

Fig 2 Weight distribution of patients before and after surgery and weight loss. \blacktriangle Mean value. IBW—ideal body weight.

gastroplasties and is currently the most widely performed bariatric operation worldwide.³

Patients and methods

We studied 29 consecutive subjects (one male) undergoing bariatric surgery. Their mean age was 39.3 (range 18–56) years. Fourteen either were current smokers or had smoked in the past. All were selected by virtue of being 100% or more above their ideal body weight or 50 kg or more above for at least three years.⁴ All the subjects had been given a supervised outpatient diet for one year and had failed to lose weight. Informed consent to the investigations and operation was obtained from all patients. Their mean weight before operation was 126.4 (range 92–174) kg. The body mass index was calculated as weight divided by the square of the height (kg/m^2).⁴

Forced expiratory volume in one second (FEV_1) and forced vital capacity (FVC) were measured by dry wedge spirometry (Vitalograph) with the patients seated after 15 minutes' rest. Peak expiratory flow (PEF) was measured by a Wright's peak flow meter. Lung volumes (total lung capacity (TLC), functional residual capacity (FRC), residual volume (RV), and expiratory reserve volume (ERV)) were measured by

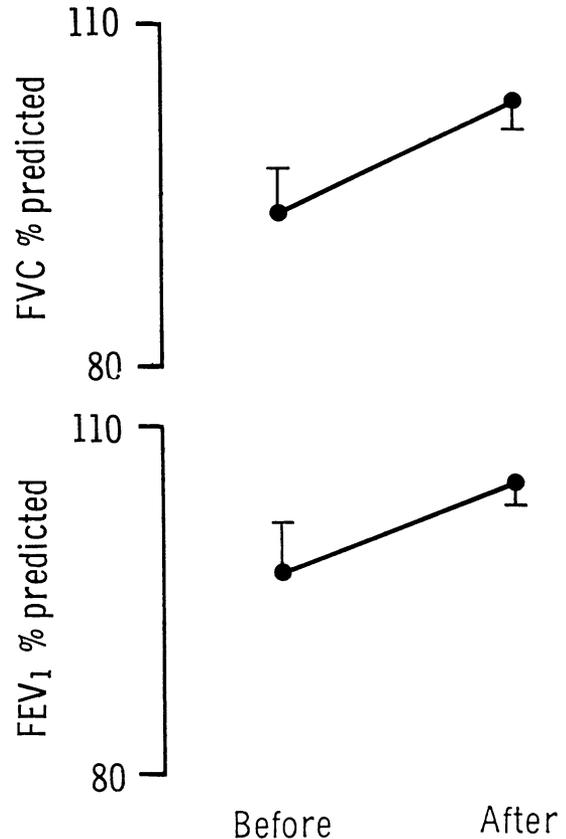


Fig 3 Mean FEV_1 and forced vital capacity (FVC), before and after weight loss. Bar lines indicate 1 SEM.

helium dilution. Carbon monoxide gas transfer (TLCO) was assessed by the single breath technique in 10 subjects. The Krogh factor (K_{CO}) was calculated as the TLCO per litre of effective alveolar volume. Arterial blood gases were obtained from the radial artery in 14 patients with a local anaesthetic, with the subject seated and breathing air after resting for 15 minutes.

Results have been expressed as percentages of predicted values.⁵ The regression equations that determine predicted values do not take weight into account except in the case of ERV.⁵ Because with the weight correction many patients would have had a negative predicted ERV, we expressed ERV as percentage predicted on the basis of ideal body weight.

Statistical analysis was by paired two tailed *t* test unless otherwise stated.

