Prevalence of Sleep Apnea and Electrocardiographic Disturbances in Morbidly Obese Patients

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Abstract

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Objective: To determine the prevalence of sleep apnea in morbidly obese patients and its relationship with cardiac arrhythmias.

Research Methods and Procedures: Fifty-two consecutive morbidly obese (body mass index $\geq$ 40 kg/m^2) outpatients from the Obesity Clinic of the National Institute of Nutrition Salvador Zubirán underwent two nights of polysomnography with standard laboratory techniques. Electrocardiographic polysomnography signals (Lead II) were evaluated by two experienced cardiologists, and sleep complaints were measured with a standard sleep questionnaire (Sleep Disorders Questionnaire). In order to make comparisons between groups with different severities of sleep-disordered breathing, we classified the patients in four groups using the apnea-hypopnea index (AHI): Group 1, AHI 5 $<$ 15 (n = 10); Group 2, AHI 15 $<$ 30 (n = 10); Group 3, AHI 30 $<$ 65 (n = 14); Group 4, AHI $\geq$ 65 (n = 17).

Results: A wide range of sleep-disordered breathing, ranging from AHI of 2.5 to 128.9 was found. Ninety-eight percent of the sample (n = 51) had an AHI $\geq$ 5 (mean = 51 $\pm$ 37), and 33% had severe sleep apnea with AHI $\geq$ 65 with a mean nocturnal desaturation time of $<$65% over 135 minutes. Electrocardiographic abnormalities were present in 31% of the patients. Cardiac rhythm alterations showed an association with the level of sleep-disordered breathing and oxygen desaturation.

Discussion: We conclude that there is a high prevalence of sleep apnea in morbidly obese patients and that the risk for cardiac arrhythmias increases in this population in the presence of a severe sleep apnea (AHI $\geq$ 65) with severe oxygen desaturation ($Sao_2 \leq$ 65%).

Key words: obstructive sleep apnea syndrome, massive obesity, cardiac rhythm disturbances, oxyhemoglobin desaturation

Introduction

It is known that obese patients have compromised respiratory function while awake and upright and that their ventilatory function may become worse when they assume the supine position and even more deteriorated when they sleep (1). Sleep apnea is well known to be associated with obesity (2–4) and is characterized by repetitive occlusion of the upper airway during sleep. The main symptoms of the obstructive sleep apnea (OSA) are heavy snoring, excessive daytime somnolence, and disturbed sleep.

Several mechanisms have been suggested to explain the relationship between obesity and sleep apnea, including alterations in upper airway structure (perhaps caused by fat deposition in the neck (5)), alterations in upper airway function, alterations in the balance between ventilatory drive and load, and obesity-induced hypoxemia (6). Regardless of the mechanism, it has been well documented that

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either medical or surgical weight reduction can have a substantial ameliorative impact on OSA (7–9).

Long-term complications of the sleep apnea include pulmonary hypertension, cardiac arrhythmias, and heart failure (10). Some investigators have suggested that cardiac arrhythmias during sleep may be an important cause of death in patients with OSA (11,12). Marked sinus bradycardia and sinus arrest are the arrhythmias most commonly observed. Tilkian et al. (13) observed ventricular tachycardia during OSA in 13% of subjects and frequent ventricular premature depolarizations were present in 67% of the 15 studied patients. Guilleminault et al. (14), in a larger sample with 400 patients, reported ventricular tachycardia in only 3% of patients and ventricular premature depolarizations in 20% of the sample. Shepard et al. (15) have reported marked increase in frequency of ventricular premature depolarizations in sleep apnea patients whose arterial oxygen desaturation fell below 60%. More recently, Flemons et al. (16) evaluated the prevalence of cardiac arrhythmias in 76 patients undergoing polysomnographic study for the evaluation of suspected OSA. They found a low prevalence of cardiac arrhythmias and no differences between patients with and without sleep apnea. These authors suggested that such inconsistencies across studies in the reported prevalence of cardiac arrhythmias result from differences in the severity of OSA.

