Respiratory system mechanics in sedated, paralyzed, morbidly obese patients

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Respiratory system mechanics in sedated, paralyzed, morbidly obese patients [1].

Appl. Physiol. 82(3): 811–818, 1997.—The effects of inspiratory flow and inflation volume on the mechanical properties of the respiratory system in eight sedated and paralyzed postoperative morbidly obese patients (aged 37.6 ± 11.8 yr who had never smoked and had normal preoperative seated spirometry) were investigated by using the technique of rapid airway occlusion during constant-flow inflation. With the patients in the supine position, we measured the interrupter resistance (Rint,rs), which in humans probably reflects airway resistance, the “additional” resistance (ΔRrs) due to viscoelastic pressure dissipation and time-constant inequalities, and static respiratory elastance (Est,rs). Intra-abdominal pressure (IAP) was measured by using a bladder catheter, and functional residual capacity was measured by the helium-dilution technique. The results were compared with a previous study on 16 normal anesthetized paralyzed humans. Compared with normal persons, we found that in obese subjects: 1) functional residual capacity was markedly lower (0.645 ± 0.208 liter) and IAP was higher (24 ± 2.2 cmH2O); 2) alveolar-arterial oxygenation gradient was increased (178 ± 59 mmHg); 3) the volume-pressure curve of the respiratory system was curvilinear with an “inflection” point; 4) Est,rs, Rint,rs, and ΔRrs were higher than normal (29.3 ± 5.04 cmH2O/l, 5.9 ± 2.4 cmH2O·l−1·s, and 6.4 ± 1.6 cmH2O·l−1·s, respectively); 5) Rint,rs increased with increasing inspiratory flow, Est,rs did not change, and ΔRrs decreased progressively; and 6) with increasing inflation volume, Rint,rs and Est,rs decreased, whereas ΔRrs rose progressively. Overall, our data suggest that obese subjects during sedation and paralysis are characterized by hypoxemia and marked alterations of the mechanical properties of the respiratory system, largely explained by a reduction in lung volume due to the excessive unopposed IAP.

Morbid obesity; anesthesia and paralysis; functional residual capacity; intra-abdominal pressure

AWAKE MORBIDLY OBESE PATIENTS present severe alterations of respiratory mechanics, particularly an increase in respiratory elastance (30) and resistance (41).

General anesthesia and paralysis, even in normal subjects, are associated with alterations in respiratory function, such as increased respiratory elastance, presence of atelectasis, and hypoxemia (8, 20). It has been suggested that these alterations may be pronounced in morbidly obese people (36, 37, 38), who do, in fact, have a substantially increased risk of perioperative respiratory complications (19, 27).

Nevertheless, there are relatively few reports dealing with the changes in mechanical properties of the respiratory system in these patients during general anesthesia and muscle paralysis (22).

In this study we used the technique of rapid airway occlusion during constant-flow (V) inflation (5, 11, 13, 15) to investigate the following in sedated, paralyzed, morbidly obese patients: 1) the effects of V, volume (ΔV), and time on the mechanical properties of the respiratory system, i.e., elastance and resistance, and 2) the adaptability of the spring and dashpot model proposed by Bates et al. (3) for interpreting respiratory mechanics. We also measured functional residual capacity (FRC) and intra-abdominal pressure to provide a further picture of the changes in respiratory system mechanics induced by sedation and paralysis in these patients. We compared our data with those reported by D’Angelo et al. (11) in normal anesthetized paralyzed subjects using the same technique.

METHODS

The investigation was approved by the institutional ethics committee, and informed consent was obtained preoperatively from each patient.

There were eight patients (4 men and 4 women) scheduled for general anesthesia for ileojejunal bypass or gastric binding. None had any history of smoking or clinical evidence of cardiopulmonary disease. Their preoperative pulmonary functional tests, done with the patients in the seated position, gave the following values: average vital capacity (VC) was 4.3 ± 1.2 liters (96.9 ± 7% of predicted), forced expiratory volume in 1 s (FEV1) was 3.3 ± 0.9 liters (91.7 ± 8.7% of predicted), and FEV1/VC was 80 ± 5.8% (98.8 ± 8.1% of predicted). Mean age and body mass index were 37.6 ± 11.8 yr (range 20–55 yr) and 48.7 ± 7.8 kg/m2 (range 40.7–64.8 kg/m2), respectively.

