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Obesity Is Associated With a Lower Resting Oxygen Saturation in the Ambulatory Elderly: Results From the Cardiovascular Health Study

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Abstract

BACKGROUND—The contribution of obesity to hypoxemia has not been reported in a community-based study. Our hypothesis was that increasing obesity would be independently associated with lower S_{pO_2} in an ambulatory elderly population.

METHODS—The Cardiovascular Health Study ascertained resting S_{pO_2} in 2,252 subjects over age 64. We used multiple linear regression to estimate the association of body mass index (BMI) with S_{pO_2} and to adjust for potentially confounding factors. Covariates including age, sex, race, smoking, airway obstruction (based on spirometry), self reported diagnosis of emphysema, asthma, heart failure, and left ventricular function (by echocardiography) were evaluated.

RESULTS—Among 2,252 subjects the mean and median S_{pO_2} were 97.6% and 98.0% respectively; 5% of subjects had S_{pO_2} values below 95%. BMI was negatively correlated with S_{pO_2} (Spearman R = −0.27, $P < .001$). The mean difference in S_{pO_2} between the lowest and highest BMI categories (< 25 kg/m^2 and ≥ 35 kg/m^2) was 1.33% (95% CI 0.89–1.78%). In multivariable linear regression analysis, S_{pO_2} was significantly inversely associated with BMI (1.4% per 10 units of BMI, 95% CI 1.2–1.6, for whites/others, and 0.87% per 10 units of BMI, 95% CI 0.47–1.27, for African Americans).

CONCLUSIONS—We found a narrow distribution of S_{pO_2} values in a community-based sample of ambulatory elderly. Obesity was a strong independent contributor to a low S_{pO_2}, with effects comparable to or greater than other factors clinically associated with lower S_{pO_2}.

Keywords

pulse oximetry; oxygen; obesity; body mass index; waist circumference; hypoxemia; pulmonary function test

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Introduction

The contribution of obesity to the resting level of S_{pO_2} has not been reported in a population-based study or in the elderly. Such information would be useful for clinicians to gauge whether decreased S_{pO_2} readings are in a range potentially explained by increased weight, and to researchers assessing the health consequences of obesity. If obesity is associated with even small changes in oxygen saturation, the exposure to lower oxygen levels may have important clinical consequences, particularly in physiologic processes like respiratory drive, which are dependent on P_{O_2} in the blood, or in situations of marginally sufficient oxygen delivery to important organs like the brain, as can be seen in the elderly.[1,2]

The Cardiovascular Health Study ascertained resting S_{pO_2} on 2,252 subjects. In this report we test our a priori hypothesis that increasing levels of obesity are associated with a lower S_{pO_2} in a population-based sample of ambulatory elderly.[3,4]

Methods

Subjects

The Cardiovascular Health Study is a population-based study of risk factors for cardiovascular disease in older adults (ages 65 and above at their baseline exam).[5] Subjects were recruited from random samples of Medicare eligibility lists in Sacramento County, California; Washington County, Maryland; Forsyth County, North Carolina; and Pittsburgh, Pennsylvania; and from age-eligible subjects in the same household. Potential subjects were excluded if they were institutionalized, wheelchair-bound in the home, or under active treatment for cancer, including hospice care, radiation, or chemotherapy. An original cohort of 5,201 subjects was enrolled in 1989–1990, and a second cohort of 687, predominately African Americans, was enrolled in 1992–1993.[3] Subjects provided written informed consent, and study methods were approved by the institutional review board at each participating center. Details of the design and recruitment have been published.[3–5] The coordinating center is based in Seattle, Washington, and has approval from the University of Washington institutional review board (37714).

QUICK LOOK

Current knowledge

Obesity causes restrictive changes to lung function, with decreased chest wall compliance and reduced lung volumes and capacities. The impact of obesity on oxygenation is less well described.

What this paper contributes to our knowledge

In a population of elderly adults, body mass index was negatively correlated with S_{pO_2}. Obesity was a strong independent contributor to a low S_{pO_2}, with effects comparable to or greater than other clinical factors commonly associated with lower S_{pO_2}. Obesity affects lung function and diminishes oxygen exchange.

