Positional vs Nonpositional Obstructive Sleep Apnea Patients*

Anthropomorphic, Nocturnal Polysomnographic, and Multiple Sleep Latency Test Data

Arie Oksenberg, PhD; Donald S. Silverberg, MD; Elena Arons, PhD; and Henryk Radwan, MD

Study objectives: **To compare anthropomorphic, nocturnal polysomnographic (PSG), and multiple sleep latency test (MSLT) data between positional (PP) and nonpositional (NPP) obstructive sleep apnea (OSA) patients.**

Design: **This is a retrospective analysis of anthropomorphic, PSG, and MSLT data of a large group of OSA patients who underwent a complete PSG evaluation in our sleep disorders unit. The patients were divided in two groups: the PP group, those patients who had a supine respiratory disturbance index (RDI) that was at least two times higher than the lateral RDI, and the NPP group, those patients in whom the RDI in the supine position was less than twice that in the lateral position.**

Subjects: **From a group of 666 consecutive OSA patients whose conditions were diagnosed in our unit from September 1990 to February 1995, 574 patients met the following criteria and were included in the study: RDI>10; age>20 years, and body mass index (BMI)>20.**

Results: **Of all 574 patients, 55.9% were found to be positional. No differences in height were observed but weight and BMI were significantly higher in the NPP group, these patients being on the average 6.5 kg heavier than those in the PP group. The PP group was, on average, 2 years younger than the NPP group. Nocturnal sleep quality was better preserved in the PP group. In this group, sleep efficiency and the percentages of deep sleep (stages 3 and 4) were significantly higher while the percentages of light sleep (stages 1 and 2) were significantly lower than in the NPP group. No differences for rapid eye movement (REM) sleep were found. In addition, wakefulness after sleep onset and the number of short arousals (<15 s) were significantly lower in the PP group. Apnea index and total RDI were significantly higher and the minimal arterial oxygen saturation in REM and non-REM sleep was significantly lower in the NPP. No differences in periodic limb movements data were found between the two groups. The average MSLT was significantly shorter in the NPP group. Univariate and multivariate stepwise logistic regression analysis showed that the most dominant variable that correlates with positional dependency in OSA patients is RDI, followed by BMI which also adds a significant contribution to the prediction of positional dependency. Age, although significant, adds only a minor improvement to the prediction of this positional dependency phenomenon. A severe, obese, and older OSA patient is significantly less likely to be positional than a mild-moderate, thin, and young OSA patient. In four obese OSA patients who lost weight, a much more pronounced reduction was seen in the lateral RDI than in the supine RDI, and three of these cases who were previously NPP became PP.**

Conclusions: **In a large population of OSA patients, most were found to have at least twice as many apneas/hypopneas in the supine than in the lateral position. These so-called "positional patients" are on the average thinner and younger than "nonpositional patients." They had fewer and less severe breathing abnormalities than the NPP group. Consequently their nocturnal sleep quality was better preserved and, according to MSLT data, they were less sleepy during daytime hours. RDI was the most dominant factor that could predict the positional dependency followed by BMI and age. RDI showed a threshold effect, the prevalence of PP in those with severe RDI (RDI≥40) was significantly lower than in those OSA patients with mild-moderate RDI. BMI showed a major significant inverse relationship with positional dependency, while age had only a minor although significant inverse relationship with it. Body position during sleep has a profound effect on the frequency and severity of breathing abnormalities in OSA patients.**

(CHEST 1997; 112:629-39)

Key words: body posture; breathing disturbances; human sleep; obstructive sleep apnea; polysomnography; sleep disorders; sleep position

Abbreviations: AI=apnea index; ANOVA=analysis of variance; A-P=anteroposterior; BMI=body mass index; Min SaO_2-REM=minimum SaO_2 level during REM sleep; Min SaO_2-NREM=minimum SaO_2 level during non-REM sleep; MSLT=multiple sleep latency test; nCPAP=nasal continuous positive airway pressure; NREM sleep=non-rapid eye movement sleep; NPP=nonpositional patients; OSA=obstructive sleep apnea; PLM=periodic limb movements; PLM I=periodic limb movements index; PLM AI=periodic limb movements arousal index; PP=positional patients; PSG=polysomnographic; RDI=respiratory disturbance index; REM sleep=rapid eye movement sleep; SaO_2=arterial oxygen saturation; TST=total sleep time; UA=upper airway; UARS=upper airway resistance syndrome

In patients with obstructive sleep apnea (OSA), the level of respiratory distress during sleep, as judged by the apnea/hypopnea index or respiratory disturbance index (RDI), is on average about 40 to 50% lower when they lie on their side than when they lie on their back (*ie,* in the supine position).[1-9] Cartwright[2] and Lloyd and Cartwright[4] defined "positional patients" (PP) as those OSA patients in whom the RDI was at least twice as high in the supine position as in the lateral position. In fact, the degree of severity of OSA in these patients is mostly related to the sleep time spent or not spent in the supine position. Those patients in whom the RDI in the supine position was less than twice that in the lateral position were called "nonpositional patients" (NPP).

The percentage of PP in OSA patients varies in different reports[2,4,10-14] from 9%[11] to 60%.[4] This variation is probably due to the small numbers and the different types of OSA patients studied. Some patients have succeeded in lowering their total RDI to normal by merely sleeping on their sides[2,10,14-19] and it has been estimated that this type of therapy alone could be successful in treating about 50% of all OSA cases.[4,20] Since OSA is present in about 9% of men and 4% of women in the middle-age population (by using a RDI≥15),[21] the implications of such an approach are obvious. Little information exists about the relationship of positional dependency to the physical characteristics of the OSA patients, the quality of nocturnal sleep, and the level of daytime somnolence.

The aim of this report is to compare anthropomorphic, nocturnal polysomnographic (PSG), and multiple sleep latency test (MSLT) data between the PP and NPP group in 574 consecutive OSA patients whose conditions were diagnosed in our Sleep Disorders Unit.

Materials and Methods

All the patients were referred to the Sleep Disorders Unit at the Loewenstein Hospital-Rehabilitation Center because of snoring complaints and/or a suspicion of OSA from September 1990 to February 1995. During this period, 666 consecutive patients were diagnosed as having OSA (RDI>10). Of these, 574 patients who were older than 20 years, had a body mass index (BMI)>20, RDI>10, and slept more than 30 min in either the supine or the lateral position were included in the analysis. These patients were divided into PP and NPP according to the criterion of Cartwright.[2] For that purpose, in addition to the overall RDI, supine and lateral RDI values were calculated for each patient. The supine and lateral RDI data define to which group (PP or NPP) each patient belongs.

The patients arrived at the sleep unit around 8 PM and the PSG recordings usually began between 10 PM and midnight.

