Chest mechanics in morbidly obese non-hypoventilated patients

W. LADOSKY, M. A. M. BOTELHO AND J. P. ALBUQUERQUE JR

Department of Internal Medicine, The University Hospital, Fed. University of Pernambuco, Recife, Brazil

Seventy-seven patients with morbid obesity, body mass index (BMI) 40–69 kg m\(^{-2}\), who were candidates for gastroplasty, were studied in our laboratory as part of a pre-operative survey. They had no complaints other than obesity and were not cyanotic. A group of 28 lean subjects (BMI 20–29.8 kg m\(^{-2}\)) who were candidates for abdominal surgery, without any respiratory complaint, were included as controls. For each patient a pulmonary function test was performed, measuring slow vital capacity with expiratory residual volume (ERV), forced vital capacity (flow/volume) and maximal voluntary ventilation (MVV).

In obese patients the MVV is reduced as BMI increases. This results in the reduction of expiratory flows and volumes. Forced expiratory volume in 1 sec (FEV\(_1\)) is reduced in proportion to the FVC reduction and is related to MVV. It is suggested that the main consequence of the burden of the chest wall by increased adipose mass is a reduction in its compliance, making inspiration increasingly difficult, and resulting in lower static volumes and flows.

**Key words:** obesity; maximal voluntary ventilation; body mass index; chest wall compliance; expiratory flows; inspiratory flows.

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**Introduction**

Morbid obesity may hinder respiratory function even in the absence of other diseases. Chen *et al.* (1), in a 6-year follow-up study in Canada, reported a decline of pulmonary function closely related to weight gain. Increased body mass index (BMI) results in an increased mass on the chest wall, including the rib cage, affecting thoracic expansion and leading to restrictive impairment of pulmonary function. In these patients respiratory compliance is reported to be reduced (2–8). However, Suratt *et al.* (9), using the pulse-flow method, did not find any correlation between BMI and chest wall compliance.

Mechanical impairment has been described (10–14) in obese patients, and is characterized by reduction of maximal voluntary ventilation (MVV). This has not been widely confirmed, as Dempsey *et al.* (15) and Kollas *et al.* (16) described that in patients with slightly increased BMI (around 40 kg m\(^{-2}\)) values for MVV were found to be normal. Ray *et al.* (17) described a reduction of MVV when the weight/height ratio exceeds 0.90, and Sahebjami *et al.* (18) found normal or low MVV depending on the subject’s weight. The deficiency in chest wall mechanics is probably due to the burden induced by increased adipose tissue over the chest. This was experimentally demonstrated by Caro *et al.* by strapping the chest wall (19).

Flow and static volumes have been described as normal (3,5,10,12,20–24) or decreased (15–18,25–26). There is, however, general agreement that expiratory residual volume (ERV) is significantly reduced (2,3,5,6,13–15, 20, 21, 24–26) in the obese patient, and that it is related to BMI. A relationship between the reduction of ERV and that of ventilation/perfusion (V/Q) had been also described (22,25).

This study was undertaken to determine the influence of body mass on chest wall mechanics, the degree of alteration in pulmonary function, and the consequence on pulmonary volumes and flows.

**Material and methods**

**SUBJECTS**

General surgery patients, endocrinology outpatients and candidates for gastroplasty with a diagnosis of morbid obesity (98 patients in total) were reported to us for pre-operative pulmonary evaluation. Only patients with a BMI greater than 40 kg m\(^{-2}\) were selected, reducing the number of candidates to 84. All subjects, in spite of their obesity, were in good health and none presented with sputum, cough or cyanosis. At spirometry, patients that presented a back-extrapolated volume of forced vital capacity (FVC) higher than 5% or could not perform a good expiratory
effort, according to the Brazilian (27), ATS (28) or ECCS (29) consensus for spirometry, were discarded from the study. The final group was therefore reduced to 77 patients (19 males and 59 females) aged from 21 to 59 years, with a BMI ranging from 40–49 kg m⁻².

The control group was composed of 28 subjects (10 males, 18 females), ranging from 21 to 56 years of age, without any pulmonary complaints. Their pulmonary function was evaluated before abdominal surgery and all had normal thorax RX. Voluntary medical students were also included as controls. All control group participants were selected on the basis of BMI ranging from 20 to 29 kg m⁻² and normal forced spirometry.

**EQUIPMENT**

A fluxometric spirometer (Beatrice, EBEM Inc., Recife, Brazil) using a Fleisch IV Pneumotachometer linked to a differential pressure transducer (Celesco, California, U.S.A.) meeting international standards, was used. The software permitted a high precision of inspiratory and expiratory flow and was therefore also suitable for the MVV measurement. As well the measurements for the flow–volume curve it also measured the back-extrapolated volume (BEV) and compared it to the FVC (BEV in FVC %).