A total of 3,326 Cardiovascular Health Study subjects, out of 4,413 still living and participating in the Cardiovascular Health Study, attended the 1996–1997 clinic visit, during which a standard 6-min walk test with oximetry was offered.[6] Approximately one third of the subjects were excluded or chose not to perform the walk test, leaving 2,273 subjects with oximetry results. Exclusions from testing included: use of an ambulatory aid such as wheelchair, crutches, walker or cane; heart attack, angioplasty, or heart surgery within past 3 months; resting heart rate < 50 or > 110 beats/min; systolic blood pressure > 200 m Hg or diastolic blood pressure > 110 m Hg; previous echocardiographic evidence of severe aortic stenosis; new or worsening symptoms of chest pain, shortening of breath, or fainting in the past 8 weeks; electrocardiogram with ventricular fibrillation, acute injury, acute ischemia, acute myocardial infarction, or any other acute cardiac condition; subject refusal or technician's discretion or resting oxygen saturation < 90% (n = 1). The 2,252 subjects included in this report consist of subjects with available pre-exercise S_{pO_2} results in addition to body mass index (BMI) and covariate measures.

S_{pO_2}

While the subject was seated, a semi-disposable oximeter sensor (D-25, Nellcor/Puritan Bennett, Pleasanton, California) was applied to the index finger of the non-dominant hand, after ensuring that nail polish was not present, and was then attached to the oximeter (N-3000, Nellcor/Puritan Bennett, Pleasanton, California). After verification of good perfusion, the values for S_{pO_2} and pulse rate were recorded.

Anthropomorphic Measurements

Anthropometric measurements were performed by trained personnel using standardized protocols. Subjects wore standard examination suits and no shoes. Height was measured with a stadiometer and weight with a balance scale, using a standard protocol during the 1996–1997 study visits. BMI was calculated as weight (kg)/height (m)^2.[2] Waist circumference was measured at the level of the umbilicus.

Covariates

Sex, race, smoking history, and medical history were self-reported. Subjects were allowed the following choices for race: white (n = 1,909), black (African American) (n = 332), American Indian/Alaska native (n = 3), Asian/Pacific islander (n = 2), and other (n = 6). Race for the purposes of this analyses was classified as African American (n = 332) and white/other (n = 2,010). Spirometry was measured following American Thoracic Society guidelines, using a water-sealed spirometer at the same visit that oximetry was performed.[7] Airway obstruction was defined as a low FEV_1/FVC and a low FEV_1, defined as below the lowest 5th percentile, based on reference equations developed from a healthy sample of Cardiovascular Health Study subjects.[8] Left ventricular ejection fraction was assessed by echocardiography on an exam 2 years prior, with impaired defined as < 45%.[9]

Statistical Analyses

Categories of BMI were defined using standard clinical definitions: normal < 25 kg/m^2, overweight 25–29.99 kg/m^2, obesity class I (30–34.99), and obesity class II or greater (> 35 kg/m^2). The covariates are described according to these categories. Categorical variables were compared using chi-square test, and continuous variable using analysis of variance. Initial correlations of S_{pO_2} with BMI, waist size, and other continuous covariates were assessed using the Spearman coefficient, due to the skewed nature of distributions of some variables. For categorical covariates, mean S_{pO_2} between categories was compared using analysis of variance, *t* tests, and the Spearman coefficient of ordinal categories with S_{pO_2} was assessed.

Exploratory analyses and graphs of BMI and S_{pO_2}, including spline models, established that the relationship was reasonably linear. Therefore, linear regression models were used to estimate independent associations of BMI or waist circumference with resting S_{pO_2}. The models were adjusted for covariates chosen based upon clinical knowledge, associations with S_{pO_2}, and effects on the magnitude of the association with obesity. Variables that were not possible confounders and were not significant were dropped. Multiplicative interactions of BMI (or waist size) with race and sex were evaluated. As a result of significant interactions for BMI and obstruction with race we analyzed data separately by race. P values were based upon 2-tailed tests and statistical significance was defined as $P < .05$. Analyses were performed with statistics software (SPSS, SPSS, Chicago, Illinois, and Stata, StataCorp, College Station, Texas.).

