The Impact of Obesity on Oxygen Desaturation during Sleep-disordered Breathing

Paul E. Peppard¹, Neil R. Ward², and Mary J. Morrell²

¹Department of Population Health Sciences, University of Wisconsin-Madison, Madison, Wisconsin; and ²Clinical and Academic Unit of Sleep and Breathing, National Heart and Lung Institute, Imperial College London, Royal Brompton Hospital, London, United Kingdom

Rationale: Obesity increases the risk and severity of sleep-disordered breathing. The degree to which excess body weight contributes to blood oxygen desaturation during hypopneic and apneic events has not been comprehensively characterized.

Objectives: To quantify the association between excess body weight and oxygen desaturation during sleep-disordered breathing.

Methods: A total of 750 adult participants in the Wisconsin Sleep Cohort Study were assessed for body mass index (BMI) (kg/m^2) and sleep-disordered breathing. The amount of SaO_2 duration, and other characteristics of 37,473 observed breathing events were measured during polysomnography studies. A mixed-effects linear regression model estimated the association of blood oxygen desaturation with participant-level characteristics, including BMI, gender, and age, and event-level characteristics, including baseline SaO_2 , change in V_T , event duration, sleep state, and body position.

Measurements and Main Results: BMI was positively associated with oxygen desaturation severity independent of age, gender, sleeping position, baseline SaO_2 , and event duration. BMI interacted with sleep state such that BMI predicted greater desaturation in rapid eye movement (REM) sleep than in non-REM sleep. Each increment of $10 \text{ kg}/\text{m}^2$ BMI predicted a 1.0% (SE, 0.2%) greater mean blood oxygen desaturation for persons in REM sleep experiencing hypopnea events associated with 80% V_T reductions.

Conclusions: Excess body weight is an important predictor of the severity of blood oxygen desaturation during apnea and hypopnea events, potentially exacerbating the impact of sleep-disordered breathing in obese patients.

Keywords: sleep apnea; overweight; hypoxia

Obesity is one of the most important risk factors for sleep-disordered breathing (SDB) (1). Epidemiological studies have shown that the prevalence of SDB is strongly associated with excess body weight (2) and that weight gain independently predicts the development of SDB (3). Moreover, most (4), but not all (5), interventional studies have shown that weight loss is effective in reducing apneas and hypopneas. As the prevalence of obesity increases across industrialized nations, it is predicted that obesity will cause a corresponding increase in the prevalence of SDB (1, 6).

The frequency of apneas and hypopneas increases with obesity (3, 7). The frequency of breathing events is an important predictor of the consequences of SDB, with a longitudinal dose-response relationship between the apnea-hypopnea index (AHI)

AT A GLANCE COMMENTARY

Scientific Knowledge on the Subject

Obesity increases the risk of sleep-disordered breathing, but the extent to which excess body weight influences the amount of blood oxygen desaturation during apneic and hypopneic events has not been characterized.

What this Study Adds to the Field

We show that body mass index is a significant independent predictor of the amount of oxygen desaturation during apneas and hypopneas. The impact of sleep disordered breathing may be greater in obesity due to exacerbation of intermittent hypoxia.

and the prevalence of cardiovascular morbidity (8). The severity of oxygen desaturation during obstructive breathing events and the cumulative burden of nocturnal hypoxia are important factors in this relationship; they also predict cardiovascular morbidity in patients with SDB, independent of the frequency of breathing events (9, 10). Because excess body weight is known to predispose to more severe oxygen desaturation during voluntary apneas (11), obese patients with SDB may be disadvantaged by greater oxygen desaturation during apneas and hypopneas and by increased event frequency.

The primary aim of the present study was to measure the impact of obesity on oxygen desaturation in a large population of 30- to 62-year-old, community-dwelling men and women studied as part of the Wisconsin Sleep Cohort Study. Multiple regression analysis of over 37,000 breathing events was used to develop a predictive model to test the hypothesis that obesity was associated with greater oxygen desaturation during apneas and hypopneas independent of confounding variables and covariates.