The aim of the present study was to determine the prevalence of sleep apnea in morbidly obese patients and to examine cardiac rhythm disturbances in patients with different severity of OSA or apnea-hypopnea index (AHI).

Research Methods and Procedures

Patients

Patients were derived from the Obesity Clinic of the National Institute of Nutrition Salvador Zubirán (INNSZ). The INNSZ is a major tertiary referral care center for all medical specialties in Mexico City. The Obesity Clinic is an INNSZ division dedicated to the comprehensive care and research of overweight individuals, staffed by endocrinologists, nutritional scientists, dieticians, and exercise physiologists. A total of 102 consecutive outpatients were asked to participate in the study, which was conducted in the Sleep Clinic of the Department of Neurology at the INNSZ. The study was approved by the local ethics committee, and all patients gave informed consent. Endocrinologists from the Obesity Clinic referred patients to the Sleep Clinic on the basis of the presence of obesity (body mass index [BMI] $\geq$ 30 kg/m^2, as defined by the World Health Organization (17)) and if they had symptoms of sleep apnea (snoring and excessive daytime sleepiness), or if they presented dyspnea, edema, and cyanosis. Eighteen referred patients did not accept referral to the Sleep Clinic. Thirteen more patients accepted referral but did not agree to stay overnight for two consecutive nights. Nineteen studied patients were excluded from this analysis because their BMI was <40 kg/m^2 and thus did not meet the criteria for morbid obesity. Fifty-two patients with morbid obesity, defined by a BMI $\geq$ 40 kg/m^2, comprised the final sample of the study and consisted of 39 women and 13 men, with a mean age of 40 $\pm$ 12 years and mean BMI of 51 $\pm$ 9 (range, 40 to 80 kg/m^2). Twenty-three patients were taking antihypertension medication, 12 nonsteroidal anti-inflammatory drugs, 8 thyroid replacement, 2 metoclopramide, 3 were receiving diuretics, and 14 received no medication at all. Concomitant medical conditions were hypertension (n = 27) (defined by systolic blood pressure $\geq$ 140 mm Hg, diastolic $\geq$ 90 mm Hg, or use of antihypertension medication); diabetes mellitus (n = 20) (following criteria from the Expert Committee on the Diagnosis and Classification of Diabetes Mellitus (18)); Cushing's syndrome (n = 1) (diagnosed by 24-hour urinary-free cortisol assay and the low-dose, high-dose dexamethasone suppression test); hypothyroidism (n = 8) (defined by Thyroid-stimulating hormone > 5.0 mU/liter or usage of thyroid replacement); and hypercholesterolemia (n = 2) (total cholesterol > 240 mg/dL and LDL-C > 160 mg/dL). Seventeen patients had multiple diagnoses, and 11 patients had no other medical condition.

Study Design and Polysomnography

The evaluation consisted of two nights of polysomnographic recording. All recordings were made on an ultrasom workstation (Nicolet Biomedical, Madison, WI) at an emulated paper speed of 10 mm/second and consisted of simultaneous monitoring of the electroencephalogram (C3/A2, O2/A1), electrooculogram, surface mentalis and anterior tibialis electromyogram, and electrocardiogram (EKG) (Lead II). Quantitative evaluation of sleep stages was made visually with conventional Rechtschaffen and Kales (19) criteria with 30-second epochs for rapid eye movement (REM) and stages of non-REM (NREM) sleep. The number of awakenings was scored as the number of times that at least one 30-second epoch of wakefulness was scored after sleep onset. Except as noted below, all data were averaged across the two recording nights. Respiratory movements were monitored using piezoelectric bands for thoracic and abdominal effort. Oral/nasal airflow was monitored by a four-bead thermistor system. Oxygen saturation was recorded with an ear pulse oximeter, expired (end-tidal) CO_2 was measured with capnography (Capnograph/Oximeter BCI-9000), and sampling was done by applying nasal prongs, consisting of a two-channel plastic tube, to the nostrils. The number of abnormal breathing events per hour of sleep was quantified as the AHI (apneas and hypopneas per hour of sleep). Apnea was defined as a decrease in respiratory airflow below 20% of the steady-state amplitude preceding the breathing event. Hypopnea was defined as a reduction in respiratory airflow between 20% and 50%. The