The PSG recordings were carried out using polygraphs (Nihon Kohden models 4321 and 4414; Tokyo, Japan) and included the following parameters: electro-oculogram (two to four channels); EEG (four to six channels); electromyogram of submental muscles (one to two channels); ECG (one channel); electromyogram of the anterior tibialis muscle of both legs (two channels); and airflow (with a nasal/oral thermistor; Nihon Kohden). chest and abdominal effort (two channels) was recorded using inductive plethysmography (Respitrace; Ambulatory Monitoring Inc; Ardsley, NY; or Resp-Ez breathing belts; Tel Aviv, Israel); arterial oxygen saturation (SaO_2) levels (one channel) by pulse oximetry (Ohmeda 37000e; Boulder, Colo) with a finger probe, and audio (one channel) by a microphone located above the patient's head at a distance of 1 m and connected to a sound level meter (Quest Electronics model 2700; Oconomowoc, Wis), were recorded. The output from the sound level meter was also recorded in parallel on a calibrated (40 to 80 dB) chart recorder at a paper speed of 10 cm/h.

The recordings were carried out at a paper speed of 10 mm/s and sleep stages were scored according to the standard criteria of Rechtschaffen and Kales.[22]

The PSG technician who followed the patient's behavior through a closed-circuit 21-inch TV monitor marked the changes in body position in two places simultaneously, on the polygraph and on the chart recorder which registered the output of the pulse oximeter data.

The PSG technician was responsible for the monitoring of one or two sleeping patients. The two TV monitors were placed side by side to allow easy visualization of all patients' body movements. Since our unit is especially interested in the effect of body position on sleep-related breathing disturbances, our PSG technicians are encouraged to pay special attention to this issue.

Apnea was defined as an episode of a complete breathing cessation of ≥10 s. Hypopneas were considered as such if a partial breathing cessation (>20% reduction in oral/nasal airflow compared with the level of the previous five breaths) occurred, accompanied by a drop of SaO_2 of at least 3%.

Apnea index (AI) and RDI were calculated as the number of apneas per sleep hour and the number of apneas+hypopneas per sleep hour, respectively.[23] Arousals were divided as shorter or longer than 15 s and were scored according to accepted definitions.[24]

Periodic limb movements (PLM), PLM index (PLM I), and PLM arousal index (PLM AI) were scored and calculated according to Coleman.[25] PLM events associated with breathing abnormalities were not taken for the analysis.

The MSLT was carried out on the basis of published guidelines[26] and included four naps at 9AM, 11AM, 1PM, and 3PM on the day after the nocturnal PSG evaluation. The MSLT was performed in all patients who complained about daytime sleepiness. BMI is weight (kg)/height $(m)^2$. In order to assess the effect of weight loss on positional dependency, four obese OSA patients (average BMI=33.6) who refused nasal continuous positive airway pressure (nCPAP) treatment had a PSG evaluation before and after they lost weight in a dietary weight reduction program.

Data Analysis

For the comparison of the different anthropomorphic, sleep, and breathing parameters between the PP and the NPP group, the data were analyzed using the two-sample Student *t* test and Bonferroni correction was used for multiple *t* tests. For the MSLT data, two-way analysis of variance (ANOVA) with repeated measurements was performed.

*From the Sleep Disorders Unit (Drs. Oksenberg, Arons, and Rodwan), Loewenstein Hospital Rehabilitation Center, Raanana, Israel, and the Department of Nephrology (Dr. Silverberg), Tel-Aviv Medical Center, Tel-Aviv, Israel.
Manuscript received May 31, 1996; revision accepted February 27, 1997.
Reprint requests: Arie Oksenberg, PhD, Sleep Disorders Unit, Loewenstein Hospital Rehabilitation Center, POB 3 Raanana, Israel

Table 1—*Anthropomorphic Data**

	PP (n=321)	NPP (n=253)	p Value
Age, yr	52.9 (10.4)	54.9 (10.1)	0.020
Weight, kg	85.7 (14.0)	92.2 (15.8)	0.003
Height, cm	170.4 (8.8)	170.1 (8.5)	0.701
BMI	29.4 (4.1)	31.9 (4.9)	0.001

*Values are mean (SD).

To estimate how RDI, BMI, and age were related to the body position dependency, the statistical analysis was performed in two steps; first, a univariate analysis (χ^2 test for categorical variables) was carried out, and subsequently a stepwise multivariate logistic regression analysis was performed to assess the simultaneous contribution of all three parameters on positional dependency. All statistical analyses were performed with a statistical software package (SPSS version 6.08; SPSS, Inc; Chicago).

Results

Of the 574 OSA patients, 321 (55.9%) were PP and the other 253 (44.1%) were NPP.

Anthropomorphic Data

The mean age difference between the two groups was 2 years and was significant (p=0.02), the PP group being younger than the NPP group (Table 1). The mean height was not significantly different in the two groups.

The BMI was significantly greater in the NPP group (p=0.001) and this was due to the significantly higher weight in this group (p=0.003); the average weight in this group was 6.5 kg more than in the PP group.

Nocturnal PSG Data

Table 2 shows data on the comparison of various nocturnal sleep parameters in the two groups of patients. After Bonferroni correction, the significance level became p=0.003 instead of p=0.05.

No significant differences between both groups were seen for total recording time (TRT) (p=0.007). Nevertheless, total sleep time (TST) and sleep efficiency (S EFF) were significantly higher in the PP group (p=0.001) for both parameters. Sleep latencies (both to stage 1 and to persistent sleep) were not significantly different between the groups. REM sleep latency from onset of persistent sleep (REM LAT) and REM latency without intervening wake time (REM LAT w/o AW) were not significantly different between the groups (p=0.004 and p=0.057, respectively). The number of REM periods and the average length of REM periods were also not different between the groups (p=0.005 and p=0.110, respectively). However, the percentages of all non-REM sleep stages (out of TST) differed significantly in the two groups. The PP group had significantly lower percentages of the lighter sleep stages (stage 1, p=0.001, and stage 2, p=0.001) and higher percentages of deeper sleep (stages 3 and 4, p=0.001). For REM sleep percentages, no statistical differences were obtained (p=0.03). Also, the duration of wakefulness after sleep onset (WASO) and the number of long arousals (>15 s) were not significantly different between the two groups. However, the number of short arousals (<15 s) was significantly greater in the NPP group (p=0.001).