METHODS

Participants

Participants were 750 adults from the Wisconsin Sleep Cohort Study (3) (described in the online supplement), an ongoing epidemiologic investigation of the natural history of SDB. The study was approved by the University of Wisconsin Health Sciences Institutional Review Board. Participants gave written informed consent.

Measurements

Body habitus, including height (m); weight (kg); and waist, neck, and hip circumference (cm), were measured using standard procedures (12). Body mass index (BMI) was calculated (kg/m^2). Data on medical history, medication use, smoking habits, and alcohol consumption were obtained by questionnaires. Participants underwent attended polysomnography (Polygraph model 78; Grass Instruments, Quincy, MA). Measurements (described in detail in the online supplement) included electroencephalography, electrooculography, chin electromyography, SaO_2 , oral and nasal airflow, thoracic and abdominal excursions, and body position.

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Correspondence and requests for reprints should be addressed to Paul E. Peppard, Ph.D., Department of Population Health Sciences, University of Wisconsin-Madison, WARF Building #685, 610 Walnut St., Madison, WI 53726. E-mail: ppeppard@wisc.edu

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Sleep scoring was performed using conventional criteria (13). Respiratory signals were analyzed with an automated Sleep Analysis Program (SAP). This software program, developed for the Wisconsin Sleep Cohort Study and designed for the detection and characterization of hypopnea and apnea events of SDB, was previously described and validated (14) and is described in the online supplement. All breathing events that produced a change in SaO_2 of 2% or greater were analyzed. The following parameters were recorded for each event: SaO_2 at the start of the event (baseline SaO_2), end-event SaO_2 , ΔSaO_2 (end-event minus initial SaO_2), relative change in Vr during an event (ΔVr), event duration (scored from the Vr signal), body position, sleep state (rapid eye movement [REM] or non-REM [NREM] sleep), and time of event (time since first event). Hypopneas were defined as a reduction in Vr of 20% or greater, and apneas were defined as cessation of breathing for at least 10 seconds. The AHI was calculated as the mean number of apneas and hypopneas associated with ΔSaO_2 2% or greater per hour of sleep ($\text{AHI}_{2\%}$). Additional models that included only events associated with ΔSaO_2 3% or greater were also examined.

Statistical Analysis

Mixed-effects linear regression models were used to examine the effect of excess body weight on ΔSaO_2 during breathing events after accounting for covariates. The primary predictor variable was BMI. Alternative body habitus measures, such as neck and waist circumferences, were examined. Additional variables examined as covariates or interacting variables included: age, gender, ΔVr , time of event, sleep state, body position in which event occurred, baseline SaO_2 , event duration, FEV_1 , FVC, FEV_1 % predicted, cigarette smoking, and alcohol consumption.

Using SAS PROC MIXED (SAS Institute Inc, Cary, NC), ΔSaO_2 was regressed on the predictor variables, which were modeled as random (intercept and baseline SaO_2) or fixed (all other predictors) effects. Interactions among predictor variables were also evaluated. Statistically significant (two-sided P value < 0.05) covariates, interactions, and quadratic terms were retained in presented models.

The primary model examined breathing events that produced 2% or greater ΔSaO_2 . Because most subjects had multiple breathing events, a within-subject correlation structure was fitted in the models. The best-fitting correlation structure (assessed by likelihood ratio tests and the Bayesian Information Criterion) was a "spatial" covariance matrix (spatial exponential anisotropic) with time of event (unequally spaced, within-subject) as the single spatial dimension (15).

RESULTS

A total of 1463 participants had technically adequate baseline polysomnography sleep studies. Of these subjects, 675 were not suitable for SAP analyses because the sleep studies were performed after the Wisconsin Sleep Cohort migrated to an alternative software platform that was not compatible with SAP analysis. Of the 788 remaining SAP-analyzable studies, 17 were excluded due to participant-reported, physician-diagnosed heart attack, heart failure, stroke, or emphysema, and 21 were excluded due to the use of sleep apnea treatment (continuous positive airway pressure) at the time of sleep study.