index or number of oxygen desaturations below 90%, 80%, and 65% per hour of sleep ($Sao_2 < 90\%$ index, $Sao_2 < 80\%$ index, and $Sao_2 < 65\%$ index, respectively) and the cumulative oxygen desaturation ($< 90\%$, $< 80\%$, $< 65\%$) times (in minutes) were also quantified. Apneas were considered central if there was cessation of respiratory effort for at least 10 seconds and obstructive if there was cessation of oronasal airflow for at least 10 seconds despite persistent respiratory effort. To make comparisons between groups with different severities of sleep-disordered breathing, we classified the patients in four groups (see Table 1): Group 1, AHI $5 < 15$ ($n = 10$); Group 2, AHI $15 < 30$ ($n = 10$); Group 3, AHI $30 < 65$ ($n = 14$); Group 4, AHI ≥ 65 ($n = 17$).

EKG signals of the second night of recording were analyzed visually on the display monitor by two experienced cardiologists who were blinded to the clinical and polysomnographic features of the patients. The EKG analysis included a display of the EKG signal with no simultaneous display of electroencephalogram signals, airflow, or respiratory effort. Periodic limb movements were scored according to Coleman's criteria (20): a movement was scored when it occurred as part of a series of four consecutive movements that were separated by at least 4 but not more than 90 seconds, with a duration between 0.5 to 5 seconds. A patient was considered to have periodic limb movements (PLM) (or nocturnal myoclonus) when the index (PLMI) (number of PLMs per hour of sleep) was ≥ 5.

Sleep Complaints

The Sleep Disorders Questionnaire (SDQ) has been shown to be a reliable instrument for supporting a diagnosis in patients suspected of having sleep apnea, narcolepsy, psychiatric, or PLM disorders (21). We used the SDQ items to determine the frequency of various symptoms of sleep apnea and other sleep disorders in morbidly obese patients. All patients completed the SDQ after the first night of polysomnographic recording.

Results

Table 1 shows the anthropometric characteristics of the patients in each severity apnea group. The anthropometric variable that best discriminated among the four groups was neck circumference.

A wide range of sleep-disordered breathing, with AHIs ranging from 2.5 to 128.9, was found. Ninety-eight percent of the sample ($n = 51$) had an AHI level of ≥ 5 (mean = 51 ± 37), and 65% of the sample ($n = 34$) had at least one oxygen desaturation below 65% ($Sao_2 < 65\%$). Hypercapnia, defined as an awake end-tidal carbon dioxide concentration > 45 mm Hg coexisted with sleep apnea in 23% of the patients ($n = 12$). These patients demonstrated a mean awake Sao_2 of 81 ± 5%, an awake end-tidal CO_2 of 50 ± 5 mm Hg, and an AHI of 61 ± 32.

Changes in sleep architecture were associated with severity of sleep apnea. The number of awakenings, arousals, and stage transitions increased as the AHI increased. Light sleep (Stage 1) increased at the expense of delta and REM sleep as AHI increased. Table 2 shows the mean value of two nights of polysomnographic recording for the sleep variables. Table 3 shows the blood gas values for each apnea group. The severe sleep apnea group (AHI ≥ 65) had more than 5 hours of severe oxygen desaturation during the

Table 1. Anthropometric characteristics of morbidly obese patients with sleep apnea