Table 2—*Nocturnal PSG Data**

	PP (n=321)	NPP (n=253)	p Value
TRT, min	422.4 (56.8)	407.4 (76.9)	0.007
TST, min	353.6 (66.4)	327.6 (78.1)	0.001†
S EFF, %	83.4 (11.0)	80.1 (12.4)	0.001†
LAT STG 1, min	12.9 (18.7)	11.7 (15.5)	0.410
LAT Perst Sleep, min	16.0 (21.3)	16.1 (19.7)	0.940
REM LAT, min	89.6 (46.6)	104.1 (63.7)	0.004
REM LAT w/o AW, min	78.0 (35.3)	85.5 (47.8)	0.057
No. of REMs	3.6 (1.5)	3.3 (1.4)	0.005
REM length, min	25.7 (20.8)	23.1 (15.3)	0.110
% STG 1	5.4 (4.2)	7.5 (7.0)	0.001†
% STG 2	55.0 (9.9)	61.3 (13.3)	0.001†
% STG 3	5.2 (3.1)	4.2 (3.0)	0.001†
% STG 4	12.9 (8.3)	9.7 (8.6)	0.001†
% STG 3+4	18.3 (10.1)	14.0 (10.1)	0.001†
% REM	19.1 (7.4)	17.5 (8.5)	0.030
WASO, min	53.6 (35.0)	64.1 (45.7)	0.004
No. of arousals >15 s	33.3 (20.3)	38.1 (48.7)	0.160
No. of arousals <15 s	159.2 (92.2)	209.6 (139.5)	0.001†

*TRT=total recording time; S EFF=sleep efficiency; REM LAT=REM latency from onset of persistent sleep (the first 10 min in which at least 8 of them were sleep); REM LAT w/o AW=REM latency without intervening awake time; STG=stage; WASO=wakefulness after sleep onset. Values are mean (SD).
†Significant differences after Bonferroni correction.

Table 3 shows a comparison of breathing parameters in the two groups of patients. All these parameters (AI, RDI, Min SaO_2-REM, Min SaO_2-NREM) were significantly different (p=0.001) in the two groups. In the NPP group, all four parameters showed a greater degree of abnormality.

Figure 1 shows an example of the SaO_2 and heart rate recordings during sleep in a typical PP. The differences in the frequency of SaO_2 desaturations and bradycardia/tachycardia episodes while sleeping on the back compared to the absence of these events while sleeping on the right side is clearly evident.

Table 4 shows a comparison of PLM data in the two groups of patients. In the PP group, 115 patients (35.8%) had PLM as a secondary diagnosis compared to 79 (31.2%) of the NPP group. This difference was not statistically significant (p=0.25). No significant differences were found between the two groups, either in the total number of PLMs, the number of PLMs causing arousals, the PLM I, or in the PLM AI.

Table 3—*Breathing Abnormalities Data**

	PP (n=321)	NPP (n=253)	p Value
AI	13.7 (15.1)	26.5 (29.4)	0.001
RDI	27.8 (17.7)	44.0 (29.7)	0.001
Min SaO_2 REM	81.1 (11.0)	72.7 (15.8)	0.001
Min SaO_2 NREM	84.7 (6.2)	81.5 (9.7)	0.001

*Values are mean (SD).

MSLT Data

In the PP group, 194 patients (60.4%) had an MSLT compared to 175 patients (69.2%) of the NPP group (Table 5). This difference was statistically significant (p=0.03). Two-way ANOVA analysis showed that the MSLT data difference between the two groups was of borderline significance (p=0.054). Nevertheless, the average sleep latency for all four naps was significantly shorter in the NPP group than in the PP group (p=0.01). In addition, it should be noted that for each of the four naps, the sleep latency was consistently longer in the PP than in the NPP group.

Effect of RDI on Positional Dependency

In order to estimate the influence of OSA severity, as expressed by RDI, on the positional dependency, the entire group of OSA patients was first divided into four different RDI categories (10 to 19.9, 20 to 29.9, 30 to 39.9, >40) and the prevalence of PP in each category was calculated. But before that, since the sleep time spent in the supine position is a major factor correlating with RDI, we evaluated the sleep time in the supine position for the four RDI categories in a random sample of 20 patients in each group. No significant differences for sleep time in the supine position were found among the four groups (p=0.35). The mean sleep time spent (min±SD) in the supine position for the four RDI categories was 131.0±72.1, 165.4±95.8, 148.2±82.6, and 174.4±81.4 min, respectively. The PP prevalence remained high and fairly steady (between 65.1% and 69.0%) in the mild-moderate categories (RDI 10 to 19.9, 20 to 29.9, and 30 to 39.9), but showed a marked and significant reduction to 32.4% in the most severe category (RDI>40), (χ^2=58.8, df=3, p=0.001; Table 6). Although a positive trend toward an inverse relationship was obtained (Kendall test), this result suggests that rather than an overall inverse relationship with positional dependency, RDI showed a threshold effect on positional dependency with a significant decrease in the prevalence of PP in the most severe RDI category. A test for the identification of the threshold was carried out and the RDI threshold point that maximized the χ^2 test for the positional dependency was RDI=40. The entire group was then divided into a nonsevere category (RDI≤40) and a severe category (RDI>40). In the nonsevere category, the PP prevalence was 66.6% compared with only 32.4% in the severe category (χ^2=58.38, df=1, p=0.0001; Fig 2).

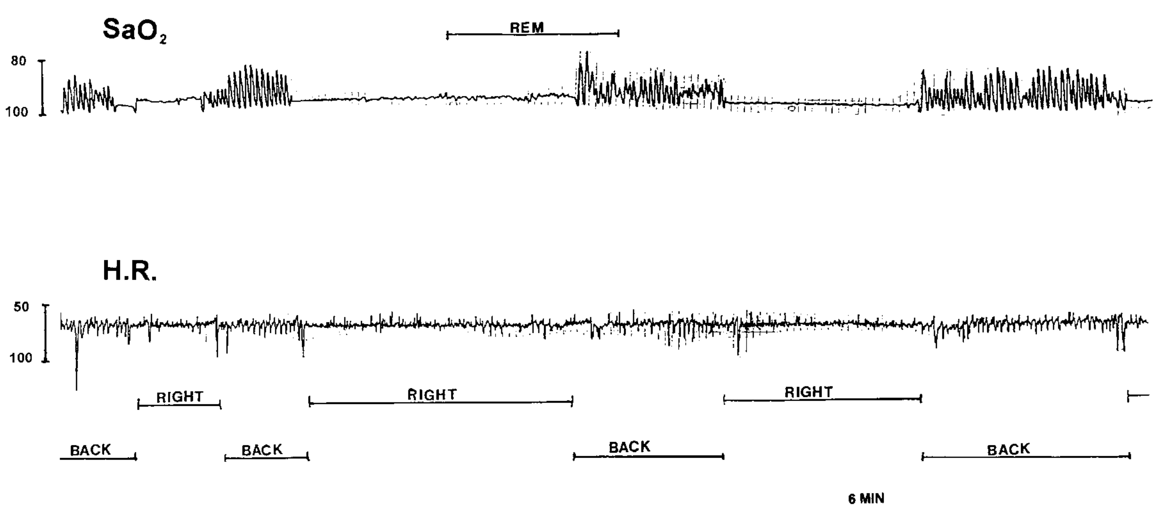

FIGURE 1. Effect of body position on obstructive sleep apnea. Heart rate (HR), *bottom,* and SaO_2 tracing, *top,* on a chart recorder at paper speed of 10 cm/h in a typical positional OSA patient (PP). In the SaO_2 tracing, each peak represents an episode of decreased oxygen saturation (desaturation) and the return to baseline (resaturation) as a consequence of apneas and/or hypopneas. Note the frequent desaturation-resaturation episodes in parallel with bradycardia/tachycardia changes in the HR tracing while sleeping on the back and their absence while sleeping on the right side. This patient achieved complete relief of breathing abnormalities during sleep by merely avoiding the supine position.