Demographic and anthropometric details of the 750 participants are presented in Table 1 and in Tables E1 through E5 in the online supplement. The mean age of the participants was 45 years, and 56% were male. Mean BMI was 29 (SD, 6) kg/m^2 , and mean $\text{AHI}_{2\%}$ was 9.5 (SD, 12.0).

TABLE 1. DESCRIPTIVE CHARACTERISTICS OF 750 PARTICIPANTS INCLUDED IN SLEEP ANALYSIS PROGRAM ANALYSIS

Variable	Mean (SD)	Range
Age, yr	45 (7)	30–62
Body mass index, kg/m^2	29 (6)	18–62
Neck circumference, cm	38 (4)	27–53
Waist:hip ratio (men)	0.95 (0.06)	0.79–1.24
Waist:hip ratio (women)	0.82 (0.07)	0.34–1.08
$\text{AHI}_{2\%}$, SAP-scored, events/h	9.5 (12.0)	0.3–91
$\text{AHI}_{4\%}$, SAP-scored, events/h	4.5 (8.7)	0.1–79
FEV_1 , % predicted	107 (16)	43–153
FEV_1/FVC	0.82 (0.07)	0.36–1.0
Epworth Sleepiness Scale	8.4 (4.3)	0–22
Current smoker, n (%)	148 (20%)	

Definition of abbreviation: AHI = apnea-hypopnea index.

The 750 participants had a mean sleep time of 6.0 (SD, 1.1) hours and a total of 37,473 breathing events. Table 2 shows the number of breathing events by BMI categories. Obese participants (BMI ≥ 30 kg/m^2) comprised 40% of the cohort but contributed 62% of breathing events.

Table 3 shows descriptive data for all breathing events. The mean oxygen desaturation (ΔSaO_2) was 4.8% (SD, 3.1%). Due to the right-skewed distribution of ΔSaO_2 , the majority of events were associated with a ΔSaO_2 of less than 4% (median ΔSaO_2 , 3.7%; range, 2–24%). Most breathing events were hypopneas (92%), and a disproportionate number of breathing events occurred in REM sleep: Mean REM sleep time was 18% of total sleep time but encompassed 26% of all breathing events.

Regression Analysis

The regression model (Table 4) fitted a significant linear relationship between BMI and ΔSaO_2 independent of age, gender, sleeping position, baseline SaO_2 , and event duration. For example, the model presented in Table 4 predicts that 50-year-old nonsmoking men with a BMI of 30 kg/m^2 and a baseline SaO_2 of 100% who experience a 30-second hypopnea with an 80% ΔVr while on their side in NREM sleep would be expected to have a mean ΔSaO_2 of 5.9% (95% confidence interval, 5.5–6.2%). The primary variable of interest in this study is BMI, with a regression coefficient of 0.055 (SE, 0.014). This indicates that for each increment in BMI of 1 kg/m^2 , a mean 0.055% greater ΔSaO_2 is expected. Thus, in the example, if BMI was 40 kg/m^2 rather than 30 kg/m^2 , with all other variables unchanged, we would predict an average 0.55% greater desaturation (i.e., total ΔSaO_2 of 6.4%). The model also showed statistically significant interactions between BMI and sleep state as well as BMI and ΔVr , indicating that: (1) the effect of BMI on ΔSaO_2 was considerably greater during REM sleep, and (2) the impact of ΔVr on ΔSaO_2 increased with increasing BMI (Table 4). Thus, for example, for hypopneas occurring during REM sleep, the coefficient would be 0.097 (= 0.055 + 0.042), and we predict an additional desaturation of approximately 1.0% for each 10 kg/m^2 increment in BMI (e.g., a total estimated mean ΔSaO_2 of 6.9% for the 40 kg/m^2 vs. 30 kg/m^2 example above). No other examined