Characteristic	Group 1, AHI 5 < 15 ($n = 10$)	Group 2, AHI 15 < 30 ($n = 10$)	Group 3, AHI 30 < 65 ($n = 14$)	Group 4, AHI ≥ 65 ($n = 17$)
Age (years)	39.9 ± 10.6	39.3 ± 12.2	40.6 ± 14.6	39.5 ± 9.6
Sex (women/men)	9/1	8/2	10/4	11/6
BMI (kg/m^2)	48.9 ± 6.7	50.5 ± 10.5	53.4 ± 10.4	52.1 ± 9.6
Neck circumference (cm)	42.6 ± 3.1	43.2 ± 5.4	43.7 ± 4.1	47.0 ± 3.4*
Thorax (cm)	123.6 ± 6.6	130.4 ± 13.5	128.5 ± 9.7	129.6 ± 7.1
Waist (cm)	129.2 ± 16.9	144.8 ± 24.0	138.4 ± 19.7	128.7 ± 14.7†
Hip (cm)	146.4 ± 14.1	153.0 ± 20.5	151.9 ± 18.8	138.8 ± 17.9‡
Waist-to-hip ratio	0.9 ± 0.1	0.9 ± 0.1	0.9 ± 0.1	0.9 ± 0.1

Data represent mean ± SD. F values refer to results of one-way ANOVA across four groups.
* $F = 7.06$, $p < 0.0002$, Scheffé multiple comparison test: Group 4 > Groups 1–3.
† $F = 2.92$, $p < 0.04$, Scheffé multiple comparison test did not differ among groups.
‡ $F = 2.75$, $p < 0.05$, Scheffé multiple comparison test did not differ among groups.

polysomnographic recording, and they spent nearly half of this time with an oxygen saturation of <65%.

Patients with an AHI ≥ 65 complained more of snoring and bothering others by loud snoring as well as falling asleep accidentally in situations that require mild to moderate attention (Table 4). Spearman correlations between complaints and measures of AHI and $Sao_2 < 65\%$ index, respectively, were as follows: SDQ item 20 (rho = 0.33, $p < 0.002$; rho = 0.47, $p < 0.0001$); SDQ item 21 (rho = 0.46, $p < 0.0001$; rho = 0.39, $p < 0.0001$); SDQ item 56 (rho = 0.39, $p < 0.008$; rho = 0.50, $p < 0.0003$). There were no statistically significant differences between the groups in the reported number of work (mean 1.2 ± 0.8) or car accidents (mean 1.4 ± 1.0), which are SDQ items 156 and 157, respectively (1 = none, 2 = one, 3 = two, 4 = three, 5 = four or more accidents).

Cardiac rhythm disturbances were present in 31% of the patients (Table 5). Cardiac rhythm alterations showed an association with the level of sleep-disordered breathing. In the case of ventricular arrhythmias, the prevalence increased more than two times with the severity of the apnea (Table 5; chi-square = 33.75, $p < 0.0001$). Typically, a repetitive cyclical decrease and increase in heart rate is observed in patients with OSA; in our morbidly obese patients, marked bradycardia (defined as a heart rate of less than 40 beats/minutes) was only present in less than 6% of the patients. Table 6 shows the heart rate for each apnea group.

Discussion

Our data showed that OSA has an exceedingly high prevalence (98%) in morbidly obese patients referred to a Sleep Disorders Clinic, and it is severe in 32.7% of the cases (AHI ≥ 65 and extended oxygen desaturation, $Sao_2 < 65\%$ for 135 minutes). Hypercapnia (awake end-tidal $CO_2 > 45$ mm Hg) coexists with OSA in less than 25% of the patients. Peiser et al. (22), in a population of morbidly obese patients scheduled for gastric surgery with complaints of excessive daytime sleepiness, loud and disturbing snoring, and restless sleep, confirmed polysomnographic OSA in 90% of the suspected patients; they estimated about 30% of the adult morbidly obese men suffer from OSA. In a subsequent study from the