Anesthesia was induced with intravenous thiopental sodium (5–7 mg/kg). Muscle relaxation to facilitate endotracheal intubation was provided with succinylcholine (1 mg/kg), and paralysis was maintained with pancuronium bromide. Patients were intubated with a Portex cuffed endotracheal tube (6.5–7.5 mm ID) and mechanically ventilated (tidal volume 8–10 ml/kg of ideal body weight, respiratory rate 11–14 breaths/min). Anesthesia was maintained with 0.5–1% isoflurane in O2:N2O (50:50%). The average time of the surgical procedure was 128 ± 45 min. After surgery, the N2O was withdrawn and patients were transferred to our intensive care unit (ICU) still sedated and paralyzed. The time elapsed from the end of the surgical procedure and the beginning of the protocol ranged between 1 and 2 h. The entire study was performed in the ICU. During the study, all subjects were sedated with intravenous diazepam (0.1–0.2 mg/kg) and paralyzed with pancuronium bromide (0.1–0.2 mg/kg). Mechanical ventilation was provided with a Siemens Servo Ventilator 900 C ventilator. The baseline ventilator setting during the study was as follows: tidal volume of 0.72 ± 0.04 liter and inspiratory flow of 0.66 ± 0.04 l/s with a mean
inspired O2 fraction of 44 ± 9% and balance N2 (air supplemented with O2). The inspired O2 fraction was set to maintain arterial O2 saturation higher than 90% and respiratory frequency to maintain normocapnia (12.4 ± 1.8 breaths/min). The duration of expiration averaged 3.81 ± 0.72 s (range 2.88 to 5.04 s). Arterial P O2 and PC O2 averaged 98 ± 15 and 36.3 ± 4 Torr, respectively.

Physiological measurements. All measurements were obtained in supine patients with zero end-expiratory pressure and no abdominal bandage. The baseline setting for each subject was kept constant throughout the experiment.

Intra-abdominal pressure. Intra-abdominal pressure was measured by using a transurethral bladder catheter inserted in the bladder before the induction of anesthesia and kept both during the surgical operation and throughout the study. The technique has been previously reported (26) and is summarized here. With use of a sterile technique, an average of 100 ml of normal saline was infused through the urinary catheter to fill the catheter tubing. The catheter was then clamped distally to the sampling membrane. A 20-gauge needle was inserted through the catheter sampling membrane, and the bladder catheter pressures were transduced by using arterial tubing to the pressure transducer (Bentley Trantec, Bentley Laboratories, Irvine, CA). Transducers were zeroed at the pubis. The mean pressures at end expiration were recorded.

FRC. FRC was measured by using a simplified closed-circuit helium-dilution method (10, 32). An anesthesia bag filled with 2 liters of a known gas mixture (13% helium in oxygen) was connected to the airway opening at end-expiration, and 15 deep manual breaths were made. The helium concentration in the anesthesia bag was then measured by using a helium analyzer (PK Morgan Chatham, Kent, UK), and FRC was computed according to the following formula: $FRC = \frac{Vi[He]}{[He]f - Vi}$, where $Vi$ is the initial gas volume in the anesthesia bag and [He] and [He] are the initial and final helium concentrations in the bag, respectively.

Respiratory mechanics. $V$ was measured with a heated pneumotachograph (Fleish no. 2) inserted between the endotracheal tube and the Y piece of the ventilator. It was connected to the breathing circuit by a cone and to a differential pressure transducer (Validyne MP 45 ± 2 cmH2O, Northridge, CA). The equipment dead space (not including the endotracheal tube) was 80 ml. Volume was obtained by digital integration of the flow signal. The response of the pneumotachograph, calibrated with the same gas mixture used during measurements, was linear over the experimental range of flows. Airway pressure (Pao) was measured proximal to the orotracheal tube by a pressure transducer (Bentley Trantec, Bentley Laboratories, Irvine, CA). The pressure-flow relationships of the orotracheal tubes were determined after each experiment by using the experimental gas mixture as described by Behrakis et al. (7) to compute the resistive pressure of the endotracheal tubes for any given flow tested. All variables were recorded on a four-channel pen recorder (Gould Brush 2400S, Gould, Cleveland, OH) and processed by an analog-to-digital converter to an IBM-compatible personal computer for storage and computations.

