

The Effect of Massive Weight Loss on Arterial Oxygenation and Pulmonary Function Tests

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Pulmonary function tests (PFTs) and arterial oxygenation were studied 24 hours preoperatively and again at varying times (5 to 20 months) during the postoperative weight loss of 37 morbidly obese patients. Among PFTs, expiratory reserve volume (ERV) demonstrated the most significant ($P < 0.001$) improvement with weight loss. No clinically relevant differences were found after weight loss in FEV₁, FVC, or MMEF. In 11 patients whose arterial blood gases were sampled before and after weight loss, a significant correlation was demonstrated between change in ERV and change in PaO₂ ($r = 0.59$, $P < 0.05$) and P(A-a)_{O₂} ($r = 0.76$, $P < 0.01$). When weight loss is expressed as either change in body mass index (Δ BMI) or as a change in percent of ideal weight (Δ wt), there existed a weight loss threshold for improvement in arterial oxygenation. A Δ BMI of greater than 20 or a Δ wt more than 100 per cent resulted in a significant improvement in PaO₂ ($P < 0.01$) or P(A-a)_{O₂} ($P < 0.05$). These data suggest that morbidly obese patients do not meaningfully improve FEV₁, FVC, or MMEF with massive weight loss. However, there is a significant improvement in ERV that directly correlates with improvement in both PaO₂ and P(A-a)_{O₂}. (Key words: Complications: obesity. Lung: function. Oxygen: tension.)

MORBID OBESITY is characterized by pulmonary dysfunction as determined by arterial blood gases and certain pulmonary function tests.^{1,2} Alexander and Peterson have shown that the circulatory effects of massive obesity are reversible with weight loss.³ It is generally assumed that pulmonary function might also improve with weight loss. However, appropriate data to confirm that assumption have yet to be demonstrated. The purpose of this study was to investigate the effect of massive weight loss on pulmonary function and indices of oxygenation [PaO₂ and P(A-a)_{O₂}].

Methods

Approval of the study protocol was obtained from the Human Subjects Committee. Morbidly obese patients (BMI > 30§) scheduled for elective jejunoileal small bowel bypass were studied 24 hours preopera-

tively, and again at varying intervals postoperatively for prospective followup. The nature of the study was explained to each patient, and informed consent obtained. All patients were free of primary cardiorespiratory disease and denied a smoking history. In addition, all patients denied respiratory symptoms, such as regular morning cough, sputum production, or wheezing.

Thirty-seven consecutive patients (34 females and 3 males) were studied. Age ranged from 21 to 51 years, with a mean \pm SEM of 32.6 ± 1.2 . Other population descriptors, including girth, and ideal weight⁴ are shown in table 1. Mean patient height was 167.5 ± 0.9 cm. Duration of weight loss ranged from 5 to 20 months, with an average duration of 10.3 months.

With the patient sitting, spirometric tracings were recorded on a 9L Collins® Spirometer. Expiratory reserve volume (ERV) and forced vital capacity (FVC) were measured directly from the tracing. Forced expiratory volume in one second (FEV₁) and maximum midexpiratory flow (MMEF) were calculated from these tracings according to standard techniques.⁵ The maximum value obtained for each measurement was used, provided that the spirogram from which the measurement was derived was within 200 ml of the maximum FVC for the patient. Predicted values for ERV were calculated by subtracting predicted residual volume (RV)⁶ from predicted FRC.⁷ All pulmonary function tests were corrected to body temperature and pressure fully saturated (BTPS).

Arterial blood gases were drawn from 11 of the study patients before and after weight loss. Blood gases were drawn after each subject had been supine and breathing room air for 15 min. Blood was collected into iced, heparinized plastic syringes and analyzed within 10 min by the same technician (D.H.) using an Instrumentation Laboratory Model® 213 blood gas analyzer. Alveolar-arterial oxygen tension difference was obtained from the following equation:

$$P(A-a)_{O_2} = 0.21(P_B - P_{H_2O}) - \frac{P_{aCO_2}}{0.8} - Pa_{O_2}$$

where P_B is barometric pressure, P_{H₂O} is water vapor tension at 37° C, and a respiratory quotient of 0.8 is assumed. Previously, Ellison *et al.*,⁸ reported no measurable change in the respiratory quotient as a result of routine surgery. That conclusion was supported by subsequent data from Spence and Smith.⁹

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§ Body mass index (BMI) = weight (kg)/height² (m²).

