; MSLT = multiple sleep latency test; MWT = maintenance of wakefulness test; OSAS = obstructive sleep apnea syndrome; RDI = respiratory disturbance index (the number of apneas and hypopneas ≥10s long causing ≥3 percent arterial oxygen desaturation per hour of sleep); REM = rapid eye movement sleep; SaO_2 = arterial oxygen saturation percentage; SL = sleep latency; SP = sleep propensity (the probability of falling asleep in a given situation at a particular time); SSS = Stanford Sleepiness Scale; VAS = visual analogue scale

Increased daytime sleepiness is an important symptom of obstructive sleep apnea syndrome (OSAS).[1] Currently the most widely used method for measuring sleepiness is the multiple sleep latency test (MSLT)[2] or some variant of it such as the maintenance of wakefulness test (MWT).[3] However, these tests are expensive and time-consuming (they take all day) so they are not always done, even when there is clearly a need for quantifying patients' sleepiness. There is also confusion about what sleepiness is and what the tests measure.

This report is about a simple new method that I have proposed for measuring daytime sleepiness—the Epworth Sleepiness Scale or ESS.[4,5] First we need to consider the conceptual basis for sleepiness and its measurement. This will involve clarification of some existing concepts and the introduction of some new ones. Only then can the rationale of the ESS be explained. This report then goes on to show how sleepiness measured by ESS is related to the severity of OSAS and to snoring, drawing comparisons with the results of other investigations that have measured sleepiness differently.

Sleepiness Reconsidered

The word sleepiness did not appear in the index of medical text books in the past. It is a concept that is still not widely used or clearly understood. There is confusion about what different people consider sleepiness to be and this has led some to use a circular argument to define sleepiness as that which is measured by one or other tests of sleepiness.

Some consider sleepiness to be a state involving feelings of tiredness or fatigue and the subjective changes that immediately precede sleep onset, as are measured by the Stanford Sleepiness Scale (SSS).[6] This has been called "subjective" sleepiness. So too has the sleepiness measured by a visual analogue scale (VAS) of sleepiness-alertness in which sleepiness is seen as a subjective state of low alertness.[7] As others have pointed out, fatigue and a low level of alertness may signal the need for sleep but they do not provide a useful measure of the likelihood of falling asleep.[8] Many insomniacs are very tired at night but cannot fall asleep readily whereas narcoleptics often fall asleep during the day without feeling tired. The "subjective" sleepiness measured by the SSS is usually not related to a subject's propensity to fall asleep, even when measured at the same time by the MSLT.[9,10] In addition, the SSS is not a unitary scale according to the results of factor analysis of its items.[11] A VAS of alertness-sleepiness is simpler and because it is a unitary scale, it appears preferable to the SSS for measuring "subjective" sleepiness.

Other people define sleepiness as "a physiological drive usually resulting from sleep deprivation"[12] or as "a physiological need-state that leads to an increased tendency to fall asleep."[13] It is said that "the presence and intensity of this state can be inferred by how readily sleep onset occurs, how easily sleep is disrupted, or how long sleep endures."[13] It is this conceptual framework that led to the development of the MSLT which, it is claimed, "establishes a setting to

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maximize the likelihood of sleep onset" while "all factors competing with falling asleep are removed from the test situation."[13] That it is possible to establish such a setting in which there is no alerting influence competing with a sleep drive is a premise rather than the subject of experimental evidence.

The MSLT measures objectively the mean sleep latency (SL) in four to six half-hour naps taken 2 h apart during the day in a comfortable, darkened room when the subject is encouraged to sleep.[2] By contrast, the MWT measures SL when the subject is asked to stay awake while sitting for 40 min at a time in a dimly lit room.[3] Another variant, the repeated test of sustained wakefulness, is similar but the subject lies down for 40 min trying to stay awake.[14] Because such tests involve objective measurements of SL, the sleepiness they measure has been called "objective" sleepiness. However, different tests that are said to measure "objective" sleepiness, such as the MSLT and MWT taken at the same time in the same subjects, give results that are not closely related. Their correlation is statistically significant (*eg*, $r=0.407$, $n=258$, $p<0.001$) but accounts for only about 20 percent of the variance in SLS.[15] This has led to the conclusion that the MSLT measures the ability to fall asleep whereas the MWT measures the ability to stay awake.[16]