Respiratory mechanics were assessed by the constant-$V$ end-inspiratory occlusion method (5, 11, 13, 15). After the occlusion, there was an immediate drop in Pao from a maximal value ($P_{\text{max}}$) to a lower value ($P_{1}$), followed by a gradual decrease, until an apparent plateau ($P_{2}$) was achieved. These plateau pressures were reached in 3–4 s, so Pao at 4 s was taken as the static end-inspiratory elastic recoil pressure of the total respiratory system (Pst,rs). During this period the reduction in pressure due to volume loss from continuing gas exchange should be negligible (11).

The immediate and slow changes in Pao were obtained by computed fitting curves, as previously described (4). The initial drop in Pao corrected for the resistive pressure drop as the result of the endotracheal tube ($P_{\text{max}} - P_{1}$), where $P_{\text{max}}$ is the peak airway pressure corrected for the endotracheal tube resistance, divided by the immediately preceding steady $V$ provides the interrupter resistance (Rint,rs). In the calculation of the Rint,rs, correction was made for the errors due to the closing time of the ventilator valve, as previously described (4). The slow decay of pressure ($P_{2} - P_{1}$) divided by the V preceding the occlusion yields the effective “additional” resistance ($\Delta R_{\text{rs}}$) as previously reported (5, 11, 13, 15). The sum of Rint,rs and $\Delta R_{\text{rs}}$ presents the total resistance of the respiratory system (Rs). $P_{2}$ (Pst,rs) divided by $\Delta V$ provides the static elastance of the total respiratory system ($E_{\text{st,rs}}$), and $P_{1}$ divided by $\Delta V$ gives the dynamic elastance ($E_{\text{dy,rs}}$).

Two sets of experiments were carried out in each patient. 1) In the iso-$\Delta V$ experiment, $\Delta V$ was kept constant at its baseline value and test breaths were performed by randomly changing the inspiratory $V$ for one breath from the basal setting to values of 0.2, 0.4, 0.6, 0.8, and 1 l/s. This was done by regulating the inspiratory time (TI) with the appropriate button on the ventilator. 2) In the iso-$V$ experiment, the basal $V$ was kept constant, and $\Delta V$ was changed for single breaths from the basal setting to values of 0.2, 0.4, 0.6, 0.8, and 1 liter in random order by changing the respiratory frequency on the ventilator. This part of the experiment served to compute the volume-pressure curve of the respiratory system. At each test breath, end-inspiratory occlusion lasting 4–5 s was performed by pressing the end-inspiratory hold button of the ventilator. After each test breath, baseline ventilation was resumed until volume, flow, and pressure records returned to baseline values.

Model fitting. Our data were analyzed in terms of the model of the respiratory system of Bates et al. (3). In its simplest form this consists of two compartments in parallel: a dashpot representing Rint,rs and a Kelvin body (Fig. 1). The latter consists of a spring representing the E_{st,rs} in parallel with a Maxwell body, i.e., a spring ($E_{2}$) and a dashpot ($R_{2}$) arranged in series.
serially, E₂ and R₂ represent the viscoelastic properties of the tissues of the lung and chest wall, and Est,rs and Rint,rs are standard elastic and resistive components, respectively. This four-element model predicts that during constant-V˙ inflation, ΔRrs should increase with Ti according to the following exponential function (11, 13, 15)

$$\Delta R_{rs} = R_2(1 - e^{-\frac{\text{Ti}}{\tau_2}})$$  \hspace{1cm} (1)

where R₂ is the resistance and τ₂ is the time constant of the Maxwell body (\(= R_2/E_2\)). Equation 1 is based on the assumption that ΔRrs reflects only the viscoelastic behavior. Measurements of ΔRrs, however, may comprise contributions due to time-constant inequalities within the lung (pendelluft) and/or chest wall (28, 31). Such contributions may be taken into account by adding a constant (A) to Eq. 1 (15)

$$\Delta R_{rs} = A + R_2(1 - e^{-\frac{\text{Ti}}{\tau_2}})$$  \hspace{1cm} (2)