Table 4—*PLM Data**

	PP (n=115)	NPP (n=79)	p Value
Total PLM	129.3 (134.9)	136.3 (122.0)	0.71
Arous PLM	62.8 (71.8)	71.0 (75.0)	0.44
PLM I	23.0 (25.0)	25.7 (24.0)	0.44
PLM AI	11.5 (13.7)	13.9 (14.4)	0.25

*All values are mean (SD). Arous PLM=number of PLM causing arousals.

Table 5—*MSLT Data**

	PP (n=194)	NPP (n=175)	p Value
Nap 1	9.9 (6.3)	8.6 (6.1)	
Nap 2	9.0 (5.9)	7.9 (5.8)	0.054
Nap 3	8.0 (5.2)	7.4 (5.6)	
Nap 4	11.6 (6.4)	10.6 (14.0)	
Av MSLT	9.6 (4.5)	8.4 (4.6)	0.01

*The values refer to sleep latency time in minutes (SD). The Av MSLT is the average sleep latency time for the four naps. The naps were carried out at 9AM, 11AM, 1PM, and 3PM.

Thus, an OSA patient with a severe RDI is less likely to be positional than an OSA patient with a mild to moderate RDI.

Effect of BMI on Positional Dependency

To estimate the correlation of BMI with positional dependency in OSA patients, the entire group was first divided into five different categories (20 to 24.9; 25 to 29.9; 30 to 34.9; 35 to 39.9; and ≥40) and the percentage of PP in each category was calculated. A steady, marked, and significant reduction was observed in the prevalence of PP with the increase in BMI in the five categories (70.5, 67.6, 46.3, 34.8, and 33.3%, respectively); χ^2=42.2, df=4, p=0.001; Table 6). In addition, when the total group was divided into two categories, nonobese (BMI≤30) and obese (BMI>30), the PP prevalence in the nonobese group was 68.0% compared to only 42.2% in the obese group (χ^2=38.61, df=1, p=0.0001; Fig 2).

Thus, BMI showed an inverse relationship to positional dependency and a nonobese OSA patient is more likely to be positional than an obese one.

Effect of Weight Loss on Positional Dependency

The data from four severely obese OSA patients who refused nCPAP treatment but successfully lost weight by changing their eating habits are summarized in Table 7. All three NPP cases (patients 1 through 3) were converted into PP cases by weight loss. In all four cases, after weight reduction, the RDI fell to normal (RDI<10) while sleeping in the lateral position. However, the RDI still remained elevated after weight loss in all four while sleeping in the supine position, and in one case (case 3), the supine RDI actually increased despite the weight loss. The weight reduction was associated with a 91.1% reduction in the RDI in the lateral position but with only a 38.9% reduction in the supine

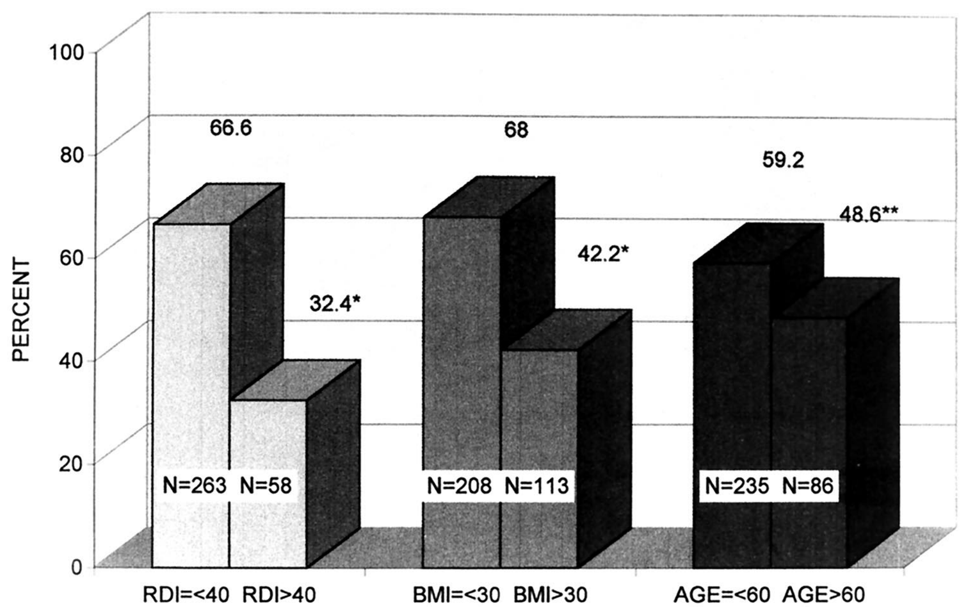

FIGURE 2. Effect of RDI, BMI and age on the prevalence of OSA positional patients (PP). The percent of PP is significantly higher in OSA patients with RDI <40, with BMI <30, and in OSA patients younger than 60 years old. Asterisk indicates p=0.0001; two asterisks, p=0.081.

Table 6—*Prevalence of PP According to Different Categories of RDI, BMI, and Age**

Variable	No. of OSA Patients	No. of PP (%)
RDI		
10-19.9	215	140 (65.1)
20-29.9	109	74 (67.9)
30-39.9	71	49 (69.0)
≥40	179	58 (32.4)
BMI		
20-24.9	44	31 (70.5)
25-29.9	262	177 (67.6)
30-34.9	175	81 (46.3)
35-39.9	69	24 (34.8)
≥40	24	8 (33.3)
Age, yr		
20-39.9	49	29 (59.2)
40-59.9	348	206 (59.2)
≥60	177	86 (48.6)

*Effect of RDI, BMI, and age on the prevalence of OSA PP. The number and percent of PP for each category are presented. The prevalence of PP shows a significant reduction as RDI (χ^2=58.8, df=3, p=0.001) and BMI (χ^2=42.2, df=4, p=0.001) increased. For increasing age, these differences were found to be only of borderline statistical significance (χ^2=5.58, df=2, p=0.06).

position. Thus, weight loss causes a much more striking improvement in the lateral RDI than in the supine RDI.

Effect of Age on Positional Dependency

To estimate the correlation of age with positional dependency in OSA patients, the entire group was first divided into three different age categories. The two youngest categories (age 20 to 39.9 years and 40 to 59.9 years) showed an equal prevalence of PP (59.2%), while in the 60+ group, the PP prevalence decreased to 48.6%. These differences were found to be only of borderline statistical significance (χ^2=5.58, df=2, p=0.06; Table 6). The entire group was then divided into a younger (age≤60 years) and older (age>60 years) group. In the younger group, the prevalence of PP was 59.2% compared with 48.6% in the older one (χ^2=5.58, df=1, p=0.01; Fig 2). Thus, age was a contributing factor of only borderline significance for positional dependency but older OSA patients were still less likely to be positional than younger ones.