One reason for the confusion in such matters is, I believe, that the generally accepted conceptual basis for sleep and wakefulness, exemplified by models such as the two-process model of Borbély and associates[17,18] is too limited. It largely ignores the idea that sleep processes (sleep drives) must continuously interact with competing wake-processes (wake drives). Knowledge of one or other of these competing drives alone is not sufficient to determine the state of sleep or wakefulness. It is the ratio of these two main variables, not their absolute magnitude, that determines the likelihood of falling asleep. In my view, sleep propensity or SP (which some people call "objective" sleepiness) is neither a state nor a physiologic drive but the probability of falling asleep at a particular time. More precisely, it is the probability of a state change from wakefulness to non-REM sleep.

Thus, SP = f (total sleep drive)/(total wake drive). This is not the same as the probability of remaining awake which is 1 − (SP), a function of SP nevertheless.

A Four-Process Model of Sleep and Wakefulness

A model of sleep and wakefulness must involve at least four processes for it to explain sleepiness. The details and ramifications of such a theory are beyond the scope of this report and will be described elsewhere. However, the essence of this model is that two sleep processes contribute to the total sleep drive and two wake processes contribute to the total wake drive. What I call the primary sleep drive is due to an intrinsic CNS process that varies with a circadian rhythm having a peak at about 10 pm to 12 midnight and a trough at about 6 to 8 am, as manifested in the circadian rhythm of delta-wave sleep.[19,20] A secondary sleep drive increases progressively during wakefulness and is modified by behavior. It is discharged progressively after the onset of non-REM sleep. Its influence is seen predominantly during the first sleep cycle when the influence of the primary sleep drive is also high.

What I call the primary wake drive is due to intrinsic activity in the reticular activating system that varies with a circadian rhythm having a peak at about 6 to 7 pm and a trough at 4 to 5 am. This is reflected in the circadian rhythm of body temperature, melatonin secretion, and REM sleep.[21,22] By contrast, a secondary wake drive is influenced very much by posture, behavior, physical activity, feelings, and mental activity generally. It is derived from the nonspecific thalamic projection system via collateral inputs from all sensory nerve tracts (exteroceptive and enteroceptive) and from the limbic system that is influenced by all CNS centers concerned with affect, volition, attention, and cognition, *etc.*[23]

In addition to its circadian rhythm, the primary wake drive has an ultradian rhythm with a cycle length of about 90 to 110 min, manifested in REM sleep cycles.[23] Similarly, the primary sleep drive has an ultradian rhythm manifested in delta-wave sleep occurrence, with a slightly longer cycle length (120 min) than the ultradian rhythm in the primary wake drive.[4] Both the primary sleep and primary wake drives can also have ultradian rhythms with longer cycle lengths of 4 to 12 h.[24] All four sleep-wake processes can vary independently but have mutual and mainly inhibitory interactions. These interactions and the relative phase relationships between their circadian and ultradian rhythms determine the usual times and duration of sleep and wakefulness, and the occurrence of REM/non-REM sleep cycles. The nature of the interactions among these four processes may be different in wakefulness, non-REM, and REM sleep, determined by specific mechanisms involved in each type of transition between states. However, some influence from all four processes continues throughout sleep and wakefulness. We are concerned here only with the transition from wakefulness to non-REM sleep. This is different, for example, from the transition from wakefulness to REM sleep, which is usually blocked, but which can occur in narcolepsy.

For most people, the SP at a particular time depends at least as much on their total wake drive as on their total sleep drive. Subjects whose primary sleep drive is very high can fall asleep at any time simply by

voluntarily reducing their secondary wake drive by lying down and relaxing with eyes closed. To ask the same subject to try and stay awake rather than fall asleep reduces his SP by increasing the secondary wake drive without changing his sleep drive. For those whose primary sleep drive is not very high, being asked to fall asleep at a particular time, when under constant surveillance to see whether they do or not, may involve them trying harder than usual to fall asleep. This too will increase their secondary wake drive and lower their SP. Thus, the SP for a particular subject depends not only on when but under what circumstances it is measured. While some aspects of different situations can be standardized (*eg*, posture), others are highly subjective, depending on their meaning for each subject, and cannot be fully controlled or even measured. Thus, SP is not independent of the circumstances in which it is measured.