**FUNCTION TESTS**

Ventilatory function was assessed by measuring vital capacity (VC), FVC and MVV, in that order.

All tests were performed in the morning with patients seated. Each one of the patients performed at least four tests. For VC measurement the best value was selected and from this manoeuvre the ERV was obtained. The flow–volume curve was calculated according to the method of D’Angelo et al. (30) and the best FVC + forced expiratory volume (FEV₁) was selected, provided that their FVC did not differ by more than 5% or the difference was not greater than 200 ml. To calculate predicted values the equations proposed by Pereira et al. (31) for the Brazilian population were adopted. Spirometric diagnoses were made according to the Brazilian Consensus of Spirometry (27).

For MVV patients were asked to perform a sequence of forced inspirations and expirations over 12 sec, within a frequency of 30–50 min⁻¹. The highest value over five trials was taken for analysis. The equations proposed by Kory et al. (32) for men and Lindall et al. (33) for women were used to calculate the predicted values.

**STATISTICS**

Epidemiology statistics software from The Emory University, Atlanta, GA, U.S.A. (Epi 6a) for epidemiological analysis was used. To analyse differences between means an ANOVA test was used; to compare diagnostics differences the χ²-test was used.

**Results**

Table 1 shows general data of the subjects and spirometric results obtained.

Data collected were divided in three groups according to patients’ BMI. Group A included subjects with BMI of 40–49 kg m⁻²; group B had BMI of 50–59 kg m⁻²; group C had BMI of 60–99 kg m⁻². Results are presented in Table 1 and the best fit line for MVV and BMI are shown in Fig. 1.

**DIAGNOSTICS**

No obstructive or combined spirometric impairment was detected in any group of obese subjects. By selection all lean subjects were spirometrically normal. As seen in Table 2,

![Table 1. Spirometric values and subject data of lean and obese patients](image-url)

<table>
<thead>
<tr>
<th>Sex (M/F)</th>
<th>Controls</th>
<th>Group A (n = 36)</th>
<th>Group B (n = 32)</th>
<th>Group C (n = 9)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>21–56</td>
<td>21–51</td>
<td>24–59</td>
<td>22–44</td>
</tr>
<tr>
<td>BMI (kg m⁻²)</td>
<td>24.79 (1.15)</td>
<td>45.90 (2.49)</td>
<td>54.40 (2.54)</td>
<td>63.50 (4.34)</td>
</tr>
<tr>
<td>FVC %</td>
<td>99.04 (2.47)</td>
<td>89.37 (2.99)</td>
<td>80.16 (3.04)</td>
<td>73.12 (2.27)</td>
</tr>
<tr>
<td>FEV₁ %</td>
<td>97.01 (2.62)</td>
<td>86.96 (3.10)</td>
<td>76.91 (3.29)</td>
<td>72.86 (4.29)</td>
</tr>
<tr>
<td>FEV₁ %</td>
<td>90.39 (1.13)</td>
<td>88.73 (0.77)</td>
<td>87.53 (1.46)</td>
<td>86.00 (2.65)</td>
</tr>
<tr>
<td>ERV (ml)</td>
<td>885 (53)</td>
<td>502 (63)</td>
<td>300 (56)</td>
<td>243 (20)</td>
</tr>
<tr>
<td>MVV %</td>
<td>124.18 (5.19)</td>
<td>104.12 (5.05)</td>
<td>93.71 (5.24)</td>
<td>75.35 (5.03)</td>
</tr>
<tr>
<td>FEF 50%</td>
<td>141.04 (9.66)</td>
<td>101.86 (4.85)</td>
<td>101.72 (7.59)</td>
<td>83.75 (9.85)</td>
</tr>
<tr>
<td>PEF %</td>
<td>97.18 (5.18)</td>
<td>90.71 (4.07)</td>
<td>90.10 (6.48)</td>
<td>85.67 (5.69)</td>
</tr>
<tr>
<td>PIF %</td>
<td>87.75 (5.33)</td>
<td>73.63 (6.05)</td>
<td>67.84 (4.21)</td>
<td>57.33 (4.52)</td>
</tr>
<tr>
<td>FIF 50%</td>
<td>133.32 (8.12)</td>
<td>108.79 (5.91)</td>
<td>108.82 (7.23)</td>
<td>86.00 (9.39)</td>
</tr>
</tbody>
</table>

Group A: BMI 40–49 kg m⁻²; group B: BMI 50–59 kg m⁻²; group C: BMI 60–99 kg m⁻².

ᵃP < 0.05;ᵇP < 0.01;ᶜP < 0.005 (all results expressed as mean ± sd)
the incidence of restrictive pulmonary impairment increases proportionally with increase in BMI.