TABLE 1. Physical Changes (Mean ± SEM) in Obese Subjects (n = 37) with Weight Loss

	Weight Loss		Mean* Difference
	Before	After	
Weight (kg)	139 ± 4	88 ± 3	52 ± 2
Girth (cm)	147 ± 2	107 ± 3	40 ± 2
BMI† (kg/m ²)	50 ± 1	31 ± 1	18 ± 1
BSA‡ (m ²)	2.39 ± 0.03	1.96 ± 0.03	0.43 ± 0.02

* Significantly different (before to after weight loss) at $P < 0.001$.
 † Body mass index (BMI) = weight/height².
 ‡ Body surface area.

Other patient data collected before and after weight loss included age, height, weight, and girth. Girth was measured at the level of the umbilicus at normal end (resting) exhalation with the patient standing. Ideal weight was obtained from the Metropolitan Life Insurance Tables.⁴ Calculated morphologic indices include body surface area (BSA) in square meters¹⁰ and body mass index (BMI).¹¹

The Student *t* test for paired data (before *vs.* after weight loss) was used to test for significant changes in pulmonary function tests and arterial blood gases. Linear regression analysis was employed to discern the degree of correlation between associated variables. Significance was defined as $P < 0.05$.

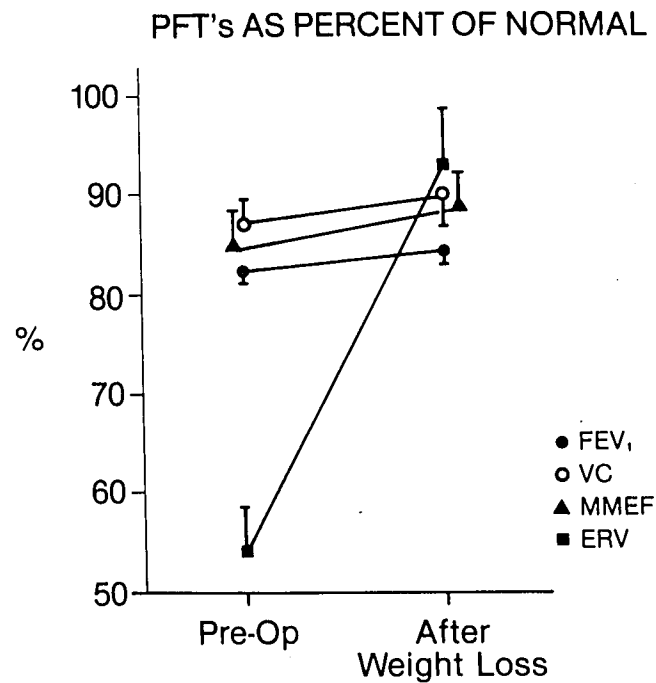


FIG. 1. Pulmonary function tests (PFTs) as per cent of normal before (preop) and after weight loss. FEV₁ (forced expiratory volume in 1 s); VC (vital capacity); MMEF (maximum mid-expiratory flow rate); ERV (expiratory reserve volume). All values ± SEM.

Results

Changes in weight, girth, BMI, and BSA are shown in table 1. Mean weight loss was 52 kg. All changes are significant at $P < 0.001$. Changes in pulmonary function tests are illustrated as a per cent of normal predicted values before and after weight loss (fig. 1). FEV₁ increased slightly but significantly from 82.4 ± 1.2 per cent to 84.2 ± 1 per cent ($P < 0.05$), while FVC and MMEF did not significantly change. There was a large and significant increase in ERV from 54.6 ± 4.1 per cent to 93.2 ± 5.7 per cent ($P < 0.001$). There was no significant correlation between the change in ERV (Δ ERV) and change in girth, weight, body mass index, or body surface area.

Table 2 summarizes the arterial blood gases obtained before and after weight loss. No significant differences were found in PaO₂, P(A-a)O₂, PaCO₂, pH, or [HCO₃⁻] over the period of weight loss. However, linear regression analysis revealed significant correlations between improvement in oxygenation [Δ PaO₂ and Δ P(A-a)O₂] and change in measured morphologic variables for these eleven patients (table 3). Improvement in PaO₂ also correlated with duration of weight loss ($r = 0.59, P < 0.05$), but was not significantly correlated with either change in girth or patient age. A significant correlation was found between Δ ERV and improvement in oxygenation. In the 11 patients whose arterial blood gases were sampled before and after weight loss, a significant correlation was demonstrated between Δ ERV and change in P(A-a)O₂ ($r = 0.76, P < 0.01$) and PaO₂ ($r = 0.59, P < 0.05$; table 3).