Table 2. Sleep variables in morbidly obese patients with sleep apnea

Variables	Group 1, AHI 5 < 15 ($n = 10$)	Group 2, AHI 15 < 30 ($n = 10$)	Group 3, AHI 30 < 65 ($n = 14$)	Group 4, AHI ≥ 65 ($n = 17$)
Total sleep time (minutes)	370.1 ± 76.0	388.6 ± 43.1	365.3 ± 66.6	401.5 ± 58.8
% Sleep efficiency	83.3 ± 13.9	86.5 ± 9.1	82.7 ± 11.8	88.8 ± 6.8
Sleep latency (minutes)	6.5 ± 10.5	8.6 ± 6.9	5.8 ± 5.4	3.6 ± 5.4
REM sleep latency (minutes)	117.6 ± 59.9	126.0 ± 101.5	131.4 ± 82.8	117.3 ± 84.0
Awakenings <1 min	17.0 ± 8.0	17.7 ± 8.3	19.5 ± 10.9	26.5 ± 18.3*
Arousals	5.9 ± 3.5	10.9 ± 9.6	9.9 ± 8.7	14.3 ± 12.7†
Stage transitions	167.9 ± 40.5	159.6 ± 38.1	169.3 ± 46.3	213.9 ± 77.6‡
% Awake	13.1 ± 12.7	10.7 ± 8.4	17.5 ± 17.1	9.9 ± 6.1
% Stage 1	10.4 ± 3.6	12.6 ± 4.6	13.9 ± 6.8	22.0 ± 12.8§
% Stage 2	41.4 ± 9.2	47.5 ± 7.9	44.3 ± 10.0	51.8 ± 13.5¶
% Stage 3 and 4	19.7 ± 7.7	11.9 ± 7.4	13.5 ± 7.1	6.0 ± 5.4**
% REM stage	12.6 ± 5.2	14.5 ± 6.2	11.5 ± 5.2	8.9 ± 5.8††
PLMI	7.0 ± 7.9	13.8 ± 14.5	14.8 ± 23.2	25.6 ± 28.1‡‡

Data represent mean ± SD. F values refer to results of one way ANOVA across four groups.
* $F = 3.14$, $p < 0.03$, Scheffé multiple comparison test did not differ between the groups.
† $F = 3.16$, $p < 0.03$, Scheffé test: Group 4 > Group 1; $p < 0.04$.
‡ $F = 5.42$, $p < 0.002$, Scheffé test: Group 4 > Groups 1–3; $p < 0.05$.
§ $F = 9.76$, $p < 0.00001$, Scheffé test: Group 4 > Groups 1–3; $p < 0.002$.
¶ $F = 4.54$, $p < 0.006$, Scheffé test: Group 4 > Group 1; $p < 0.02$.
** $F = 17.88$, $p < 0.0001$, Scheffé test: Group 4 < Groups 1–3; $p < 0.02$.
†† $F = 4.67$, $p < 0.005$, Scheffé test: Group 2 > Groups 1,3,4; $p < 0.008$.
‡‡ $F = 3.48$, $p < 0.02$, Scheffé test: Group 4 > Group 1; $p < 0.03$.

Table 3. Oxygen saturation level (Sa_{O_2}) in percent measured by ear oximetry and end-tidal carbon dioxide concentration (ET_{CO_2}) in morbidly obese patients with sleep apnea