Results

The subjects were followed up for a mean of 26 (range

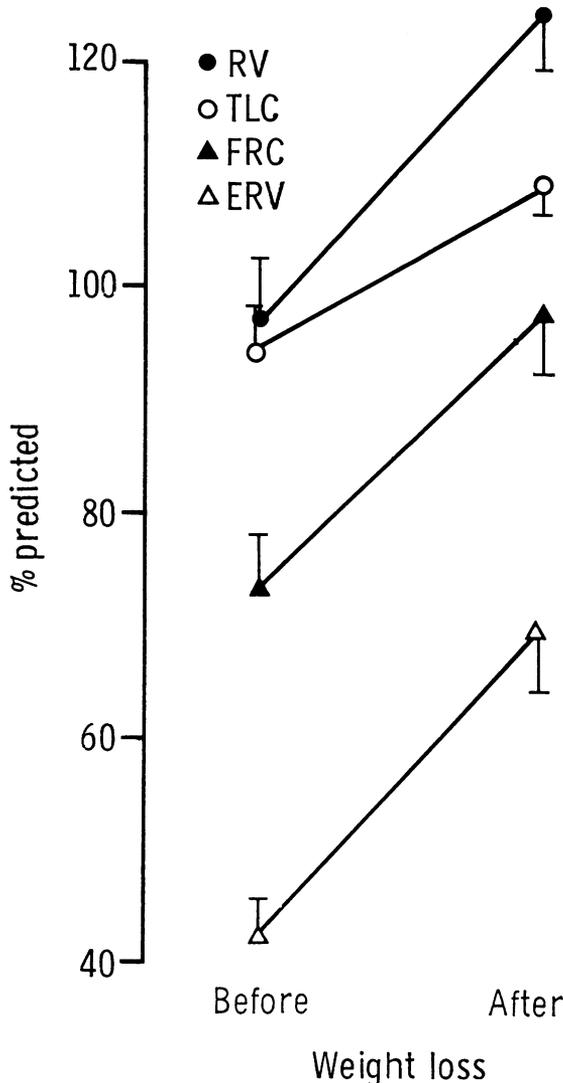


Fig 4 Mean residual volume (RV), total lung capacity (TLC), functional residual capacity, and expiratory reserve volume before and after weight loss. Bar lines indicate 1 SEM.

2–66 months. All lost weight—mean 34.2 (range 2–64) kg (fig 2). The main changes in dynamic and static lung volumes are summarised in figures 3 and 4. Before weight loss FVC, FEV₁, and PEF were not significantly below predicted values; after weight loss, however, there was a small but significant rise in FVC of 7% ($p = 0.002$) and in FEV₁ of 6% ($p = 0.017$), with no change in PEF.

FRC, TLC, RV, and ERV were all reduced before

surgery, especially ERV (figs 3 and 4). All showed a significant increase after weight loss. When values for the whole group were expressed as change in % predicted values from preoperative values RV increased by 26%, TLC by 14%, FRC by 37%, and ERV by 54%. Similar changes were seen in smokers and non-smokers. There was no difference in lung function between smokers and non-smokers before surgery. After surgery and weight loss, however, FRC, TLC, and RV (but not ERV) were larger than predicted in smokers and significantly greater than in those who had never smoked ($p < 0.05$, unpaired t test).

Arterial blood gas analysis showed an initial slight hypoxaemia, with a mean arterial oxygen tension (Pao₂) of 10.6 (SEM 1.64) kPa, which improved after weight loss to normal values (13.0 (0.55) kPa, $p < 0.005$: fig 5). Arterial carbon dioxide tension (Pco₂) fell from 5.2 (0.6) to 4.6 (0.16) kPa ($p < 0.05$) after surgery, suggesting an improvement in either ventilation or gas exchange. There was, however, no significant change in TLCO or KCO in the 16 subjects who had the measurements made before and after surgery. Mean (SEM) TLCO was 8.32 (0.93) mm min⁻¹ kPa⁻¹ before and 8.12 (0.67) after weight loss; KCO was 1.83 (0.1) before and 1.76 (0.07) mmol min⁻¹ kPa⁻¹ l⁻¹ after weight loss.

Improvement in blood gas tensions and lung function variables did not correlate with change in weight, whether actual weight loss, percentage ideal body weight change, or change in body mass index or weight loss index.

Tables of the full data are available (from JSM).

Discussion

The benefits of loss of weight on lung function in the morbidly obese are seen clearly in this study of 29 patients. There was a significant improvement in lung function, most notably a 54% increase in ERV and improvement in gas exchange.

Physicians interpreting lung function results should bear in mind obesity as a cause of a decrease in lung volumes or hypoxaemia, and a case may be made for noting weight routinely when patients are assessed in the laboratory.

Obesity probably causes respiratory embarrassment by several mechanisms. Our study has emphasised that ERV, RV, FRC, and TLC are reduced by comparison with values after weight loss (fig 4), most of the change in TLC being accounted for by a change in ERV and RV. This, in the presence of a normal FEV₁ and vital capacity, suggests that splinting of the diaphragm by intra-abdominal fat may prevent its full excursion, and this would account for some of the changes. It has been suggested that pulmonary compliance is decreased by