Results

The sample included 2,252 ambulatory subjects in the Cardiovascular Health Study. Subjects with S_{pO_2} measures were younger, were less likely to be female and African American, had less cardiopulmonary disease by self-report and testing, and had smoked less than those who did not attempt the test or those who did not attend the clinic visit (Table 1). Although subjects with S_{pO_2} measures had lower mean waist circumference than those without measures, mean BMI was similar among the 2 groups.

The median BMI was 26.3 kg/m^2 (IQR 23.8–29.1). Subjects were distributed as follows across BMI categories: normal or underweight (35%), overweight (45%), obesity class I (15%), and obesity class II or greater (5%). Higher BMI categories were related to younger age, female sex, African American race, less current smoking, greater waist circumference, and higher FEV_1/FVC (Table 2).

The mean and median S_{pO_2} were 97.6 ± 1.74% and 98.0% (IQR 97–99%), respectively. The distribution of values was skewed to higher values, with a range from 81% to 100% (Fig. 1). Only 2 subjects had S_{pO_2} values below 90%, one of which (81%) was removed from further analyses as an outlier. Five percent of subjects had S_{pO_2} values below 95%.

BMI as a continuous variable was negatively correlated with S_{pO_2} (Spearman R = −0.27, $P < .001$). In unadjusted analyses, mean S_{pO_2} differed significantly by category of BMI, with a linear relationship between mean S_{pO_2} and BMI category (Table 3). Mean S_{pO_2} also differed significantly by race, sex, airway obstruction, obesity, smoking status, emphysema diagnosis, and left ventricular function. In addition, S_{pO_2} was also significantly associated with the log of pack-years (r = −0.14, $P < .001$), deteriorating airway obstruction, as measured by FEV_1/FVC (r = 0.12, $P < .001$), and age (r = −0.04, $P = .045$).

In multivariable linear regression analyses the negative association between S_{pO_2} and BMI was significant, and there was an interaction between BMI and race ($P = .05$). For white/other there was a −0.14 difference in S_{pO_2} with each unit increase in BMI (Table 4). For African Americans the difference was smaller (−0.09) although still highly statistically significant (see Table 4). The unadjusted regression line between S_{pO_2} and BMI by race is provided in Figure 2. There was also an interaction between airway obstruction and race (−2.35% decline in S_{pO_2} in African Americans, vs −0.98% for white/other in those with obstruction, compared to those without obstruction). Other covariates significantly related to S_{pO_2} included age and current smoking. Higher waist size was also an independent predictor of a lower S_{pO_2} ($P < .001$), but there were no significant interactions between race and obesity when using waist size as the index of obesity in the model (Table 5).

Restricting regression analyses to subjects with stable weight (within 4.55 kg, *n* = 1,752) in the year prior to the oximetry measurement did not appreciably alter the results.

Discussion

The average resting S_{pO_2} in a community-based population of older people who agreed to participate in a timed walk was approximately 98%, and values were rarely below 95%. To our knowledge, there is no comparable information available from a large community-based population.

Obesity was a major determinant of hypoxemia in this community-based sample. This suggests that the effects of obesity on gas exchange in adults may be under-appreciated relative to other clinical entities that are widely perceived as being associated with hypoxemia (ie, smoking, heart failure, obstructive lung disease), but had a similar magnitude of association with S_{pO_2} levels. To our knowledge, only one other study has reported a lower S_{pO_2} associated with obesity, in 871 emergency department patients and hospital workers with a median age of 38 and who did not have cardiopulmonary complaints.[10] Our study confirms that this association is present in a much larger elderly community-based sample, and that it persists after more comprehensive adjustment for confounders, including physiologic measures of cardiac and respiratory function. The subjects in the prior study had a median S_{pO_2} of 99%; while values below 97% were rare (5.7% prevalence) and 99–100%

was the most frequent S_{pO_2}. The lower S_{pO_2} values seen in the Cardiovascular Health Study cohort are probably due to a much higher age range.