TABLE 2. DISTRIBUTION OF PARTICIPANTS (N = 750) AND BREATHING EVENTS (N = 37,473) BY BODY MASS INDEX (BMI) CATEGORY

	BMI Category				
	<25 kg/m^2	25–29.9 kg/m^2	30–34.9 kg/m^2	35–39.9 kg/m^2	≥ 40 kg/m^2
Participants, n (%)	199 (27)	257 (34)	184 (25)	57 (8)	53 (7)
Breathing events, n (%)	3,889 (10)	10,380 (28)	13,210 (35)	5,013 (13)	4,981 (13)

TABLE 3. DESCRIPTIVE DATA FOR SLEEP ANALYSIS PROGRAM-ANALYZED BREATHING EVENTS (N = 37,473)

Variable	Mean (SD)	Median	Range
ΔSaO_2 , %	4.8 (3.1)	3.7	2.0–24.0
SaO_2 at start of event, %	95 (3)	95	79–100
SaO_2 at end of event, %	90 (4)	91	75–98
ΔVt , %	74 (21)	77	20–100
Event duration, s	29 (13)	26	10–90

variables (e.g., age and gender) were found to significantly interact with BMI. The interaction of sleep state and BMI from the regression model is depicted in Figure 1A for men and in Figure 1B for women. The additional model that included only events associated with $\Delta\text{SaO}_2 \geq 3\%$ yielded very similar results to those presented in Table 4. In the $\Delta\text{SaO}_2 \geq 3\%$ model, the analogous coefficient for BMI in Table 4 (i.e., 0.055; SE, 0.014) was 0.052 (SE, 0.015).

The regression model also revealed the importance of other variables (in addition to BMI) in determining the ΔSaO_2 (Table 4). The supine position was associated with increased desaturation, predicting an estimated 0.54% greater ΔSaO_2 compared with that expected during similar events occurring in the prone or lateral position (Figure 2). As expected, longer event duration and greater ΔVt produced greater ΔSaO_2 . After accounting for event-level characteristics, increasing age and smoking also independently predicted greater oxygen desaturation. Men were estimated to have an average 0.26% (SE, 0.08%) greater ΔSaO_2 than women. That is, if a hypopnea with a given set of characteristics (duration, ΔVt , etc.) was expected to produce a 5.00% ΔSaO_2 in a man, then a similar hypopnea in a woman would be expected to produce a 4.74% ΔSaO_2 .

Eight percent of all events were identified by SAP as apneas. Due to the relatively few apnea events, we had insufficient power to perform apnea-only modeling. However, in the re-

gression analysis of all respiratory events (Table 4), an indicator variable for apnea was significant (coefficient, 0.25; SE, 0.07), indicating that apneas produced greater oxygen desaturation than might be expected from extrapolating hypopnea events to $\Delta\text{Vt} = 100\%$.

In addition to BMI, we examined other measures of body habitus in relation to oxygen desaturation, including neck and waist girth and the waist:hip ratio. None of these measures was a substantially better predictor of ΔSaO_2 than BMI. However, we have included regression model results in the online supplement (Table E7) that describe the estimated sex-specific associations of BMI, neck girth, waist girth, and waist:hip ratio with ΔSaO_2 . We also examined the addition of spirometric measures of lung function, including FEV₁% predicted and FEV₁/FVC, to the presented models. In this population of healthy working adults, these variables had no appreciable effect on the other predictor variable coefficients, nor were they statistically significant predictors of ΔSaO_2 independent of the other variables in the model.

DISCUSSION

The findings of this study show that BMI is an important predictor of SaO_2 during SDB. The regression model estimated a linear relationship between BMI and ΔSaO_2 independent of age, gender, sleeping position, baseline SaO_2 , and event duration. The model also showed two interactions. First, the impact of BMI is significantly modified by sleep state; thus, ΔSaO_2 during apnea or hypopnea is predicted to be greater in REM compared with NREM sleep but is especially so in persons with higher BMI. Second, during hypopnea, the (expected) positive association between ΔVt and ΔSaO_2 is most marked in obese individuals.