Regardless of individual responses, some situations or activities are more likely than others to promote a change in the secondary wake drive with a consequent change in SP. This characteristic can be called the soporific nature of the activity or situation. Lying down is more soporific than sitting, but walking is much less soporific than either. The MSLT, MWT, and related tests each measure the SP under different circumstances, none of which is entirely representative of a patient's daily activities. The SP measured in one such test may well be correlated significantly with the SP in another, but they will not necessarily be closely related, as others have found.[15,16]

A subject's SP at a particular time will be influenced by the many factors that affect the four sleep and wake drives. These include the time of day, previous sleep deprivation, the effects of drugs on the CNS, the subject's age, the presence of sleep disorders such as OSAS, and the patient's physical, cognitive, and affective state at the time. Although the SP will inevitably vary during the day and from day to day, different subjects will have a mean SP over prolonged periods (at least a week) about which fluctuations occur. I call this long-term characteristic the average sleep propensity (ASP). It is this ASP that a chronic sleep disorder such as OSAS affects by increasing long term the total sleep drive but not the wake drive.

Rationale of the ESS

The ESS is a simple questionnaire that asks the subject to rate on a scale of 0 to 3 the chances that, as part of his "usual way of life in recent times," he would doze in each of eight different situations.[4] The situations were chosen on *a priori* grounds to vary in their soporific nature from highly soporific ("lying down to rest in the afternoon when circumstances permit") to much less soporific ("sitting and talking to someone").[5] The ESS score is the sum of eight item scores and can range from 0 to 24. Initial results suggest that the clinically "normal" range of ESS scores is 2 to 10 with a normal distribution statistically and a model score of 6. However, this "normal" range may have to be modified as more data become available.

ESS scores are significantly but not highly correlated with SL measured in MSLTs ($r = -0.514$, $n = 27$, $p < 0.01$).[4] Item and factor analyses have shown that the ESS is a unitary scale with high internal consistency (Cronbach's alpha = 0.88).[5] The ESS has a high test-retest reliability over a period of 5 months in normal subjects ($r = 0.822$, $n = 87$, $p < 0.001$).[5] The high initial scores of patients with OSAS return to "normal" after nasal CPAP treatment for at least 3 months.[5] The ESS is conceptually unique in its ability to measure ASP over its whole range from very low levels in some insomniacs to very high levels in patients with severe OSAS and narcolepsy.[4] However, it was anticipated earlier that ESS scores and ASP may also differ between subjects because of one or more psychophysiologic traits that influence their sleep and wake drives in the absence of recognized sleep disorders.

Each ESS item gives an estimate of SP in one of eight specific situations whereas the total ESS score gives a measure of the more general parameter, ASP, relating to a range of situations that are commonly met in daily life. Thus, the ESS is the subjective equivalent of eight different objective tests such as the MSLT, each one of which can measure SP in only one situation. What the ESS lacks by way of objective accuracy with each item score as a measure of a situation-specific SP it makes up for by increasing the number and range of situations and by making them more relevant to daily life.

The ESS does not measure "subjective" sleepiness as described above and is not an alternative to the SSS or to a VAS of alertness-sleepiness. As with any questionnaire-based method, the ESS is limited by the subject's ability to read and comprehend the questionnaire and to answer the questions honestly. This disadvantage must be weighed against its considerable advantages in terms of cost and ease of administration when compared with all-day laboratory tests.

Present Investigation

The aim of the present investigation was to provide further evidence about the validity of the ESS against the conceptual background outlined above. The clinical use of the ESS is illustrated by showing how ESS scores differ between patients who have either OSAS or primary snoring and how their ASP is related to various parameters of OSAS when assessed by multiple regression analysis. Comparisons with other published results show that the severity of OSAS is related at least as closely to ESS scores as it is to SLs measured

objectively by MSLTs or MWTs.