The effects of obesity on lung function are well described.[1,2] Obesity is associated with restrictive ventilatory impairment and diminished FEV_1, FVC, vital capacity, total lung capacity, functional residual capacity and expiratory reserve volume.[11,12] These changes are felt to be due to the added mechanical load of adipose tissue, which reduces chest wall compliance and impedes diaphragm descent. The pulmonary function changes are mild but may be more significant with severe central obesity. Also obesity may cause peripheral airways disease and air trapping. Our results show that obesity is associated with oxygen saturation, even after adjusting for airways obstruction. The most likely direct mechanism of increased BMI on reduced oxygen saturation would be increased ventilation/perfusion mismatch, with a possible contribution from decreased ventilation and increased oxygen consumption.

The magnitude of the correlation (R = −0.27) and the decrease in S_{pO_2} associated with increasing BMI was modest (approximately 1% lower values in subjects with class II obesity than in those with normal BMI). The prognostic importance of this finding with regards to worse outcome in this cohort is uncertain, and prospective study is needed to assess its impact.

The change in S_{pO_2} per unit change in BMI was 43% less in African Americans, relative to white/other. African Americans on average have lower abdominal visceral adipose tissue than white/other.[13] Higher visceral fat has been associated with decreased lung volumes, most likely due to impaired descent of the diaphragm.[14] The relationship with lower abdominal visceral adipose tissue in African Americans is supported by the absence of a significant interaction between waist size and African American race on S_{pO_2}.

Several limitations should be considered when evaluating the results of this study. There was a high rate of discretionary exclusions from the 6-min walk test. We did not independently verify the accuracy of our S_{pO_2} measurements using arterial blood gases in a subsample of the cohort. We relied on the long-term accuracy of the pulse oximeters (stated by the manufacturer to be within 4%), and did not use an S_{pO_2} validation instrument, but this is rarely done in clinical practice. The oximetry measurements were made in 1996–1997, and the results do not reflect improvements in accuracy and reliability of pulse oximetry technology that may have occurred since then. Echocardiography was performed 2 years prior to the S_{pO_2} measurement and may have led to measurement error regarding left ventricular impairment at the time of oximetry. Echocardiography did not include assessment of diastolic dysfunction, which is common in the elderly and may lead to decreased S_{pO_2}. We did not consider use of medications (ie, inhalers) that may have influenced S_{pO_2}. Carboxyhemoglobin can cause elevation of S_{pO_2} readings and may have led to overestimation of S_{pO_2} in heavy smokers. The study results do not apply to the supine position or exercise, but these settings would be very useful to explore.

Conclusions

In conclusion, we found a narrow distribution of S_{pO_2} values in a community-based sample of ambulatory elderly in whom S_{pO_2} values below 95% were rare. Obesity was a strong independent contributor to a low S_{pO_2}, with effects comparable to or greater than other clinical factors commonly associated with lower S_{pO_2}. This suggests that the effects of obesity on lung function diminish oxygen exchange. The prognostic implications of this finding with regards to long-term outcomes are unclear and require further prospective evaluation,

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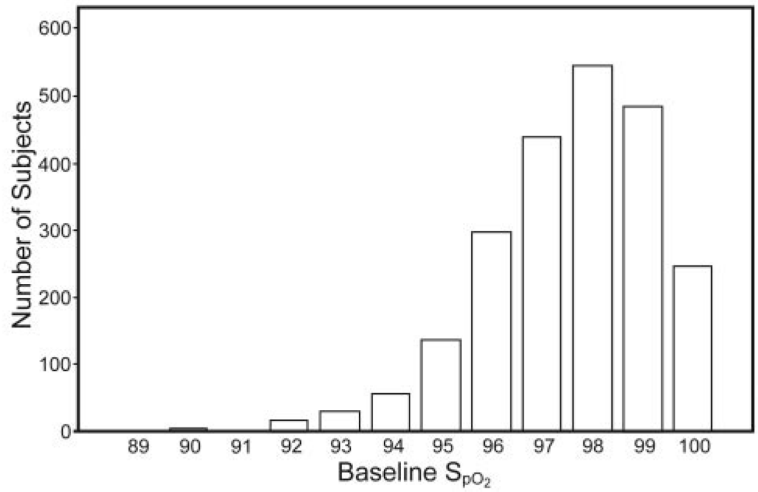

Fig. 1.
The distribution of resting S_{pO_2} in 2,252 older people.