Stepwise Multivariate Logistic Regression Analysis

To estimate the relative influence of each of these independent variables on the positional dependency, a stepwise multivariate logistic regression model was built and analysis was performed. Table 8 summarizes the results of this analysis. As can be seen, the variable that most significantly predicted positional dependency was RDI which had a greater improvement goodness of fit (χ^2=58.38, df=1, p=0.0001). The second variable included into the model was BMI, also with an improvement goodness of fit (χ^2=38.61, df=1, p=0.0001). The last variable included into the model was age (χ^2=5.58, df=1, p<0.018).

Discussion

This study has shown that 55.9% of the 574 adult OSA patients whose conditions were diagnosed in our sleep unit have at least twice as many apneas/hypopneas in the supine than in the lateral position. These PP were found to be younger and weigh less than the NPP. In addition, they had fewer and less severe breathing abnormalities than the NPP group. Consequently, their nocturnal sleep quality was better preserved and, according to MSLT data, they were less sleepy during daytime hours. This high prevalence of the positional dependency phenomenon found in our OSA patients is similar to that found by other investigators,[4,13,20] but our study is based on a much larger sample than those previous ones. This high percentage of PP underlines the major contribution played by body position during sleep on the occurrence and severity of OSA.

The most dominant variable that correlates with positional dependency in OSA patients is RDI, followed by BMI which also adds a significant contribution to the prediction of positional dependency. Age, although significant, adds only a minor improvement to the prediction of this positional dependency phenomenon.

PP were found to be younger than NPP. The difference in the mean age between the PP group and the NPP group was small (2 years) but significant. Support for this result was obtained when the entire OSA patient group was divided into two age categories. The prevalence of PP in the youngest category (age≤60 years) was found to be significantly higher (59.2%) than the 48.6% observed in the older group (age>60 years), and when age was included in the multivariate stepwise regression analysis, it showed a small but still significant contribution to the prediction of positional dependency. These results differ from those of others.[13,27]

RDI showed a strong relationship to positional dependency. First of all, by the univariate analysis, it was found that the prevalence of PP in the severe OSA patients is significantly lower than that found in the mild to moderate OSA patients. Second, the multivariate analysis showed that RDI is the most dominant factor that could predict the positional dependency. Thus, by knowing the RDI of an OSA patient, the chances of predicting correctly if the patient is positional or not are quite high, and higher than if only the BMI is known and certainly if only age is known. We, as others,[28] found that most of the adult mild-moderate OSA patients are positional or,

Table 7—*The Effect of Weight Loss on Positional Dependency**

	Case No.			
	1	2	3	4
Age, yr/sex	45/M	40/M	58/M	45/M
Duration of follow-up, mo	7	20	3	3
Initial weight, kg	103.0	121.5	98.5	92.0
Final weight, kg	87.0	89.0	82.0	82.0
Δ weight	−16.0	−32.5	−16.5	−10.0
% Δ weight	−15.5	−26.7	−16.8	−10.9
Initial BMI	30.7	38.4	34.5	30.7
Final BMI	25.9	28.8	28.7	27.4
RDI total				
Initial	80.1	85.9	58.4	83.1
Final	21.1	9.0	15.7	25.2
RDI supine				
Initial	90.2	94.5	65.6	105.2
Final	33.7	15.6	83.7	65.9
Δ RDI	−56.5	−78.9	+18.1	−39.3
% Δ RDI	−62.6	−83.5	+27.6	−37.4
RDI lateral				
Initial	70.0	63.7	55.7	47.3
Final	0	3.5	6.3	9.0
Δ RDI	−70.0	−60.2	−49.4	−38.3
% Δ RDI	−100	−94.5	−88.7	−81.0

*Total RDI is the RDI for the total sleep period. Initial refers to the values obtained at the first PSG evaluation. Final refers to the values obtained at the PSG evaluation after weight loss. Duration of follow-up is the time in months between the first and the second PSG evaluation.

expressing this in another way, that most of the breathing abnormalities occur when they sleep in the supine position. Consequently, these OSA patients are the ones in whom positional therapy (avoiding the supine posture) should play an important role in their treatment. Some of them will eliminate all the breathing abnormalities merely by only avoiding the supine posture. On the contrary, patients with severe OSA are less likely to be positional. Their condition is so severe that they have breathing abnormalities in all different body postures. For them, nCPAP treatment is certainly the treatment of choice.

If mild-moderate OSA patients are mainly PP, it could also mean that "positionality" is perhaps a characteristic of the natural development of the OSA entity, and as the severity increases (as occurs with increase in weight), the positional OSA patient may convert into a nonpositional one. The reverse appears also to be true and this was demonstrated in those obese OSA patients who after losing weight were converted from NPP into PP (Table 7). With this reasoning in mind, it is also possible that alcohol intake and sleep deprivation could convert a PP into a NPP, but this has yet to be proven. A similar parallelism seems to exist in the natural development of snoring. The spouse/partner of a typical snorer patient often notes that initially the patient snored only when sleeping on the back but as the severity of the snoring increased (often related to a gain in weight), snoring is also present when sleeping on the sides.

Some authors[2,5] have found that PP is weight-dependent, being more common in the less obese. Others have found no differences in BMI between the PP and NPP group.[13] The anthropomorphic data in our study demonstrated a marked inverse relationship between the prevalence of PP and the degree of severity of obesity.

Whereas 68% of those with a BMI ≤30 were PP, this was the case for only 42.2% of those with a BMI >30. The finding in our study that NPP were on average 6.5 kg heavier than PP suggests that approx-

Table 8—*Multivariate Stepwise Logistic Regression**

Variable	β	SE	WALD	df	OR (95% CI)	p Value
RDI>40	0.6493	0.0988	43.1522	1	4.16 (2.9-5.9)	0.0001
BMI>30	0.4811	0.0917	27.5186	1	2.91 (2.1-4.0)	0.0001
Age>60 yr	0.2254	0.0983	5.2521	1	1.53 (1.1-2.1)	0.0219

*The variable that most significantly explains positional dependency in OSA patients was RDI, followed by BMI and age. OR=odds ratio; CI=confidence interval; WALD=statistical test used in the regression.

imately this amount of weight would have to be lost to convert a NPP into a PP. These results also imply that in a population of OSA patients who are on average more obese than those seen by us, the prevalence of PP could be significantly less than that found in the present study.

Lloyd[5] found that the degree of obesity correlates better with the RDI in the lateral position than in the supine position, suggesting that weight loss is more effective in improving breathing abnormalities in the lateral than in the supine position. In the four obese patients with severe OSA presented in Table 7, we clearly verified this dominant relationship between weight loss and the improvement of breathing abnormalities during sleep in the lateral position compared to the supine position. It should be noted that all four patients achieved a normal RDI (<10) after weight reduction when they avoided the supine position.