Our model also showed that additional variables were significant predictors of the severity of the oxygen desaturation. Supine sleeping position was an independent predictor of greater

TABLE 4. ΔSaO_2 PREDICTION MODEL AND THE IMPACT OF CHANGING INDIVIDUAL VARIABLES ON AN EVENT WITH A PREDICTED ΔSaO_2 OF 5.9% (THE PREDICTED ΔSaO_2 FOR AN EVENT WITH THE CHARACTERISTICS INDICATED IN THE "BASELINE VALUE" COLUMN)

Variable*	Coefficient† (SE)	Baseline Value	Comparison Value	Expected Difference in ΔSaO_2 (%)	Expected new ΔSaO_2 (%)
BMI‡	0.055 (0.014)	30 kg/m ²	40 kg/m ²	+0.55	6.4
Sleep state‡	0.39 (0.05)	NREM sleep	REM sleep	+0.39	6.3
ΔVt ‡	0.030 (0.002)				
(ΔVt) ²	0.00054 (0.00005)	80%	90%	+0.35	6.2
Baseline SaO_2	0.52 (0.04)				
(Baseline SaO_2) ²	0.025 (0.003)	100%	98%§	−0.93	4.9
Event type	0.25 (0.07)	Hypopnea	Apnea	+0.25	6.1
Body position	0.54 (0.07)	Lateral	Back	+0.54	6.4
Event duration	0.025 (0.002)				
(Event duration) ²	0.00016 (0.00005)	30 s	60 s	+0.88	6.8
Time of event	−0.022 (0.011)	1st Event	1 h After 1st event	−0.02	5.9
Gender	−0.26 (0.08)	Male	Female	−0.26	5.6
Age	0.028 (0.006)	50 yr	60 yr	+0.28	6.2
Smoking	0.20 (0.07)	Nonsmoking	1 Pack/d	+0.20	6.1

* To aid interpretation of the model intercept (5.88; SE, 0.17), select variables (BMI, ΔVt , baseline SaO_2 , event duration, and age) were "centered" in fitting the model by subtracting constants from their observed values as indicated by the "Starting value" column (i.e., BMI was centered at 30 kg/m² ["BMI" = BMI − 30]; ΔVt at 80% [" ΔVt " = ΔVt − 80%]; etc.).

† All coefficients (including quadratic and interaction terms) are significantly different from 0 with a $P < 0.001$, except for smoking ($P = 0.008$) and time of event ($P = 0.04$).

‡ There were also significant interactions of BMI × sleep state (interaction coefficient, 0.042; SE, 0.009) and BMI × ΔVt (interaction coefficient, 0.00082; SE, 0.00018), indicating that the association of BMI and ΔSaO_2 is stronger in REM compared with NREM sleep and that the effect of ΔVt on ΔSaO_2 increases with increasing BMI.

§ The fitted quadratic relationship between baseline SaO_2 and ΔSaO_2 suggested that baseline $\text{SaO}_2 < 80\%$ was strongly associated with greater ΔSaO_2 reductions but also that there are modest decreased expected reductions in ΔSaO_2 with increasing baseline SaO_2 for baseline $\text{SaO}_2 > 80\%$.

|| Comparing a hypopnea with $\Delta\text{Vt} = 80\%$ to an apnea with $\Delta\text{Vt} = 100\%$ would yield an expected 1.1% greater ΔSaO_2 (i.e., $5.9 + 1.1 = 7.0\%$ "expected new ΔSaO_2 ").

Figure 1. Predicted ΔSaO_2 for a hypopnea (100% baseline SaO_2 , 30 seconds in duration, with an 80% ΔV_T in supine 50-year-old subjects) by body mass index and sleep state in (A) men and (B) women. Error bars indicate upper (REM) and lower (NREM) 95% confidence limit for predicted mean ΔSaO_2 .

oxygen desaturation, compared with the lateral or prone position. The SaO_2 at the start of the breathing event (baseline SaO_2), and the reduction in tidal volume occurring during hypopnea, were also predictors of oxygen desaturation.