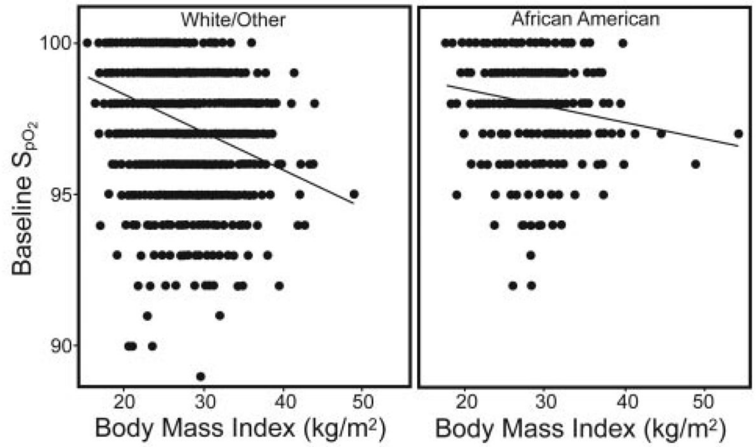

Fig. 2.
Unadjusted regression of S_{pO_2} and body mass index by race.

Table 1

Comparison of Cardiovascular Health Study Subjects With and Without Oximetry Results: 1996–1997

	With S_{pO_2}	Without S_{pO_2}	*P*
n	2,273	2,139	
Age, y	77.4 ± 4.3	79.8 ± 5.6	<.001
Female, %	60.4	63.1	.035
African American, %	14.8	18.8	.005
Physician diagnosis, %			
Heart failure	5.7	16.5	<.001
Emphysema	3.3	5.8	<.001
Asthma	5.9	7.4	.03
Bronchitis	6.3	7.5	.06
Pneumonia	2.4	4.9	<.001
Current smoker, %	6.9	8.6	.02
Pack years	14.9 ± 23.3	16.5 ± 25.4	<.001
Waist circumference, cm	96.6 ± 12.7	98.1 ± 14.3	.003
Body mass index, kg/m^2	26.8 ± 4.4	27.1 ± 5.2	.055
FEV_1, L	1.95 ± 0.59	1.84 ± 0.64	<.001
FVC, L	2.52 ± 0.73	2.39 ± 0.78	<.001
FEV_1/FVC	0.77 ± 0.08	0.77 ± 0.09	.056
Airway obstruction, %	3.9	6.4	.005
Decreased left ventricular ejection fraction, %	8.1	10.9	.02

± Values are mean ± SD.

Table 2

Characteristics of the Cardiovascular Health Study Subjects With S_{pO_2} Values, Stratified by Body Mass Index Category*

	Normal	Overweight	Obese Class I	Obese Class II	*P* for Comparison Across Categories
n (total 2,252)	797	1,013	336	106	
Age, mean ± SD y	78.4 ± 4.78	77.3 ± 4.04	76.1 ± 3.82	76.1 ± 3.35	< .001
Female, %	63.4	55.1	65.2	75.5	< .001
African American, %	9.8	14.5	23.2	27.4	< .001
Heart failure, %	6.3	5.0	6.5	6.6	.60
Emphysema, %	4.4	2.8	3.0	1.9	.20
Asthma, %	4.7	6.2	8.1	5.7	.16
Bronchitis, %	6.5	5.8	5.9	9.9	.43
Pneumonia, %	2.2	2.6	1.8	4.9	.33
Current smoker, %	9.9	5.0	6.6	2.9	<.001
Pack years, median (IQR)	0 (0–22.6)	0.1 (0–24.8)	0 (0–23.5)	0.5 (0–0.45)	.53†
Waist circumference, mean ± SD cm	86.3 ± 8.7	98.3 ± 7.9	108.3 ± 9.1	121.8 ± 10.5	< .001
FEV_1, mean ± SD L	1.92 ± 0.59	2.01 ± 0.59	1.89 ± 0.56	1.82 ± 0.53	< .001
FVC, mean ± SD L	2.52 ± 0.74	2.60 ± 0.73	2.39 ± 0.67	2.30 ± 0.65	< .001
FEV_1/FVC, mean ± SD	0.76 ± 0.08	0.78 ± 0.07	0.79 ± 0.08	0.79 ± 0.06	< .001
Airway obstruction, %	5.7	3.3	2.0	3.4	.02
Decreased left ventricular ejection fraction %	9.4	8.3	8.3	6.6	.74