Figure 2 illustrates the change in P(A-a)O₂ [Δ P(A-a)O₂] as a function of weight loss expressed as change in weight for each patient in BMI units. A Δ BMI < 20 results in no significant change in P(A-a)O₂ or PaO₂, while a Δ BMI > 20 results in a significant improvement in P(A-a)O₂ ($P < 0.05$) and PaO₂ ($P < 0.01$). A similar relationship exists for weight loss expressed as a per cent of ideal weight. A weight loss of less than 100 per cent of ideal weight results in no signifi-

TABLE 2. Arterial Blood Gas Values (Mean ± SEM) in Obese Subjects (n = 11) Before and After Weight Loss

Arterial Blood Gas Values	Weight Loss		Mean Difference	P Value*
	Before	After		
PaO ₂ (torr)	83.2 ± 2.5	87.2 ± 1.7	4.0 ± 1.8	0.053
P(A-a)O ₂ (torr)	22.6 ± 2.8	19.7 ± 1.9	2.9 ± 2.1	0.202
PaCO ₂ (torr)	36.8 ± 1.1	35.9 ± 0.6	0.9 ± 0.7	0.250
pH	7.43 ± 0.01	7.43 ± 0.01	0.00 ± 0.01	0.877
HCO ₃ ⁻ (mEq/l)	23.4 ± 0.9	22.9 ± 0.6	0.5 ± 1.0	0.610

* Not statistically significant before to after weight loss.

cant change in $P(A-a)_{O_2}$ or Pa_{O_2} , but a weight loss of more than 100 per cent of ideal weight results in a significant improvement in $P(A-a)_{O_2}$ ($P < 0.05$) or Pa_{O_2} ($P < 0.01$). There was no significant correlation between improvement in oxygenation [$\Delta P(A-a)_{O_2}$ or ΔPa_{O_2}] and final weight as expressed by any of the morphological indices (weight, BMI, per cent of ideal weight, BSA, or girth).

Discussion

FVC and MMEF did not improve significantly with weight loss. Although FEV_1 showed a statistically significant increase ($P < 0.05$) with weight loss, the change from 82.4 per cent to 84.2 per cent of normal is not clinically relevant. Of the four spirometry measurements taken preoperatively, FVC, MMEF, FEV_1 , and ERV, ERV was the most abnormal. Thus, it is not surprising that the greatest change in any of the tests of pulmonary function was that observed in ERV. Despite the improvement in ERV with massive weight loss, this change did not correlate with amount of weight loss or with change in girth.

The fact that FVC did not increase and ERV did, suggests that end expiratory lung volume (FRC) increased. Improved ERV as an index of FRC may account for the salutary effect of massive weight loss on oxygenation. This is supported by the correlation of increased ERV with improvement in both Pa_{O_2} and $P(A-a)_{O_2}$. A physiologic basis for this correlation is

TABLE 3. Patient Variables Correlated with Improvement in Oxygenation in Eleven Obese Subjects

Patient Variable	Oxygenation Variable			
	$\Delta P(A-a)_{O_2}$ * (torr)		ΔPa_{O_2} † (torr)	
	Correlation Coefficient (r)	Significance (P)	Correlation Coefficient (r)	Significance (P)
Age	0.28	0.201 (NS)‡	0.01	0.486 (NS)‡
Duration of weight loss	0.73	0.005	0.59	0.028
Weight loss	0.77	0.003	0.65	0.015
Δ BMI (body mass index)	0.82	0.001	0.75	0.004
Δ Per cent ideal weight	0.82	0.001	0.77	0.003
Δ BSA	0.73	0.005	0.61	0.023
Δ Girth	0.59	0.027	0.45	0.080 (NS)‡
Δ ERV	0.76	0.005	0.59	0.036

* Change (Δ) in alveolar to arterial oxygen tension difference.
 † Change (Δ) in arterial oxygen tension.
 ‡ NS = not significant.

described by Craig *et al.*¹² They describe obesity as one of the conditions which promote breathing at reduced lung volumes, especially in the supine position. In addition, LaBlanc *et al.*¹³ point out that with the