METHODS

All subjects were fee-paying patients, investigated in the Sleep Disorders Unit at Epworth Hospital, a private hospital in Melbourne, Australia. They answered the ESS without assistance at the end of their first interview. They had one night's diagnostic polysomnography with sleep staging by EEG (electrode positions C4-A1), left and right EOG, submental EMG, measurements of nasal and oral airflow, thoracic and abdominal respiratory movements, arterial oxygen saturation measured by a pulse oximeter with the probe on the fifth finger, ECG, left and right leg movements monitored by a ceramic strain gauge over each tibialis anterior muscle, snoring noise analysis, and continuous infra-red video and sound-track recording.[25] Patients were not permitted any alcohol on the day of polysomnography.

After investigation, there were 108 consecutive patients who were diagnosed as having primary snoring and 165 with OSAS. All these patients had been reported to snore at home, on most if not all nights, and most for whom reports were available were said to "stop breathing" during their sleep. On this basis, all were suspected of having OSAS. The respiratory disturbance index (RDI) was calculated for each patient as the number of apneas and hypopneas per hour of sleep associated with at least 3 percent arterial oxygen desaturation. There were 108 patients who had an RDI <5 and who consequently were diagnosed as having primary snoring rather than OSAS. Those with an RDI between 5 and 24.9 were diagnosed as having "mild" OSAS, between 25 and 49.9 as "moderate" OSAS, and 50+ as "severe" OSAS. These categories were chosen fairly arbitrarily, each representing about one third of the range of RDIs (5 to 70). Different categories of OSAS had been used in an earlier investigation with the ESS.[4] Snoring, apneas, or hypopneas that ended in arousal were not distinguished from those without arousal, nor were patients distinguished on the basis of cardiac arrythmias or ischemia associated with their OSAS.

Patients were excluded from this study if they had another sleep disorder such as restless legs syndrome, periodic limb movement disorder, or narcolepsy in addition to primary snoring or OSAS. Thirty-two of the patients with primary snoring and 55 of those with OSAS had been the subjects of an earlier investigation with the ESS.[4] There were 91 men and 17 women among the 108 primary snorers, and 156 men and 9 women among the 165 patients with OSAS. Ages ranged from 18 to 77 years. The body mass index (BMI) was calculated from the weight and height of each patient (kg/m^2). The lowest arterial oxygen saturation (SaO_2 percent) recorded overnight was noted as the "minimum SaO_2" for each patient.

Statistical Methods

Overall differences between the primary snorers and the three categories of OSAS were tested by repeated one-way ANOVA. Posthoc Scheffé tests were used to test differences between paired groups.[26] The Scheffé test is conservative and is suitable for groups of unequal size. A matrix of Pearson correlation coefficients was calculated for five variables in the 165 ungrouped OSAS patients. Multiple regression analysis was used to relate ESS scores to age, BMI, RDI, and minimum SaO_2. Statistical significances was accepted at $p<0.05$ in two-tailed tests.

RESULTS

Analysis of Variance

The results for primary snorers and the three categories of OSAS are summarized in Table 1. There were many more patients with primary snoring or "mild" OSAS than there were with "moderate" or "severe" OSAS. Between these groups there were significant overall differences in the patients' ages, BMI, RDI, minimum SaO_2, and ESS scores. However, posthoc Scheffé tests did not reveal significant differences in age between any paired groups. The BMI was significantly lower for primary snorers than for each grade of OSAS ($p<0.001$). These grades did not differ significantly in BMI between themselves ($p>0.22$) but the percentage of obese patients, with BMI >30, tended to increase from 35.2 percent in "mild" OSAS, to 41.5 percent in "moderate," and 63.2 percent in "severe" OSAS.

As expected, RDIs differed significantly between each of the three grades of OSAS ($p<0.001$). These categories had been chosen on *a priori* grounds to be mutually exclusive. The minimum SaO_2 also differed significantly in posthoc tests among each of these three categories ($p<0.001$).

The primary snorers had significantly lower ESS scores than patients in each of the three categories of OSAS ($p<0.001$). ESS scores increased significantly with each step from "mild" to "moderate" and "severe" OSAS ($p<0.005$). There were scores in the range 11 to 15 in all four groups and a considerable overlap among them. All patients with "severe" OSAS had scores of 10 or more.

