

Waist-to-Hip Ratio Is Associated With Pulmonary Gas Exchange in the Morbidly Obese*

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Background: Morbidly obese individuals (*ie*, body mass index [BMI], ≥ 40 kg/m²) may have a pulmonary gas exchange impairment due to the large fat mass surrounding their abdomen.

Purpose: To examine the effect of the waist-to-hip (W/H) ratio on pulmonary gas exchange in the morbidly obese.

Methods: Twenty-five morbidly obese individuals (mean [\pm SD] age, 39 ± 10 years; mean BMI, 49 ± 7 kg/m²; mean body fat, $50 \pm 6\%$; mean waist circumference, 135 ± 15 cm; mean W/H ratio, 0.97 ± 0.11) scheduled for bariatric surgery were recruited. Arterial blood was sampled in duplicate after 5 min of rest sitting upright.

Results: The mean PaO₂ at rest was 88 ± 7 mm Hg (range, 72 to 108 mm Hg), the alveolar-arterial oxygen pressure difference (P[A-a]O₂) was 19 ± 9 mm Hg (range, 1 to 37 mm Hg), and the PaCO₂ was 38 ± 3 mm Hg (range, 32 to 44 mm Hg). Linear regression showed that 32% and 36%, respectively, of the variance in the P(A-a)O₂ and PaO₂ were explained by the W/H ratio ($p < 0.004$ for both). As well, 20% of the variance in PaCO₂ was explained by the W/H ratio ($p = 0.02$). Men had larger W/H ratios ($p < 0.01$) and poorer gas exchange ($p = 0.06$) compared to women (mean difference: PaO₂, -7 mm Hg; P[A-a]O₂, 6 mm Hg).

Conclusion: Morbidly obese men showed a trend to have poorer pulmonary gas exchange compared to morbidly obese women, and a significant part of the blood gas status in these patients is associated with the W/H ratio. (CHEST 2007; 131:362–367)

Key words: arterial blood gases; gas exchange impairment; metabolic syndrome

Abbreviations: BMI = body mass index; LLN = lower limit of normal; P(A-a)O₂ = alveolar-arterial oxygen pressure difference; $\dot{V}O_2$ = oxygen consumption; W/H = waist-to-hip

Pulmonary gas exchange may be affected by morbid obesity. Morbidly obese individuals (*ie*, body mass index [BMI], ≥ 40 kg/m²) have poorer exercise capacity and may also have poorer pulmonary gas exchange compared to healthy, nonobese counterparts because of the added energy needed to move

fat mass.¹ The increase in mechanical ventilatory constraints and lower lung volumes from large amounts of abdominal fat causes poor lung function and is thus one exercise-limiting factor in morbidly

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obese individuals.² The decrease in lung volumes, specifically expiratory reserve volume (as an index of decreased functional residual capacity) is a cause for poor gas exchange in the lung.³

A pulmonary gas exchange impairment at rest may be a prognostic marker for postoperative pulmonary complications. Very few studies have examined pulmonary gas exchange in morbidly obese persons, and those studies have had varied results. Some⁴ have shown that PaO₂ and PaCO₂ remain normal at rest in the morbidly obese, while others^{3,5,6} have shown mild hypoxemia (PaO₂, approximately 78 to 83 mm Hg) with normal PaCO₂ values at rest. However, those studies have provided mean arterial blood gas values and did not provide the individual responses of pulmonary gas exchange in the obese. Indexes of obesity, such as BMI, total body weight, body fat percentage, and waist-to-hip (W/H) ratios, may be related to gas exchange impairments in the morbidly obese, and knowing the individual data in these studies would allow a dissection of the relationship between the magnitude of obesity and the gas exchange impairment.

An attempt has been made to investigate the effect of W/H ratio on pulmonary gas exchange in the morbidly obese. A large W/H ratio indicates that a substantial portion of fat mass is surrounding the thorax, which could lead to ventilation-perfusion abnormalities and an increase in the alveolar-arterial oxygen pressure difference (P[A-a]O₂) while lowering the PaO₂. Consequently, the purpose of this study was to examine the effect of obesity, as depicted by W/H ratios, on P(A-a)O₂ and PaO₂. Our hypothesis was that, as a group, morbidly obese men would have a larger W/H ratio compared to morbidly obese women, and that those with a higher W/H ratio would show poorer pulmonary gas exchange at rest compared to those with a lower W/H ratio.