$P(A-a)O_2$ CHANGES WITH WEIGHT LOSS

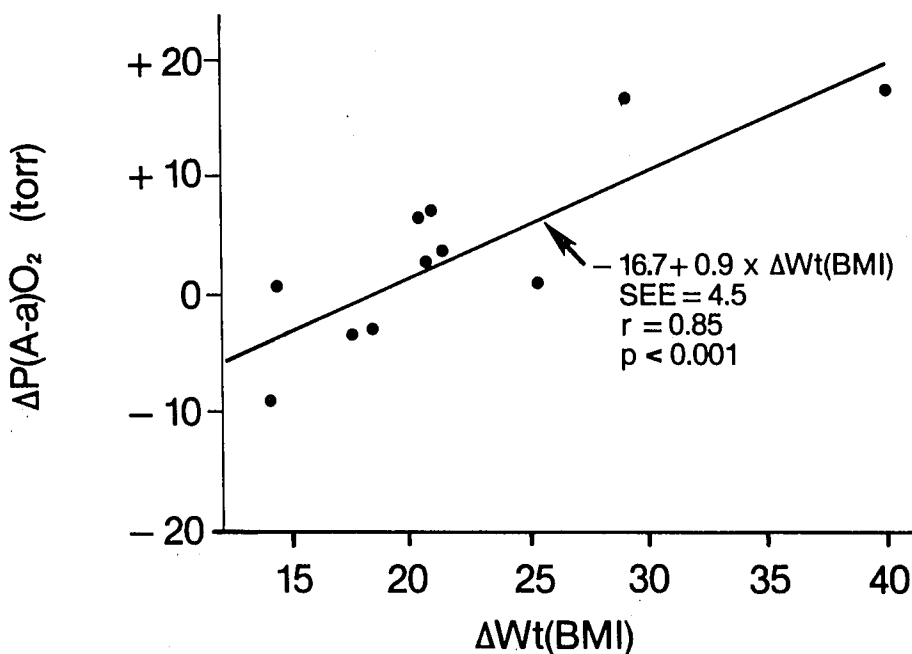


FIG. 2. Alveolar to arterial oxygen tension difference [$P(A-a)_{O_2}$] in torr with weight loss expressed as change in weight (ΔWt) in units of body mass index (BMI) in eleven morbidly obese patients. A change in body mass index (ΔBMI) of ≥ 20 results in a significant improvement in oxygenation.

decrease in FRC observed in obesity, there develops an increased probability that closing capacity will fall within tidal breathing. Ventilation to affected regions is impaired, thereby lowering regional ventilation/perfusion (\dot{V}/Q) ratios and promoting arterial hypoxemia.

The change in Pa_{O_2} with weight loss was nearly significant ($P = 0.053$), and if more than 11 patients had been studied, a significant difference might have been demonstrated. However, the number of patients was limited by the difficulties associated with long-term followup in this study population.

The best morphological correlates with improvement in oxygenation were weight loss expressed as a per cent of ideal weight and weight loss expressed as a change in BMI. For the practicing clinician, BMI can be calculated much easier than referring to published tables for looking up ideal weight. Normal BMI is less than 27, and a BMI of greater than 30 is indicative of excessive obesity.¹⁴

The confounding effect of amount of time since jejunioleal bypass must be considered in any discussion of the effects of weight loss after such a procedure. Bray¹⁵ describes a catabolism phase and an adaptive phase after bypass. The former lasts about 6 months and represents the period of maximal malnutrition, maximal weight loss, and most profound intestinal malabsorption. Malabsorption and malnutrition gradually subside as the intestine adapts. When this happens, weight loss slows and a new steady weight occurs. Whether these adverse metabolic effects influence the respiratory quotient, lung volume, pulmonary perfusion, and subsequent oxygenation was not addressed in the present study. Indeed, the two patients with the least weight loss (fig. 2), were both only five months after operation. They may not have reached their "adaptive phase," thus explaining the lack of demonstrated significant improvement in oxygenation.

We therefore conclude that in the absence of intrinsic respiratory disease, morbidly obese subjects do not meaningfully improve FEV_1 , FVC, or MMEF with massive weight loss. However, there is a significant increase in ERV that directly correlates with improvement in both Pa_{O_2} and $P(A-a)_{O_2}$. Arterial blood gases were not significantly improved over the period

of observation, but changes in Pa_{O_2} and $P(A-a)_{O_2}$ were correlated with weight loss expressed in terms of body mass index. Patients who decreased BMI by at least 20, showed a significant improvement in oxygenation, as expressed by either Pa_{O_2} or $P(A-a)_{O_2}$.

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