Correlation and Regression Analysis

The matrix of Pearson correlation coefficients for five variables in the 165 ungrouped patients with

Table 1—*The Number of Patients in Each Group and the Mean ± SD for Age, Body Mass Index (BMI), Respiratory Disturbance Index (RDI), Minimum Arterial Oxygen Saturation Recorded During Apneas Overnight (Min^m SaO_2%), and Scores on the Epworth Sleepiness Scale (ESS)*

	Primary Snoring	OSA			ANOVA (p)
		Mild	Moderate	Severe	
No. of Ss	108	105	41	19	—
Age, yr	47.2±11.0	50.4±8.8	51.4±11.1	46.1±10.4	<.05
BMI	26.9±3.8	29.5±4.4	30.8±5.9	31.8±5.5	<.001
RDI	—	12.1±5.4	34.8±9.4	56.6±5.9	<.001
Min^m SaO_2%	—	80.7±7.8	71.1±10.2	60.1±11.4	<.001
ESS score	8.0±3.5	11.0±4.2	13.0±4.7	16.2±3.3	<.001
ESS range	0-15	2-22	5-22	10-23	—

Table 2—*Pearson Correlation Coefficients Between Age, Body Mass Index (BMI), Respiratory Disturbance Index (RDI), Minimum Arterial Oxygen Saturation (SaO_2) Recorded During Apneas Overnight, and Score on the Epworth Sleepiness Scale (ESS) for 165 Patients With OSAS*

	Age, yr	BMI	RDI	SaO_2	ESS
Age, yr	1.000				
BMI	−0.183*	1.000			
RDI	−0.051	0.201†	1.000		
SaO_2	0.119	−0.284‡	−0.693‡	1.000	
ESS	0.026	0.193*	0.439‡	−0.404‡	1.000

*p<0.05.
†p<0.01.
‡p<0.001.

OSAS is shown in Table 2. Seven correlations were statistically significant, the highest being between RDI and minimum SaO_2 ($r = -0.693$, $p<0.001$). ESS scores were correlated independently and significantly with RDI, minimum SaO_2, and BMI. The highest of these correlations was with RDI ($r = 0.439$). Multiple regression analysis was used to define more clearly the relationship of these interrelated variables to ESS scores and hence to ASP. The multiple regression analysis initially included age, BMI, RDI, and minimum SaO_2 as independent variables and ESS score as the dependent variable. The multiple correlation coefficient ($R = 0.474$) was statistically significant ($F = 11.58$; $df = 4,160$; $p<0.001$). The only significant beta-weight was for RDI (beta = 0.299, $p<0.002$), but that for minimum SaO_2 approached significance (beta = −0.180, $p = 0.07$). Neither age nor BMI contributed significantly to this multiple regression.

The multiple regression analysis was repeated in a forward stepwise fashion, beginning with RDI as the only independent variable and ESS score as the dependent variable. Other independent variables were added one by one but were retained only if the proportion of variance in ESS scores explained by the regression was increased significantly as a result. With this method ESS scores were related to RDI as before (beta = 0.305, $p = 0.002$) but minimum SaO_2 now made a significant additional contribution to the regression (beta = −0.193, $p<0.05$). The multiple correlation coefficient ($R = 0.460$) was again significant ($F = 21.77$, $df = 2,162$, $p<0.001$). The regression equation was (ESS score) = 16.11 + 0.082 (RDI) − 0.078 (minimum SaO_2). Thus, the ESS of these patients was related to the severity of their OSAS mainly in relation to the frequency of apneas and hypopneas causing arterial oxygen desaturation of at least 3 percent, and to a lesser extent to the severity of hypoxemia overnight. Of the total variance in ESS scores for the 165 patients, the RDI explained 19.2 percent and the minimum SaO_2 explained another 1.9 percent. This left 78.9% of the variance in ESS scores and presumably in ASP unexplained by those variables and not related to OSAS.

ESS scores differed widely among the snorers (0 to 15) and overall were higher than those reported previously for nonsnoring control subjects, the mean of which was 5.9±2.2 (SD) and the range of which was 2 to 10.[4] This difference was statistically significant ($F = 10.55$, $df = 136$, $p<0.01$). The distribution of snorers' scores was bimodal, which peaks at 5 and 9. There were 25.9 percent of snorers with ESS scores between 11 and 15, above the "normal" upper limit of 10, and at levels of ASP associated more typically with "mild" to "moderate" OSAS. For comparison, 53.3 percent of patients with "mild" OSAS had scores of 11 or more, as did 73.2 percent with "moderate" OSAS and 89.5 percent with "severe" OSAS. Thus, there was evidence that a minority of heavy snorers had increased levels of ASP whereas the majority were "normal" in this respect.