Parameters	Group 1, AHI 5 < 15 (n = 10)	Group 2, AHI 15 < 30 (n = 10)	Group 3, AHI 30 < 65, (n = 14)	Group 4, AHI ≥ 65 (n = 17)
Awake				
Mean % Sa_{O_2}	92.1 ± 2.8	90.0 ± 5.5	89.7 ± 4.7	82.8 ± 9.3*
ET_{CO_2} (mm Hg)	40.4 ± 5.3	38.8 ± 5.7	39.2 ± 7.2	42.8 ± 9.2
Sleep				
Mean % Sa_{O_2} in NREM	89.1 ± 4.0	85.5 ± 11.8	83.6 ± 8.5	70.8 ± 11.5†
ET_{CO_2} (mm Hg) in NREM	40.6 ± 4.3	39.3 ± 5.9	40.1 ± 7.4	44.3 ± 10.9
Mean % Sa_{O_2} in REM	87.5 ± 6.1	81.4 ± 12.7	72.5 ± 12.9	59.5 ± 12.9‡
ET_{CO_2} (mm Hg) in REM	42.2 ± 5.4	40.3 ± 6.5	43.2 ± 8.8	46.1 ± 12.6
Total time of oxygen desaturation < 90% (minutes)	185.0 ± 145.5	190.8 ± 115.0	225.8 ± 98.0	305.3 ± 81.4§
Total time of oxygen desaturation < 65% (minutes)	0.4 ± 0.9	29.8 ± 81.9	34.3 ± 50.1	134.5 ± 101.5¶
Sa_{O_2} < 90% index	6.2 ± 4.9	9.7 ± 9.9	13.5 ± 11.8	9.5 ± 15.3
Sa_{O_2} < 80% index	4.1 ± 7.4	4.1 ± 7.7	13.9 ± 13.9	15.4 ± 18.4**
Sa_{O_2} < 65% index	0.3 ± 0.7	4.6 ± 11.4	13.3 ± 20.3	41.5 ± 29.5††

Data represent mean ± SD. F values refer to results of one-way ANOVA across four groups.
* $F = 10.81$, $p < 0.0001$, Scheffé test: Group 4 < Groups 1–3; $p < 0.003$.
† $F = 18.93$, $p < 0.0001$, Scheffé test: Group 4 < Groups 1–3; $p < 0.0001$.
‡ $F = 28.29$, $p < 0.0001$, Scheffé test: Group 4 < Groups 1–3; $p < 0.0001$; Group 3 < Group 1, $p < 0.0001$.
§ $F = 7.45$, $p < 0.0002$, Scheffé test: Group 4 > Groups 1,2; $p < 0.004$.
¶ $F = 17.82$, $p < 0.0001$, Scheffé test: Group 4 > Groups 1–3; $p < 0.0001$.
** $F = 4.84$, $p < 0.004$, Scheffé test: Groups 1,2 < Group 4; $p < 0.05$.
†† $F = 22.49$, $p < 0.0001$, Scheffé test: Group 4 > Groups 1–3; $p < 0.0001$.

same investigators, reporting on a small sample of 17 obese patients evaluated using polysomnography before gastric bypass surgery, Charuzi et al. (23) estimated a much lower prevalence of sleep apnea (12% for men and 0.3% for women). Rajala et al. (24) reported a higher OSA prevalence (40.7%) in 27 morbidly obese patients with BMI > 40 kg/m^2. In this sample OSA was more common among men (76.9%) than women (7.1%). Unfortunately, other polysomnographic data, aside from the oxygen desaturation index, were not provided. In a more complete report in which one night of polysomnography was carried out in 250 patients with a diagnosis of obesity (BMI ≥ 27.8 kg/m^2) (25), the frequency of severe sleep apnea in the morbidly obese patients (BMI ≥ 39 kg/m^2) was approximately two times the frequency in the severely obese patients (BMI 35 to 39 kg/m^2) for both men and women (50% vs. 20% for men; 3.5% vs. 2.4% for women).

Unlike these other studies, in our study in patients with BMI ≥ 40, only one woman of 39 did *not* have sleep apnea (AHI < 5). When we applied the definition of an apnea index of 5 to define sleep apnea instead of the combined index of apnea and hypopnea, the prevalence of OSA was reduced somewhat for the women to 59% (23/39) but not for the men, where it remained the same (100%, 13/13). Nonetheless, the overall prevalence of sleep apnea in women remains considerably higher in our data than in those of other investigators. The difference in our data relative to other reports could be due to several factors. For example, previous studies included patients with lower overall BMI and did not control for body weight across different levels of sleep apnea, as we did. Another possibility is that differences in the definition of sleep-disordered breathing may have played a role, i.e., some studies reported only an apnea index rather than the AHI. In our data, however, altering this definition did not impact upon the relatively high prevalence of sleep apnea in the obese women.