Constants A, R₂, and τ₂ were assessed by fitting Eq. 2 to the experimental data of ΔRrs vs. Ti. The time constant of the exponential was allowed to vary independently. Because during constant-V˙ inflation Ti = AV/V, Eq. 2 can be rewritten

$$\Delta R_{rs} = A + R_2(1 - e^{-\frac{\Delta V}{V/V_\text{˙}}})$$  \hspace{1cm} (3)

This implies that at a fixed ΔV ΔRrs will decrease with increasing V, whereas at a constant V ΔRrs will increase exponentially with ΔV.

Statistical analysis. Analysis of regression was done by using the least squares method. Morbidly obese patients and 16 normal anesthetized paralyzed subjects (11) were compared by using Student's unpaired t-test (2). P < 0.05 was accepted as statistically significant. Values are means ± SD.

RESULTS AND DISCUSSION

FRC. The average FRC was 0.645 ± 0.208 liter in these obese subjects during sedation and paralysis, which is consistent with previous observations in the same kind of patients (10). This is markedly lower than values reported during anesthesia and mechanical ventilation in normal subjects (1.8 and 2.3 liters with body plethysmography (21, 40), 2.6 liters with nitrogen washout (24), 1.7 and 1.9 liters with the helium dilution technique, as in the present study (25, 32)).

Differences in FRC might be related not only to body habitus but also to the inappropriateness of this measurement technique in obese patients, to the effects of duration of anesthesia (all patients were studied 2 h after induction of anesthesia), and to the effects of abdominal surgery.

FRC measurements may likewise have been affected by airway closure or bag shrinkage. Airway closure may interfere with correct mixing of helium between the bag and the lung according to three possibilities: 1) the closed airways do not open in any phase of bag ventilation; 2) at the first inflation the closed airways open, but during the next expiration they close and remain closed during further inflations, so part of the helium is trapped and not recollected into the bag; and 3) at each inflation the airways open during inspiration and close during expiration.

In the first case FRC will be underestimated. However, this was unlikely in our study because the closed airways usually reopen at inspiratory transpulmonary pressures in a range of 12–20 cmH₂O (14, 18). By using deep breaths, as we did, the range of inspiratory pressures reached is definitely higher.

Nevertheless, part of the helium may remain trapped after the first inflation or at each inflation during expiration, but in all these cases our FRC would be overestimated rather than underestimated.

Another technical problem derives from gas volume loss during rebreathing due to the gas exchange. However, because this loss should be counteracted by the increase in volume due to temperature and humidity changes, the total volume (bag + lung) probably remains constant (9, 17).

The duration of anesthesia and the abdominal surgery should not have affected FRC measurements. Lung volume reportedly does not progressively decrease in normal persons (25) or in obese subjects (10) with the duration of anesthesia. It is also not influenced by the abdominal surgery, at least in obese patients (10).

Several mechanisms have been proposed to explain the reduction in FRC after induction of anesthesia and paralysis in normal persons, including atelectasis and airway closure in the dependent part of the lung, increased thoracic blood volume, or a change in surfactant function (34). Reduced lung volume in obese patients during sedation and paralysis results in airway closure above a tidal volume, whereby the dependent parts of the lung may not participate in gas exchange on account of atelectasis (23).