Discussion

The results provide further evidence that ESS scores give valid measurements of ASP or daytime sleepiness as defined herein. The scores increased linearly with the severity of OSAS and distinguished primary snorers from patients with OSAS, even of mild degree. In the patients with OSAS, it was the frequency of apneas and hypopneas that bore the closest relationship to their ASP as measured by ESS scores. The degree of hypoxemia, measured by the minimum SaO_2 overnight, was significantly correlated with the RDI but was much less important as an independent predictor of ASP. Similar conclusions have been reached by others who measured "objective" sleepiness by MSLTs[27] or by MWTs.[28]

It seems that OSAS increases the total sleep drive mainly by fragmenting sleep with repeated arousals that have the same effect as sleep deprivation. This would increase the ASP so long as the total wake drive did not also increase, and there is no evidence that it does. However, the same increase in sleep drive due to OSAS can be expected to have differing effects on the ASP depending on the coexisting levels of wake drive in different patients. It may be that hypoxemia during apneas does not much affect the sleep or wake drives until the SaO_2 reaches quite low levels (possibly <75 percent) so that its overall effect on ASP for most patients is small.

In an earlier investigation of patients with OSAS, the correlation between SL measured in MSLTs and their RDI (or closely related variables such as apnea index or respiratory arousal index) was −0.36.[27] In another investigation, the correlation between SL measured in MWTs and RDI was −0.35.[28] By contrast, others have failed to find a significant relationship between MSLT results and RDI or related

variables.[29] The correlation between ESS score and RDI in the present investigation was 0.439. This suggests that ASP measurements by means of the ESS are at least as useful clinically as the results of much more time-consuming and expensive laboratory tests such as the MSLT.

In the multiple regression analysis reported by Roehrs et al,[27] the best 3 of 11 predictor variables in patients with OSAS could account for 21 percent of the variance in SL measured in MSLTs. In the present investigation the same percentage of variance in ESS scores was accounted for by the only two significant predictors of the severity of OSAS (RDI and minimum SaO_2). All these results emphasize that the majority (79 percent) of variance in the measurements of ASP between subjects with OSAS is related to factors other than their OSAS. Some of this variance would be due to errors in measurement. However, there are substantial differences in ASP, even between normal subjects,[30] that may be due, at least in part, to long-term differences in psychophysiologic traits affecting either their sleep drives, their wake drives, or both. These may be partly learned and partly determined genetically, as are several other aspects of sleep habits.[31] These differences must be considered in conjunction with the many other factors, including chronic sleep disorders such as OSAS, that influence the ASP in particular subjects.

The levels of ASP in habitual snorers, without OSAS, have received little attention in the past. Snorers do not usually complain of excessive daytime sleepiness as do patients with OSAS. The MSLT was not designed to measure differences in sleepiness within the "normal" range and, before the ESS, there was no other standardized method available for this. Nevertheless, Guilleminault et al[32] have recently described a group of 15 heavy snorers who had slightly increased levels of daytime sleepiness (measured by MSLTs) as a result of brief arousals from sleep associated with partial upper airway obstruction that was not sufficient to be called obstructive apnea or hypopnea and that did not cause arterial oxygen desaturation. This increased SP and the arousals were removed by experimental nasal CPAP treatment, even though that was not clinically indicated and the patients had not complained of excessive daytime sleepiness.

The recordings in the present investigation were not scored for such brief arousals. Nevertheless, the results from the analysis of ESS scores are consistent with the idea of increased ASP in a minority of heavy snorers, but with the majority being "normal." This does not mean that snoring may not have other deleterious (*eg*, cardiovascular) effects apart from those due to its association with OSAS.[33]

The present results and the conceptual basis of the ESS described herein indicate that, although the questionnaire is based on retrospective reports of the subjects' usual behavior, the ESS is capable of measuring ASP, which others might call "objective" sleepiness.

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