Previous studies evaluating the effect of obesity on oxygen desaturation during SDB have not systematically examined or accounted for potential interacting or confounding factors (16, 17). In the present study, we found that body habitus predicts oxygen desaturation severity during apneic and hypopneic events independent of individual- and event-level characteristics. Sleeping position (18), sleep state (19), duration of breathing event (20), gender (21), and baseline oxygen saturation (22) have been previously reported to influence the severity of oxygen desaturation during an apneic/hypopneic event. Our model has also shown that these variables are significant independent predictors of the severity of oxygen desaturation; thus, for instance, supine sleeping position predicted greater oxygen desaturation during breathing events, compared with the lateral or prone position. Moreover, our model demonstrates that male gender, increasing age, and smoking are significant independent predictors of ΔSaO_2 .

Mechanisms by which Obesity Increases ΔSaO_2 during Apnea and Hypopnea

The rate of desaturation during apnea is influenced by many physiological variables, including alveolar volume (23). The reservoir of oxygen in the lung is proportional to the alveolar volume, as shown in healthy individuals who experience significantly greater ΔSaO_2 after voluntary apnea at residual volume compared with voluntary apnea at total lung capacity (24). The

Figure 2. Predicted ΔSaO_2 for a hypopnea (100% baseline SaO_2 , 30 seconds in duration, with an 80% ΔV_T during REM sleep, in supine 50-year-old subjects) by body mass index and sleeping position in women. Error bars indicate upper (supine) and lower (lateral) 95% confidence limit for predicted mean ΔSaO_2 .

effect of excess body weight on lung volume is the principal mechanism by which BMI influences ΔSaO_2 (23), an effect likely accentuated in the presence of greater whole-body oxygen demand (25). Excess body weight causes significant reduction in all lung volumes, with the greatest reduction in FRC and expiratory reserve volume (ERV) even in mildly obese and overweight individuals (26–28). In morbid obesity, FRC may approach residual volume (26). In SDB, ERV has been shown to be strongly correlated with the severity of apnea-induced desaturation (29) and mean nocturnal oxygen saturation (17, 30). Weight loss has been shown to result in improvements in overnight oxygen saturation in parallel with increase in the FRC and ERV (31).

In addition to reducing the reserve of oxygen in the lung, excess body weight affects the ventilation-perfusion ratio, another important determinant of ΔSaO_2 (23). In obesity, the combined effect of a reduction in FRC and an increase in closing volume (32) results in a greater propensity for small airway closure, causing ventilation-perfusion mismatching and pulmonary shunting, thereby exacerbating oxygen desaturation (33, 34).

The effect of obesity on lung volume and rate of oxygen consumption may also explain the significant interaction between BMI and sleep state predicted by our model. During REM sleep, further increase in upper airway resistance (35) may increase the work of breathing, while reduction in lung volumes and ventilation-perfusion mismatching (36) increase the rate of oxygen desaturation. Moreover, chest wall excursion is reduced during the atonia of REM sleep, and there is a greater dependence upon the diaphragmatic contribution to ventilation (37, 38), which may be impinged by abdominal adiposity.

Reduction in lung volume could also explain the greater oxygen desaturation observed in the supine sleeping position. Compared with the sitting and lateral decubitus positions, supine posture causes a significant reduction in FRC and ERV in healthy individuals (39, 40).