MATERIALS AND METHODS

Each subject was required to participate in one testing session

Subjects

Twenty-five morbidly obese individuals (mean [\pm SD] age, 39 \pm 10 years; mean BMI, 49 \pm 7 kg/m²; mean body fat proportion, 50 \pm 6%; mean waist circumference, 135 \pm 15 cm; and mean W/H ratio, 0.97 \pm 0.11) who were scheduled for bariatric surgery were recruited. These individuals were community-dwelling, ambulatory persons who were scheduled for laparoscopic weight reduction surgery. Twenty individuals also had normal lung function, as determined by spirometry (*ie*, FEV₁, > 80% predicted,⁷ and normal FEV₁/FVC ratio or FVC of > 0.70). Five subjects did not meet the criteria for normal spirometry (FEV₁, 67 to 79% predicted; and FEV₁/FVC ratio, 58 to 65% predicted). Also, five subjects were below the lower limit

of normal (LLN) for FEV₁,⁷ and two of those five subjects also had FVC values below the LLN. Therefore, seven subjects in total did not have an FEV₁ of > 80% predicted⁷ or an FEV₁/FVC ratio of > 0.70, or were below the LLN for either FEV₁ or FVC. Excluded from the population of the morbidly obese were individuals with (1) BMI \geq 70 kg/m²; (2) respiratory, renal, or hepatic failure; (3) metastatic disease; (4) senility, Alzheimer disease, or other dementias; and (5) the inability to comprehend the instructions during tests. All subjects signed an informed consent form. This study was approved by the Research Ethics Board of the McGill University Health Centre.

The cardiopulmonary variables oxygen consumption (\dot{V} O₂), minute ventilation, carbon dioxide production, respiratory exchange ratio, and the concentration of mixed expired CO₂ were assessed at rest with a metabolic cart (model VMX 229LV; SensorsMedics; Yorba Linda, CA) using the breath-by-breath option. The mean of the 5-min values (averaged over 20-s intervals) was used in the calculations. Heart rate was recorded using a three lead ECG (Cardiacap/5; Datex Ohmeda; Louisville, CO).

Body Composition, Venous Blood Samples, and Arterial Cannulation

Before measuring the resting \dot{V} O₂, height, weight, and body composition were assessed. Lean and fat mass were measured from an 8 polar bioelectrical impedance device that has been validated for the morbidly obese.⁸ Venous blood hematocrit, progesterone and hemoglobin levels, WBC count, RBC count, fasting blood glucose level, and glycosylated hemoglobin concentration, along with a complete lipid profile were measured in each subject. Then, after performing an Allen test, arterial cannulation of the radial artery was performed by an anesthesiologist under local anesthesia (2% lidocaine) using a 20-gauge 1.25-inch length needle (Cathlon Clear Needle; Johnson & Johnson; Arlington, TX).

Arterial blood gases were measured after the patient had rested for 5 min, sitting upright on a chair. The average of the duplicate samples was recorded. All blood gas samples were corrected for changes in arterial blood temperature. A rapid-response, polytetrafluoroethylene (Teflon; Dupont; Wilmington, DE)-coated thermocouple (IT-18; Physitemp Instruments; Clifton, NJ) passed through the rubber part of the extension set (Interlink, Y type, model number JC6613; Baxter Healthcare Corp; Deerfield, IL) and was positioned in the hub of the radial artery catheter. A three-way stopcock was connected to a rubber extension set (Interlink; Baxter Healthcare Corp), and the other end was connected to an IV extension line and saline solution flush fluid transducer (Transpac IV Monitoring Kit; Abbott Laboratories; North Chicago, IL). The catheter, extension set, and stopcock were routinely flushed with heparinized saline solution. Prior to obtaining each arterial blood gas sample, 5 mL of arterial blood was rapidly withdrawn to eliminate the dead space and to see blood temperature increases (5-mL Luer-Lok Tip; Becton-Dickinson; Franklin Lakes, NJ). The highest recorded temperature seen from the thermocouple during this withdrawal was recorded as the arterial blood temperature. Immediately after the 5-mL withdrawal, another 2 mL was withdrawn for actual blood gas analysis using a special syringe (Arterial Blood Sample Syringe, with dry lithium heparin for gases and electrolytes; SIMS Portex Inc; Keene, NH). These blood gas samples were withdrawn over a 10-s period to reduce the fluctuations of blood gas tensions over a given respiratory cycle. Any bubbles were expelled within 5 s of sampling. The samples were stored on ice and analyzed within 10 min of sampling.

Arterial blood PO₂, and PCO₂ were measured directly with a blood gas analyzer (ABL725 Blood-Gas Analyzer; Radiometer;

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