MVV

Increase in BMI induces a highly significant decrease of MVV ($t = 2.18, P < 0.01$) but the negative correlation was non-linear ($r = -0.12$). These results become more impressive when compared to data from lean subjects, where the relationship between BMI and MVV is positive and more linear ($r = 0.34$). As seen in Fig. 1 this correlation is reversed when obese and lean subjects are compared.

STATIC VOLUMES

As shown in Fig. 2 FVC and FEV$_1$ decrease in obese patients as BMI increases. In group A both are already significantly lower ($P < 0.05$) when compared to lean controls. The reduction of expiratory volumes is closely related to reduction of the MVV, and is a consequence of the increase in BMI. In group C volumes are severely reduced and are significantly different from group A ($P = 0.001$) (Table 1). However the Tiffeneau index (TI) remains constant in all groups and comparable to normal controls. As presented in Table 1 ERV is significantly reduced in all groups and follows the reduction of MVV.

FLOWS

Instant expiratory and inspiratory flows are reduced in obese subjects as long as MVV is reduced as a consequence of increased BMI (Table 1). The ratio of peak flow to MVV in obese patients is lower when compared to lean subjects, indicating a more important decrease of MVV than peak expiratory flow (PEF). The same was observed for instant flows at median and low volumes (Table 3).

The ratio between PEF and peak inspiratory flow (PIF), which is around 1 in lean subjects, increases significantly as BMI increases (Fig. 3, Table 4). The ratio between FEF50 and FIF50, however, does not change significantly in obese patients, and is comparable to normal lean subjects.

Discussion

The high percentage value of MVV found for the control group suggests that the predicted value for the Brazilian population must be different, and probably higher, than that proposed by Kory et al. (32) for male and by Lindall et al. (33) for female American subjects. However, as this is a constant error depending on a permanent factor, the results obtained may be interpreted and compared to the other parameters observed. It is evident that a study to determine the predicted MVV value for a Brazilian population should be done; this is already underway.

As observed in Fig. 1, an increase in BMI induces a significant reduction of MVV. In the control group, with BMI 20–29.9 kg m$^{-2}$ (mean ± sd = 24.79 ± 1.15 kg m$^{-2}$), the increase in body weight corresponds to a slightly greater MVV, perhaps indicating a better chest wall performance. However, in morbidly obese patients, increase
Table 3. Relationship between MVV and flows

<table>
<thead>
<tr>
<th></th>
<th>Lean</th>
<th>Group A</th>
<th>Group B</th>
<th>Group C</th>
</tr>
</thead>
<tbody>
<tr>
<td>MVV/FEF50</td>
<td>38±8 (3-29)</td>
<td>27±96 (1-43)³</td>
<td>29±52 (2-17)³</td>
<td>26±32 (2-26)³</td>
</tr>
<tr>
<td>MVV/FEF75</td>
<td>95±36 (7-18)</td>
<td>81±35 (4-97)</td>
<td>81±48 (7-82)</td>
<td>61±32 (5-77)³</td>
</tr>
<tr>
<td>MVV/PEF</td>
<td>94±17 (5-80)</td>
<td>90±71 (4-07)</td>
<td>90±1 (6-48)</td>
<td>87±61 (5-69)</td>
</tr>
<tr>
<td>MVV/FIF50</td>
<td>25±77 (3-38)</td>
<td>25±10 (1-70)</td>
<td>27±55 (92-01)</td>
<td>26±23 (2-74)</td>
</tr>
<tr>
<td>MVV/FIM</td>
<td>23±67 (3-51)</td>
<td>24±68 (1-32)</td>
<td>24±11 (1-60)</td>
<td>23±22 (2-20)</td>
</tr>
</tbody>
</table>

Results calculated from Table 1 and represent the mean ratio between MVV and the corresponding flow in each group. Data are mean (±SEM).

³P<0.05.

Fig. 3. Graphical representation of Table 4. Line represents the best fit for MVV, points representing mean ±sem in each group. Bars represent the relationship between peak expiratory/inspiratory flows (dashed) and median expiratory/inspiratory flows (crossed) in control group (N), obese with BMI 40–49·9 kg m⁻² (A), 50–59·99 kg m⁻² (B) and 60–69·99 kg m⁻² (C).

of BMI induces a significant corresponding decline of MVV. These results are in agreement with some others studies in the literature (10–14). As Dempsey et al. (15) and Kolias et al. (16) found a normal MVV in obese patients, we re-calculated the BMI of their subjects and found a mean around 44 for the former and 37 for the latter. It is therefore possible that they have studied patients who have not reached a BMI high enough to hinder the chest wall mechanics.