Our primary measure of body habitus, the BMI, does not characterize the peripheral and central distribution of excess body fat (41). Therefore, we examined alternative measures of body habitus. Waist girth might be expected to show similar, if not stronger, associations with ΔSaO_2 as BMI because central adiposity may have greater impact on lung function due to diaphragmatic displacement and thoracic wall impingement by visceral and chest wall fat. Although BMI is highly correlated with end expiratory lung volume (42), a recent study has reported that

waist girth is strongly associated with impaired lung function independent of BMI (43). In our sample, the sex- and age-adjusted correlation of BMI and waist girth was 0.90, and only BMI was a significant predictor of ΔSaO_2 when both measures were included in our model simultaneously. Thus, BMI and waist girth might be viewed as essentially interchangeable in our data. We chose to focus our results on BMI because this is the most commonly used metric of excess body weight.

Strengths and Limitations

A strength of our study is the use of data from a large community-based sample. Over 37,000 breathing events were detected in participants with a wide spectrum of body habitus and severity of SDB, allowing examination of the independent impact of individual variables and interaction between variables. Previous studies investigating the impact of obesity on ΔSaO_2 have only evaluated selected populations of sleep clinic patients (16) or patients assessed for bariatric surgery (17). Because there was little racial diversity in our sample (94% white; see Table E4), we were not able to examine whether associations between obesity and ΔSaO_2 varied by race.

Two limitations need to be considered when interpreting the findings of our study. First, detailed pulmonary function testing was not available in our study population. Excess body weight predominantly results in a restrictive lung defect with reduction in FRC and ERV (26), and it is possible that inclusion of these variables in our predictive model may have altered the measured impact of BMI on ΔSaO_2 . The lack of lung volume measurements also limits our physiological interpretation of the model; although we speculate that the impact of excess body weight on lung volume is the key mechanism by which obesity causes greater ΔSaO_2 , we cannot rule out other factors. For instance, right to left cardiac shunting causes increased oxygen desaturation (44), and recent reports have identified a higher prevalence of patent foramen ovale in patients with sleep apnea compared with persons without sleep apnea (45, 46). Although we cannot exclude unidentified patent foramen ovale causing greater ΔSaO_2 in our study population, we are not aware of any data to suggest that patent foramen ovale becomes more prevalent as BMI increases. Second, although the SAP uses calibrated respiratory inductance plethysmography for measurement of respiratory events, the calibration may become unreliable after a change in sleeping position (47). However, because the SAP measures relative differences in $\dot{V}_T$ based on the preceding breaths, this limitation is unlikely to have strongly influenced our results.

The present study concentrated on oxygen desaturation during discrete apnea and hypopnea breathing events, but obese individuals are also at risk of nocturnal hypoventilation and hypercapnia. This issue could not be evaluated in our model but may also be important in view of the association of the obesity hypoventilation syndrome with increased mortality and morbidity (48).

Clinical Implications

Our predictive model provides clinically important information to show that obese individuals are exposed to a greater burden of oxygen desaturation during sleep-disordered breathing. Although this analysis does not confirm the long-term adverse consequences of greater oxygen desaturation in obese individuals, the detrimental effects of intermittent hypoxia are well described (49), and there is evidence to show that the severity of intermittent hypoxia is important in the pathophysiologic consequences of SDB. For example, hypopneas associated with a 4% or greater ΔSaO_2 are a better predictor of prevalent cardiovascular disease compared with events associated with a 3% or less ΔSaO_2 (10), and, in a recent, novel experimental model of intermittent hypoxia in humans, healthy

adults exposed to intermittent hypoxia during sleep over a 2-week period developed a significant increase in waking mean SaO_2 of 5 mm Hg (50).

Summary

The body mass index is an important predictor of the severity of oxygen desaturation during apneic and hypopneic events of sleep-disordered breathing, independent of age, gender, sleeping position, smoking history, baseline SaO_2 , and event duration. The association of BMI with oxygen desaturation is particularly strong in REM sleep. Excess body weight has been widely demonstrated to be strongly associated with a greater risk of having SDB and with an increased number of breathing events among persons with SDB. For the first time, we have comprehensively quantified the additional burden that excess body weight has on the severity of individual breathing events. These findings contribute to a fuller understanding of the impact of excess body weight on SDB.

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