* BMI categories: normal = < 25 kg/m^2, overweight = 25–29.99 kg/m^2, obese class I = 30–34.99 kg/m^2, obese class II = > 35 kg/m^2

† Log of pack-years

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Table 3

Mean S_{pO_2} by Subject Characteristics

	n	S_{pO_2} (%) mean ± SD	*P*
Body mass index	2,252		< .001
Normal		98.02 ±1.6	
Overweight		97.51 ±1.6	
Obese class I		96.90 ±1.8	
Obese class II		96.69 ±1.8	
Sex	2,252		< .001
Male		97.32 ±1.7	
Female		97.72 ±1.7	
Race	2,252		< .001
African American		97.99 ±1.6	
White/other		97.49 ±1.7	
Smoking	2,222		< .001
Never		97.73 ±1.7	
Past		97.40 ±1.7	
Current		97.33 ± 1.8	
Pack years	2,199		< .001
≤ 60		97.6 ± 1.7	
> 60		96.8 ± 1.9	
Airway obstruction	1,972		< .001
No		97.60 ±1.7	
Yes		96.49 ±1.9	
Emphysema	2,247		< .001
No		97.59 ±1.7	
Yes		96.83 ±1.7	
Asthma	2,211		.24
No		97.57 ±1.7	
Yes		97.45 ±1.6	
Heart failure	2,122		.01
No		97.16 ± 1.7	
Yes		97.59 ± 1.8	
Left ventricular function	2,058		.14
Normal		97.60 ±1.7	
Borderline		97.35 ±1.9	
Impaired		96.92 ±1.8	

Table 4

Multivariable Analysis of Body Mass Index in Relation to S_{pO_2}, by Race Category

	Coefficient (95% CI)	*P*
White/other		
Body mass index	−0.14 (−0.16 to −0.12)	< .001
Age	−0.04 (−0.06 to −0.02)	< .001
Male	−0.26 (−0.42 to −0.10)	.002
Smoking status		
Never		
Past smoker	−0.12 (−0.28 to −0.05)	.17
Current smoker	−0.48 (−0.82 to −0.14)	.005
Airway obstruction	−0.98 (−1.38 to −0.57)	< .001
Emphysema	−0.34 (−0.77 to 0.10)	.13
African American		
Body mass index	−0.09 (−0.13 to −0.05)	< .001
Age	−0.05 (−0.09 to −0.01)	.02
Male	−0.65 (−1.05 to −0.25)	.002
Smoking status		
Never		
Past smoker	−0.17 (−0.56 to 0.22)	.40
Current smoker	−0.50 (−1.22 to −0.22)	.17
Airway obstruction	−2.35 (−3.67 to −1.03)	< .001
Emphysema	−0.64 (−2.04 to 0.76)	.37

Table 5

Multivariable Analysis of Waist Circumference in Relation to S_{pO_2} for Race Groups Combined

	Coefficient (95CI)	***P***
Waist	−0.40 (−0.05 to −0.03)	< .001
Age	−0.03 (−0.05 to −0.01)	< .001
Male	−2.02 (−0.36 to −0.05)	.10
African American	0.50 (0.29 to 0.71)	< .001
Smoking status		
Never		
Past smoker	−0.09 (−0.25 to 0.06)	.25
Current smoker	−0.38 (−0.68 to −0.07)	.02
Obstruction	−1.04 (−1.43 to −0.65)	< .001
Emphysema	−0.42 (−0.84 to 0.00)	.05