The MVV manoeuvre represents the integration of many factors in the pulmonary mechanics such as endurance, airway diameter and respiratory muscle strength (34–37), and is related to chronic airway obstruction (38). In our patients MVV, FVC and FEV₁ were correlated to BMI. In Fig. 2 it can clearly be observed that an increase of BMI corresponds to a decrease of the other three parameters. In contrast to the results found by Suratt et al. (9), we observed that in each obese group, FVC and FEV₁ are significantly reduced. This has also been described by others (2,4,24) and closely follows the decrease in MVV. This strengthens the point that FVC is reduced probably as consequence of the decrease of the elastic property of the chest wall, leading to diminution of its compliance (5) and, therefore, to the reduction of lung volumes (28); in this case, evaluated as reduction of MVV. FVC and FEV₁ also follow such a decrease. The Tiffeneau index, however, shows a comparable value among the groups and is comparable to that observed in normal patients. Therefore, the reduction of FEV₁ may just be a consequence of FVC reduction, which is also the result of MVV reduction due to lower chest wall compliance.

FEV₁ and MVV have been suggested to be indicative of obstructive impairment (36,37). However, the reduction of the expiratory flows could be considered as a result of the diminution of MVV. We therefore suggest that the reduction observed in FEV₁ is not due to an impairment or reduction of the bronchial diameter, but follows the reduction of MVV and the failure in chest wall mechanics, which is also a consequence of increased weight over the chest.

Inspiratory flows are influenced by the mechanical properties of the lung and the chest wall, in this case through inspiratory muscle strength (39). Decrease of inspiratory flows (Table 1) indicates an increase in air trapping, which is correlated to the reduction of MVV, following an increase in BMI. This is confirmed by the reduction of ERV in the same proportion in each group of obese patients. ERV has been described to be the most affected expiratory parameter in obese patients (14), which has been extensively confirmed (2–6,13,15,22,24–26), including results from our subjects. As ERV depends on respiratory muscles strength and chest wall compliance its decrease in obesity is due to the reduction of MVV in consequence of the increased loading of the chest wall (4). ERV reduction may be followed by a reduction in V/Q, which leads to a reduction of FVC (22,25). As our patients were not hypoventilated this is probably not the case in our study.

PEF is effort-dependent and is related to how fast pleural pressure can be increased at the beginning of the forced expiration (40). It is also dependent on the force applied to the chest wall and its elasticity (41). D’Angelo et al. (30,42) demonstrated that the decrease in lung elastic recoil accounts for PEF reduction, even in normal subjects. On the other hand the PIF depends on muscular drive, especially that of the diaphragm, and chest wall...
compliance. A significant reduction of PEF and PIF was observed in our obese subjects, especially in those with BMI higher than 50 kg m$^{-2}$. This closely follows the reduction of MVV and also supports the hypothesis that in obese patients the increased weight over the chest wall affects its mechanics, causing a significant reduction in MVV and, therefore, reduction in volumes and flows.

As seen in Table 4 the ratio between PEF and PIF increases in all group of obese patients with increase in BMI. This indicates that inspiratory drive is more affected in obese patients than expiratory mechanics, probably due to lower chest wall compliance. Further studies in diaphragmatic mechanics and trans-diaphragmatic pressures are necessary to confirm these suggestions.

The same increase, however, was not observed when the ratio of FEF50 : FIF50 was analysed, suggesting that the medium flows are less affected by lower compliance of the chest wall in obese subjects.

These results support the idea that the first consequence of increased mass over the chest in the morbid obesity is the reduction of chest wall compliance. This leads to a reduction of MVV, the consequence of which is lower values for FVC indicating a restrictive impairment. This becomes more frequent and more serious as BMI increases. As the reduction in FEV$_1$ and PEF follows that of FEV$_1$ with normal TI, conclude that these impairments are a consequence of the reduction of FVC and are not indicative of airways obstruction.

### Acknowledgements

The authors wish to thank Mr José Edilson Anastácio Rocha for his skilful technical assistance.

### References


### Table 4. Relationship between maximal expiratory and maximal inspiratory flows as well as median expiratory and inspiratory instant flows in obese individuals and control leans subjects

<table>
<thead>
<tr>
<th></th>
<th>Lean</th>
<th>Group A</th>
<th>Group B</th>
<th>Group C</th>
</tr>
</thead>
<tbody>
<tr>
<td>PEF/PIF</td>
<td>1.08 (0.06)</td>
<td>1.27 (0.06)</td>
<td>1.33 (1.00)</td>
<td>1.49 (0.09)</td>
</tr>
<tr>
<td>FEF50/FIF50</td>
<td>1.00 (0.06)</td>
<td>0.98 (0.05)</td>
<td>0.99 (0.08)</td>
<td>0.96 (0.06)</td>
</tr>
</tbody>
</table>