In our study, neck size, rather than BMI or waist-to-hip ratio, was closely associated with sleep apnea. These data allow us to extend to the morbidly obese population previous

Table 4. Sleep complaints in morbidly obese patients with sleep apnea

	AHI			
SDQ items	**Group-1 5 < 15**	**Group-2 15 < 30**	**Group-3 30 < 65**	**Group-4 ≥65**
SDQ item 20: I snore in my sleep.	3.4 ± 1.4	3.3 ± 1.1	3.8 ± 1.6	4.5 ± 0.9*
SDQ item 21: I am told I snore loudly and bother others.	2.2 ± 1.6	2.2 ± 1.5	2.9 ± 1.3	4.0 ± 1.2†
SDQ item 56: In the past 6 months, I have fallen asleep accidentally in some of these situations: eating a meal, talking on the phone, talking to someone, riding in a bus or car, watching TV, at a theater, reading a book, or at a lecture.	2.4 ± 1.4	2.3 ± 1.0	2.6 ± 1.4	3.9 ± 1.2‡

Scale is as follows: 1 = never, 2 = rarely, 3 = sometimes, 4 = usually, 5 = always. Data show the mean ± SD. *F* values refer to results of one-way ANOVA across four groups.
* $F = 4.71$, $p < 0.005$, Scheffé test: Group 4 > Groups, 1–3; $p < 0.05$.
† $F = 9.75$, $p < 0.0001$, Scheffé test: Group 4 > Groups 1–3; $p < 0.05$.
‡ $F = 4.25$, $p < 0.02$, Scheffé test: Group 4 > Groups 1–3; $p < 0.05$.

findings suggesting that obesity mediates its effects on sleep-disordered breathing primarily through fat deposition in the neck (5).

Maislin et al. (26), using an index from respondents' self-reports of the frequency of loud snoring, breathing cessation, and snorting and gasping, found that this symptom frequency index was useful in discriminating patients with and without sleep apnea only if the patient was not extremely obese (i.e., BMI < 40 kg/m^2). In individuals who had a BMI > 40 kg/m^2 and had a high risk for sleep apnea (higher than 80%), sleep apnea symptoms were not predictive of who did or did not have sleep apnea. By contrast, we have reported that the complaints of snoring and sleepiness (SDQ items 20, 21, and 56) increased as the level of sleep-related breathing disorder increased in morbidly obese patients.

Table 5. EKG abnormalities during sleep in morbidly obese patients with sleep apnea

Cardiac abnormalities	**AHI < 65 (*n* = 34)***	**AHI ≥ 65 (*n* = 17)†**	**% Total (*n* = 51)**
Without abnormalities	25 (73.5)	10 (58.8)	68.6
Sinus arrhythmia	4 (11.8)	4 (23.5)	15.7
Ventricular arrhythmias	5 (14.7)	6 (35.3)	21.6
Ventricular premature complexes	4 (11.8)	4 (23.5)	15.7
Tachycardia	1 (2.9)	2 (11.8)	5.9
Supraventricular arrhythmias	3 (8.8)	4 (23.5)	13.7
Supraventricular premature complexes	2 (5.9)	4 (17.7)	9.8
Tachycardia	1 (2.9)	1 (5.9)	3.9
Ischemic segment alterations	1 (2.9)	4 (23.5)	9.8
Wandering pacemaker	0 (0.0)	1 (5.9)	2.0

Values are number of patients. Numbers in parentheses, %. For comparison of groups for any arrhythmias, chi-square = 33.75, $df = 5$, $p < 0.0001$.
* In four patients more than one EKG abnormality was present.
† In four patients more than one EKG abnormality was present.