The fact that the PP group had a higher sleep efficiency shows that they enjoy a better sleep quality than the NPP group. A better preserved sleep architecture in the PP group is also noted as judged by higher percentages of deep (stages 3 and 4) and lower percentages of light (stages 1 and 2) sleep stages. Two other important PSG features demonstrating the more disturbed sleep architecture in the NPP group were the increased number of short arousals (<15 s) and the increased time spent awake after sleep onset. The difference in the sleep architecture between the two groups is not surprising. Since PP had fewer and less severe breathing abnormalities as judged by the AI and RDI values and by the min SaO_2 values in both REM and NREM sleep, their sleep was less disturbed. Only one other study[13] in a smaller sample of patients also found similar differences in sleep architecture and severity of breathing abnormalities between the PP and NPP groups.

No differences in either the total number of limb movements or limb movements causing arousal or in the indexes per sleep hour of both parameters were observed between the two groups. This suggests that the difference in the number of short arousals (<15 s) between these two groups is mainly accounted for by the respiratory disturbances and not by the PLMs.

We found no information in the literature about daytime sleepiness in the PP group compared to the NPP group. The percentage of patients who had an MSLT was significantly higher in the NPP group. Since all patients were selected to carry out the MSLT based on the presence of complaints about daytime sleepiness *before* the nocturnal PSG was carried out (consequently before knowing if they belong to the PP or NPP group), and before the MSLT test was performed, this result shows that patients in the NPP group complained more frequently about daytime sleepiness than patients in the PP group. Although the two-way ANOVA for our MSLT data results in a borderline ($p=0.054$) significant difference between the two groups, for each of the naps, the differences were consistent. In addition, our MSLT data show that the NPP group had a significantly shorter average MSLT than the PP group. Thus, these data are consistent with the higher prevalence of the daytime sleepiness complaints in the NPP group and also show that patients of the NPP group are objectively sleepier than those in the PP group. These MSLT data also correlate with the nocturnal sleep data. The NPP group had higher RDI values and more severe breathing abnormalities, all of which cause a more disturbed sleep. Nevertheless, because the PP group had a longer TST than the NPP group, the differences obtained in the MSLT could be due to a combination of better sleep quality in addition to an increased TST in the PP group.

Cephalometric data have shown that many OSA patients have a narrower pharynx than non-OSA control subjects.[29-31] Adopting the supine position from the sitting position causes a further narrowing of all segments of the pharynx during the awake state in normal[32-34] as well as in OSA patients.[35,36] This pharyngeal cross-sectional narrowing is probably due mainly to the effect of gravity, which increases the apposition of the soft palate, uvula, tongue, and epiglottis to the posterial pharyngeal wall.[32-36] This gravity effect also causes an increase in the tongue cross-sectional area,[33] uvular width,[36] and soft palate thickness,[33] which would also contribute to the reduced pharyngeal cross-sectional area. These anatomic changes, which occur in the upper airway (UA) by adopting the supine position, are followed by one major physiologic change: an increase in the UA resistance. The direct consequence of this is that breathing during sleep becomes more difficult, increasing the probability of the occurrence of episodes of partial UA obstruction (manifested as snoring and/or hypopneas) or complete UA obstruction (manifested as apneas).

Since our results demonstrate that the body position effect is more dominant in the mildest forms of OSA, we suggest that the recently described upper airway resistance syndrome (UARS),[37] which appears to be the mildest form of UA disturbance during sleep, occurring even in nonsnoring sleepy patients, may be caused mainly by sleeping in the supine position. If this is true, avoiding the supine position during sleep might be enough to prevent the increase of UA resistance, and as a consequence, avoid the need for nCPAP treatment, which has been shown to be associated with very poor compliance in these UARS patients.[38] Most important perhaps, at least for some patients, avoiding the supine position could prevent the gradual progression over time from partial to complete pharyngeal obstruction during sleep. This proposed relationship between UARS and the supine position needs to be evaluated.

One limitation of our study is that we studied the body position effect without taking into consideration the effect of head/neck position on the occurrence and severity of breathing abnormalities during sleep. Future studies should consider this aspect, which might also be of significance. Currently data on this point are limited and conflicting.[34,39-41]

Although several studies have already shown a marked improvement in breathing function by sleeping in the lateral position,[1-9] the anatomic and physiologic mechanisms responsible for this phenomenon have not yet been clarified. In normal awake subjects, Jan et al[34] recently showed that even though pharyngeal areas were smaller in the supine than in the sitting position, no differences were found between the lateral and supine postures. However, the anatomic and functional aspects of the pharynx change markedly during sleep.[29-31,37,42] Unfortunately, studies comparing the pharyngeal cross-sectional areas in the lateral vs supine position in normal subjects and especially in OSA patients during sleep are lacking and are urgently needed. Shepard and Thawley,[43] in OSA patients, studied the effect of body position and sleep stage on the regions over which the UA collapses. They found that in most patients, the site and extent of the UA collapse were similar in the supine and lateral position independent of the sleep stage. This suggests that although body position plays an important role in determining whether UA collapse occurs, when it does, the anatomic location and the extent of the collapse are similar in the supine and lateral position.

Two studies have examined the anatomic changes in the UA in a PP group and compared them to either unselected OSA patients[44] or to an NPP group.[45] Kovacevic-Ristanovic et al[44] found that the PP group had a significantly larger posterior airway space, a less elongated soft palate, and somewhat more prominent retrognathia than unselected OSA patients. Pevernagie and Shepard[45] have recently described for the first time the differences in the size and shape of the UA in the PP and NPP groups while subjects were awake. These differences were found mainly in the velopharyngeal segment of the UA (the retropalatal area) where the minimal cross-sectional area is normally located. The size of the UA was significantly different in the two groups; the minimal cross-sectional area of the PP group was almost twice that of the NPP group in both the supine and right lateral positions. The UA shapes of the two groups were also different—elliptical in the PP group and circular in the NPP group. The differences in shape were due predominantly to the significantly greater lateral diameter in the PP group, the anteroposterior (A-P) diameter being no different in the two groups. The PP group, due in part to the gravity effect on the soft tissues, which reduces the A-P diameter significantly,[45] presents breathing abnormalities during sleep while adopting the supine position. These data also suggest that when the lateral position is adopted by these patients, the A-P diameter is increased, and the lateral walls are far enough apart that they will not come together and block the pharyngeal lumen. Thus, sufficient airway space is preserved to avoid a complete collapse of the UA. In the NPP group, however, the pharyngeal cross-sectional area is reduced to about half of that of the PP group due primarily to a much reduced lateral diameter, and changing to the lateral position cannot prevent the pharyngeal collapse.[45]

Despite the high prevalence of positional dependency in OSA observed in this study and others, and despite the encouraging preliminary results of using positional therapy of Cartwright et al[15] many years ago, it is surprising how few investigations have been carried out on the therapeutic efficacy of avoiding the supine position during sleep in PP. Moreover, despite the standards of practice recommendation that require monitoring of body position in PSG studies,[46] it appears that only a few sleep disorders units are recording and analyzing their sleep-related breathing abnormalities data, including RDIs, by body position. In addition, how precise would be the estimation of the results of any surgical or other intervention procedure in OSA patients without taking into account the sleep time spent in the supine position in the pre- and post-PSG? An inexplicable worsening after surgery for OSA in a positional OSA patient could be simply due to the much longer sleep time spent in the supine position in the postintervention PSG, compared to that time in the presurgery PSG and not to a real failure of the surgery. The crucial issue is that in positional OSA patients, the severity of the disease is mainly related to the sleep time spent or not spent in the supine posture.