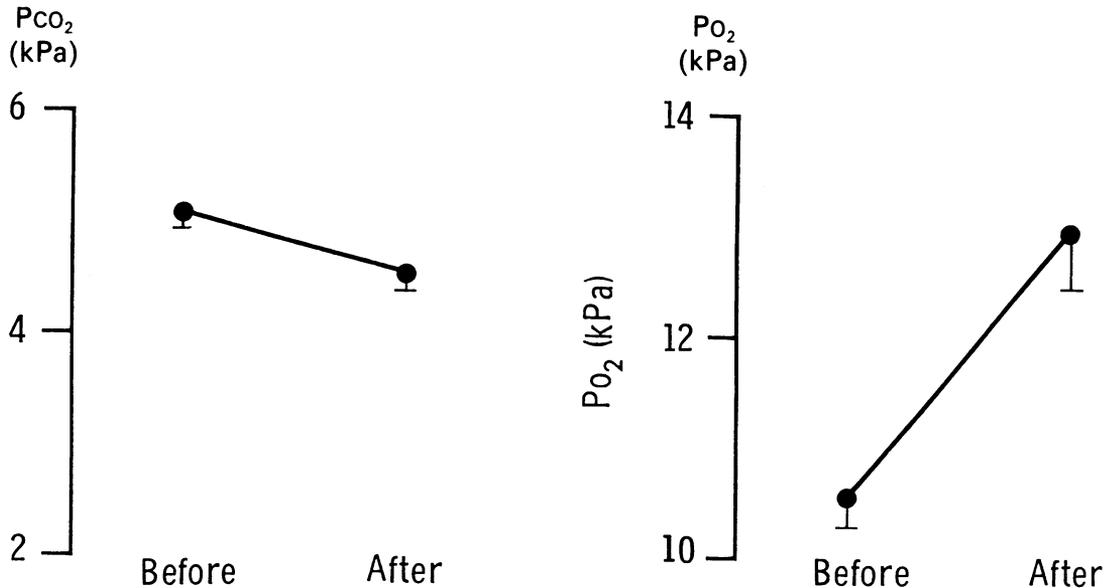


Fig 5 Mean arterial carbon dioxide and oxygen tensions (PCO_2 and PO_2) before and after surgery.

the deposition of subcutaneous adipose tissue, acting as a fat envelope, in the chest wall.⁶ Respiratory muscle function may also be compromised, both by an initial mechanical disadvantage caused by fat and by the need to use more energy to expand the lungs than is used by a lean individual.⁷ Gas exchange deficits are evident from our study. This may be due to a combination of ventilation-perfusion mismatch and hypoventilation. Causes of ventilation-perfusion mismatch include underventilation of well perfused lower lung regions, which has been observed in obesity.^{8,9} Airway closure was detected by the nitrogen washout method in some obese patients during tidal breathing. This was reversed by weight loss and an increase in the FRC.¹⁰

The fact that carbon monoxide uptake did not change after weight loss suggests that ventilation-perfusion mismatch was less important than hypoventilation in causing the abnormal gas exchange. This is supported by observations of hypoventilation and disordered breathing patterns in the obese.² The abnormal breathing pattern may be due to an abnormality of central control or possibly obese individuals reach the limit of the amount of work that can be achieved by the respiratory muscles. If a central abnormality were present an attenuated ventilatory response to carbon dioxide and to hypoxia would be expected. In a study of four obese patients before and after weight loss there was a fall in the slope of ventilatory response to carbon dioxide when weight was lost, the reverse of what would be expected.¹¹ Thus the available data favour the idea that ventilation is

limited by the increased work of breathing in obesity, relative hypoventilation occurring because of respiratory muscle fatigue and chronic inability to respond to hypercapnia and hypoxia.⁷

Most previous studies of the effect of bariatric operations have shown an improvement in arterial oxygenation only in a relatively few patients. Other workers, like us, have been unable to show a convincing correlation between loss of weight and improvement of lung function variables. In one study that purported to show a positive relation between reduction in shunt and weight loss only six of 11 patients showed improved oxygenation and loss of weight.¹² The relation between weight loss and improvements in measured lung function variables is not clear. Our results have too much scatter to show a definite link. Perhaps a different index, such as pulmonary compliance or a measurement of respiratory muscle fatigue, might relate more directly to weight loss.

Before surgery there was no difference in spirometric values and lung volumes between smokers and non-smokers, whereas after weight loss smokers were found to have significantly more air trapping than the non-smokers, who had reached normal predicted respiratory values. The effect of smoking on lung function tends to be obscured by morbid obesity.

Patients with this degree of obesity have a high risk of postoperative complications, but vertical banded gastroplasty results in less metabolic disturbance than alternative operations. In this series no patient died in the postoperative period, though two patients died at

home of pulmonary embolism six and seven weeks after their operation.

Vertical banded gastroplasty has been carried out in this hospital in 60 patients, including the 29 reported here. Major wound infection has occurred in six cases and minor infection (reddening and serous discharge) in 17. Other complications, such as thromboembolism (1 case), chest infection (8), and gastric bleeding (1), added to the morbidity and late complications included the two deaths from pulmonary embolism, staple line dehiscence (4 cases), stoma stenosis (5), and regaining of weight (4). The life expectancy of morbidly obese people is considerably reduced, a trend that may be reversed by bariatric surgery. With better respiratory function after weight loss these patients can look forward to increased mobility.

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