Table 6. Heart rate in morbidly obese patients with sleep apnea

	AHI			
Wake and sleep state	**Group 1, 5 < 15**	**Group 2, 15 < 30**	**Group 3, 30 < 65**	**Group 4, ≥65**
Awake (beats/minute)				
Minimum	53.7 ± 9.2	57.3 ± 11.6	54.6 ± 9.1	55.8 ± 16.2
Maximum	91.2 ± 9.6	94.9 ± 11.6	99.5 ± 9.5	103.5 ± 11.7*
Mean	73.2 ± 9.6	76.8 ± 10.2	79.9 ± 10.3	86.3 ± 11.8†
NREM sleep (beats/minute)				
Minimum	52.1 ± 7.9	53.1 ± 11.1	51.9 ± 9.9	50.2 ± 14.7
Maximum	91.8 ± 12.3	95.3 ± 10.7	103.4 ± 8.6	106.6 ± 15.2‡
Mean	72.6 ± 10.1	75.9 ± 11.1	79.0 ± 11.0	86.0 ± 13.3§
REM sleep (beats/minute)				
Minimum	54.1 ± 8.3	54.5 ± 13.1	53.2 ± 9.6	53.5 ± 16.3
Maximum	90.0 ± 12.5	94.2 ± 9.9	102.3 ± 8.8	105.6 ± 14.2¶
Mean	72.9 ± 10.1	75.6 ± 11.3	81.1 ± 10.5	88.2 ± 13.3**

Data represent mean ± SD. F values refer to results of one-way ANOVA across four groups.
* $F = 5.92$, $p < 0.001$, Scheffé test: Group 4 > Group 1; $p < 0.003$.
† $F = 6.72$, $p < 0.0004$, Scheffé test: Group 4 > Groups 1,2; $p < 0.04$.
‡ $F = 9.81$, $p < 0.0001$, Scheffé test: Group 1 < Groups 3,4; $p < 0.03$, Group 2 < Group 4, $p < 0.02$.
§ $F = 6.35$, $p < 0.0006$, Scheffé test: Group 4 > Groups 1,2; $p < 0.04$.
¶ $F = 9.14$, $p < 0.0001$, Scheffé test: Group 4 > Groups 1,2; $p < 0.02$.
** $F = 8.91$, $p < 0.0001$, Scheffé test: Group 4 > Groups 1,2; $p < 0.004$.

We found that cardiac arrhythmias were prevalent in morbidly obese patients with severe OSA (AHI ≥ 65) in whom oxygen saturation was below 65%, on average, for more than one third of the sleep time. Previously some have questioned whether there is a relationship between sleep apnea and cardiac arrhythmias (16,27). Flemons et al. (16), in a prospective study of consecutive patients referred to a Canadian sleep clinic, demonstrated that sleep apnea does not confer an increased risk for cardiac arrhythmias for most of the 76 apnea patients with AHI > 10 studied. These results were obtained even though those patients with sleep apnea were somewhat older, more obese, and more likely to be hypertensive than were patients without sleep apnea. In that study, the prevalence of any type of cardiac arrhythmias was very low, and it did not differ significantly between patients with sleep apnea and controls without apnea. In contrast with the report of Flemons et al., previous studies have noted a high prevalence of cardiac arrhythmias in patients with sleep apnea, ranging from 48% to 74% (14,15). It has been argued that such discrepant results can be explained in terms of selection bias (inclusion of subjects with more severe sleep apnea and more significant underlying disease) and the absence of a control group (16) or the presence of confounding factors such as age and obesity in the earlier studies (27). In this study we compared subgroups of morbidly obese patients of the same age and body weight but with different severity of sleep apnea and found that the prevalence of cardiac arrhythmias was related to the severity of sleep apnea. Our data provide evidence that sleep apnea, when severe (AHI ≥ 65, with extended oxygen desaturation $SaO_2 < 65\%$), is associated with a greater likelihood of cardiac arrhythmias.

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