In some recent reviews on OSA, the role of positional therapy has not even been mentioned.[47,48] One possible explanation is that many PP still continue to snore, and sometimes loudly, when they sleep in either the lateral or prone position. Consequently, their spouses continue to complain about snoring. Thus, even though in many cases this therapy may clearly produce a major improvement to the breathing function during sleep, the fact that snoring continues in the lateral or prone positions may limit the long-term effectiveness of this approach. Another possible explanation is that this positional therapy is not a radical solution for the most severe OSA patients who are the ones that urgently search for treatment. For these patients, nCPAP is at present the treatment of choice. Nevertheless, some of those obese patients with severe OSA who refuse nCPAP treatment adopt a sitting posture during sleep, achieving an improvement in their breathing function during sleep. This type of postural therapy has also been objectively shown to improve the breathing function during sleep in these patients.[49] In addition, if those patients lose enough weight to become PP, they can be successfully treated by positional therapy as was demonstrated in the four obese OSA patients in Table 7.

We place a tennis ball into a pocket of a wide cloth band or belt attached around the abdomen so that the ball lies in the center of the back. When the patient rolls onto the back he feels the pressure of the ball and instinctively rolls back onto his side again. Several other methods have been used to train patients to avoid the supine position. Some may use a T-shirt with a long vertical pocket holding three or four tennis balls along the back. This is perhaps less likely to slip out of place during sleep. Cartwright et al[15] used an alarm system that momentarily woke the patients whenever they lay on their backs. The time spent in the supine position while using this alarm system decreased from 51.4 to 2.1% of TST. After the patient went home and practiced avoiding supine sleep without an alarm for 3 months, a repeat PSG evaluation, however, revealed that the actual sleep time spent in the supine position was 24.1%. Although some recent preliminary studies[50] have provided some data on the long-term efficacy of positional therapy, more studies investigating this issue are urgently needed. This is particularly the case because of the difficulties often experienced by OSA patients in complying with current therapeutic modalities such as nCPAP[51] and weight reduction,[52] and the uncertain results of surgical interventions[53] and prosthetic intraoral devices.[54]

In a recent study[55] we investigated the effect of avoiding the supine position during sleep for a 1 month period on 24-h blood pressure (BP) in 13 positional OSA patients. In all the patients (hypertensive and normotensive patients) there was a reduction in the 24-h mean BP values. A significant reduction was observed for the mean 24-h, for the mean awake BP, and mean asleep BP. BP variability and BP load also fell significantly. Since, as shown in the present study, the majority of OSA patients have supine-related breathing abnormalities, and since about a third of all hypertensive patients have OSA, avoiding the supine position during sleep, if confirmed by future larger studies, could become a new nonpharmacological form of treatment for many hypertensive patients.

In conclusion, this study has shown, in a large population of OSA patients, that the majority (55.9%) were found to have at least twice as many apneas/hypopneas in the supine than in the lateral position. These PP are on the average younger and weigh less than NPP. They also had fewer and less severe breathing abnormalities than the NPP group. Consequently, their nocturnal sleep quality was better preserved and, according to MSLT data, they were less sleepy during daytime hours. The likelihood of being an OSA PP is correlated with RDI, BMI, and age, generally in a reverse relationship, *ie,* an OSA patient with a severe RDI, and who is obese and older than age 60 years, is significantly less likely to be positional than a OSA patient with a mild-moderate RDI, who is nonobese and younger than 60 years of age. The above data stress the profound effect of body position during sleep on the frequency and severity of breathing abnormalities in OSA patients. The data also reinforce the crucial necessity of not only monitoring body position during PSG evaluation of every suspected OSA patient, but also of reporting the severity of OSA (RDI) according to body position.

ACKNOWLEDGMENTS: We would like to thank Yael Vila for her advice and assistance in the statistical analysis, Danna Gal for preparing the figures and tables, and the technical team of the Sleep Disorders Unit at the Loewenstein Hospital-Rehabilitation Center for the dedicated and responsible work that they performed.

References

1 Lerner SA, Cecil WT. The effect of sleeping posture on obstructive sleep apnea [abstract]. Chest 1984; 86:327
2 Cartwright RD. Effect of sleep position on sleep apnea severity. Sleep 1984; 7:110-14
3 Phillips BA, Okeson J, Paesani D, et al. Effect of sleep position on sleep apnea and parafunctional activity. Chest 1986; 90:424-29
4 Lloyd SR, Cartwright RD. Physiologic basis of therapy for sleep apnea [letter]. Am Rev Respir Dis 1987; 136:525-26
5 Lloyd SR. The sleep position effect in sleep apnea as a continuous variable [abstract]. Sleep Res 1988; 17:14
6 Miki H, Hida W, Kikuchi Y, et al. Effect of sleep position on obstructive sleep apnea. Tohuku J Exp Med 1988; 156(suppl): 143-49
7 Demirozu MC, Elsasser S, Gazeroglu HB, et al. The effect of body weight, posture and sleep stage on obstructive sleep apnea syndrome [abstract]. Sleep Res 1990; 19:319
8 Demirozu MC, Razzetti A, Gazeroglu HB, et al. Sleep body position influences apnea frequency and duration in obese and non-obese patients with obstructive sleep apnea [abstract]. Sleep Res 1991; 20A:302
9 Diaz T, Norman SE, Kiel M, et al. A comparison of apnea/hypopnea index and its relationship to sleep stages and body posture during the first and second half of a diagnostic polysomnogram in patients with sleep apnea syndromes [abstract]. Sleep Res 1991; 20:233
10 Kavey NB, Blitzer A, Gidro-Frank S, et al. Sleeping position and sleep apnea syndrome. Am J Otolaryngol 1985; 6:373-77
11 George CF, Millar TW, Kryger MH. Sleep apnea and body position during sleep. Sleep 1988; 11:90-99
12 Miles L, Bailey A. Evaluation of sleep apnea treatment must be related to sleeping position [abstract]. Sleep Res 1990; 19:256
13 Pevernagie DA, Shepard JW. Relations between sleep stage, posture and effective nasal CPAP levels in OSA. Sleep 1992; 15:162-67

14 Braver HM, Block J. Effect of nasal spray, positional therapy and the combination thereof in the asymptomatic snorer. Sleep 1994; 17:516-21
15 Cartwright RD, Lloyd S, Lilie J, et al. Sleep position training as treatment for sleep apnea syndrome: a preliminary study. Sleep 1985; 8:87-94
16 Cartwright RD, Ristanovic R, Diaz F, et al. A comparative study of treatments for positional sleep apnea. Sleep 1991; 14:546-52
17 Jackson EI, Schmidt HS. Modification of sleeping position in the treatment of obstructive sleep apnea [abstract]. Sleep Res 1982; 11:149
18 Chaudhary BA, Chaudhary TK, Kolbeck RC, et al. Therapeutic effect of posture in sleep apnea. South Med J 1986; 79:1061-63
19 Katz A, Dinner DS. The effect of sleep position in the diagnosis of obstructive sleep apnea: a word of caution [abstract]. Cleve Clin Q 1992; 59:634-36
20 Dyonzak J, Cartwright RD. Prevalence of positional differences in obstructive sleep apnea. Sleep Res 1993; 22:191
21 Young T, Palta M, Dempsey J, et al. The occurrence of sleep-disordered breathing among middle-aged adults. N Engl J Med 1993; 238:1230-35
22 Rechtschaffen A, Kales A, eds. A manual of standardized terminology techniques and scoring system for sleep stages of human subjects. Los Angeles: Brain Information Service/ Brain Research Institute, University of California at Los Angeles, 1968
23 Guilleminault C. Sleep and breathing in sleeping and waking disorders: indications and techniques. Menlo Park, Calif: Addison-Wesley, 1982; 155-82
24 American Sleep Disorders Association. EEG arousals: scoring rules and examples, Sleep 1992; 15:173-84
25 Coleman RM. Periodic movements in sleep (nocturnal myoclonus) and restless legs syndrome. In: Guilleminault C, ed. Sleeping and waking disorders: indications and techniques. Menlo Park, Calif: Addison-Wesley, 1982; 265-95
26 Association of Sleep Disorders Centers Task Force on Daytime Sleepiness. Guidelines for the multiple sleep latency test (MSLT): a standard measure of sleepiness. Sleep 1989; 9:519-24
27 Nichols CD. Differential effects of obesity and body position in older vs younger patients with obstructive sleep apnea [abstract]. Sleep Res 1994; 23:298
28 Swieca J, Westbrook PR. Relationship between body position dependence of apnea and hypopnea and overall severity of sleep-disordered breathing [abstract]. Sleep Res 1994; 23:333
29 Fleetham JA. Upper airway imaging in relation to obstructive sleep apnea. Clin Chest Med 1992; 3:399-416
30 Hudgel DW. The role of upper airway anatomy and physiology in obstructive sleep apnea. Clin Chest Med 1992; 3:383-98
31 Pepin JL, Levy P, Veale D, et al. Evaluation of the upper airway in sleep apnea syndrome. Sleep 1992; 15:S50-55
32 Fouke JM, Strohl KP. Effect of position and lung volume on upper airway geometry. J Appl Physiol 1987; 63:375-80
33 Pae E-K, Lowe AA, Sasaki K, et al. A cephalometric and electromyographic study of upper airway structures in the upright and supine positions. Am J Orthod Dentofacial Orthop 1994; 106:52-59
34 Jan MA, Marshall I, Douglas NJ. Effect of posture on upper airway dimensions in normal human. Am J Respir Crit Care Med 1994; 149:145-48
35 Brown IB, McClean PA, Boucher R, et al. Changes in pharyngeal cross-sectional area with posture and application of continuous positive airway pressure in patients with obstructive sleep apnea. Am Rev Respir Dis 1987; 138:628-32
36 Yildirim N, Fitzpatrick MF, Whyte KF, et al. The effect of posture in upper airway dimensions in normal subjects and in patients with the sleep apnea/hypopnea syndrome. Am Rev Respir Dis 1991; 144:845-47
37 Guilleminault C, Stoohs R, Clark A, et al. A cause of excessive daytime sleepiness: the upper airway resistance syndrome. Chest 1993; 104:781-87
38 Guilleminault C, Kim Y-D, Stoohs R. Upper airway resistance syndrome. Oral Maxillofac Sur Clin North Am 1995; 7:243-56
39 Safar P, Esscaraga LA, Chang F. Upper airway obstruction in the unconscious patient. J Appl Physiol 1959; 14:760-64
40 Rubinstein I, McClean PA, Boucher R, et al. Effect of mouthpiece, noseclips and head position on airway area measured by acoustic reflections. J Appl Physiol 1987; 63:1469-74
41 Wilson SL, Thach BT, Brouillette RT, et al. Upper airway potency in the human infant: influence on airway pressure and posture. J Appl Physiol 1980; 48:500-04
42 Hudgel DW, Brooks BI, Harasick TM. Measurements of awake upper airway caliber do not predict upper airway resistance during sleep [abstract]. Am Rev Respir Dis 1989; 139:A374
43 Shepard JW Jr, Thawley SE. Localization of upper airway collapse during sleep in patients with obstructive sleep apnea. Am Rev Respir Dis 1990; 141:1350-55
44 Kovacevic-Ristanovic R, Alder G, Lloyd S, et al. Cephalometric analysis in positional sleep apneics [abstract]. Sleep Res 1989; 18:249
45 Pevernagie DA, Stanson AW, Sheedy BK, et al. Effects of body position on upper airway size of patients with obstructive sleep apnea. Am J Respir Crit Care Med 1995; 152: 179-85
46 Martin RJ, Chairman; Block AJ, Cohn MA, Conway WA, et al. Indications and standards for cardiopulmonary sleep studies. Sleep 1985; 8:371-79
47 Kryger MH. Management of obstructive sleep apnea. Clin Chest Med 1992; 13:481-92
48 Polo O, Berthon-Jones M, Douglas NJ, et al. Management of obstructive sleep apnea/hypopnea syndrome. Lancet 1994; 344:656-60
49 Mcevoy RD, Sharp DJ, Thornton AT. The effect of posture on obstructive sleep apnea. Am Rev Respir Dis 1986; 133: 662-66
50 Freebeck P, Stewart D. Positional training while you sleep [abstract]. Sleep Res 1995; 24:235
51 Kribbs NB, Pack AI, Kline LR, et al. Objective measurements of patterns of nasal CPAP use by patients with obstructive sleep apnea. Am Rev Respir Dis 1993; 147:887-95
52 Strobel RJ, Rosen RC. Obesity and weight loss in obstructive sleep apnea: a critical review. Sleep 1996; 19:104-15
53 Rodenstein DO. Assessment of uvulopalatopharyngoplasty for the treatment of sleep apnea syndrome. Sleep 1992; 15:556-62
54 Clark GT, Arand D, Chung E, et al. Effect of anterior mandibular positioning in obstructive sleep apnea. Am Rev Respir Dis 1993; 147:624-29
55 Berger M, Oksenberg M, Silverberg DS, et al. Avoiding the supine position during sleep in obstructive sleep apnea (OSA) patients: the effect on 24 hr blood pressure. J Hum Hypertension 1997 (in press)