The formation of atelectasis during anesthesia, documented by computed-tomographic studies (8, 34, 38), and the presence of airway closure, may both be due to the lung weight (8) and to a loss of muscle tone, which in turn causes a cephalad displacement of the diaphragm in response to unopposed intra-abdominal pressure (16, 24, 29). The same mechanisms operating in obese persons, in whom the intra-abdominal pressure is presumably higher because of the greater abdominal content, could lead to a larger decrease in lung volume because of more formation of atelectasis and airway closure (38). In fact, in five of our obese subjects, mean intra-abdominal pressure was 24 ± 2.2 cmH₂O, markedly higher than reported in normal patients during the first 24 h after abdominal surgery (6.8 ± 3.7 cmH₂O) (26). Additionally, our obese subjects had an increased alveolar-arterial oxygenation gradient, on average 178 ± 59 mmHg. This may be due to reduced FRC, with consequent pulmonary atelectasis or airway closure occurring within a tidal breath (23).

Volume-pressure curve of the respiratory system and Est,rs. The average volume-pressure curves in normal and obese subjects are shown in Fig. 2 and could be fitted, for all patients, to a power function of the type

$$P_{st,rs} = a' \cdot \Delta V^b$$  \hspace{1cm} (4)

where a' is the pressure at ΔV of 1 liter and b' is a dimensionless number. The average coefficients a' and b' in obese patients were, respectively, 24.9 ± 4.6 and 0.65 ± 0.09 cmH₂O/l, significantly different (P < 0.01)
from those reported in normal subjects ($a' = 14 \pm 2$ and $b' = 0.95 \pm 0.01$ cmH$_2$O/l).

These data indicate an overall higher elastance in obese subjects and the presence of an "inflection point," or "zone," at pressures between 5 and 15 cmH$_2$O, after which the volume increases linearly with pressure. In normal subjects no inflection zone was identified. In accordance with the pattern of the volume-pressure curves, the $E_{st,rs}$ measured at fixed flow during baseline ventilation is higher ($P < 0.01$) in obese patients than in normal subjects (29.3 ± 5.04 vs. 14.5 ± 2.1 cmH$_2$O/l; Table 1). More importantly, whereas $E_{st,rs}$ in normal subjects did not change with increasing volume (straight volume-pressure curve), in obese patients it dropped sharply along the inflection zone (from 0 to 0.6 liter), subsequently remaining roughly constant (Fig. 3A). As expected (11), $E_{st,rs}$ was not influenced by $V$ (Fig. 3B).

Increased stiffness of the respiratory system, i.e., a higher $E_{st,rs}$, has already been described in conscious obese persons (30). Possible causes include increased stiffness of the chest wall and/or lung parenchyma or a decrease in overall lung volume. Naimark and Cherniak (30) found abnormal chest wall elastance in conscious obese subjects and attributed the increase to the greater adiposity around the ribs, diaphragm, and abdomen or to limited movement of the ribs caused by thoracic kyphosis and lumbar hyperlordosis from excessive abdominal fat content (30, 36, 37). However, Suratt et al. (35) found no alterations to chest wall elastance.

Whatever the real behavior of the chest wall elastance, the lung volume reduction we observed might explain the higher elastance of the respiratory system. It is well established, in fact, that $E_{st,rs}$ depends on lung volume (the lower the volume, the higher the elastance) (1). Moreover, the possible negative effects of anesthesia duration on $E_{st,rs}$ can be reasonably ruled out because several authors found no deterioration in respiratory mechanics during the course of anesthesia after induction (6). The changes in $E_{st,rs}$ with increasing pressure and volume in obese subjects may be interpreted as a recruitment of atelectatic lung regions.

Table 1. Standard respiratory mechanics data of morbidly obese patients

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>$R_{rs}$, cmH$_2$O·l·s$^{-1}$</th>
<th>$R_{int,rs}$, cmH$_2$O·l·s$^{-1}$</th>
<th>$\Delta R_{rs}$, cmH$_2$O·l·s$^{-1}$</th>
<th>$E_{st,rs}$, cmH$_2$O/l</th>
<th>$\tau_{rs}$</th>
<th>$R_{int,rs}/E_{st,rs}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>9.1</td>
<td>2.7</td>
<td>6.4</td>
<td>31.1</td>
<td>0.09</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>14.5</td>
<td>9.2</td>
<td>5.3</td>
<td>28.6</td>
<td>0.32</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>7.9</td>
<td>3.4</td>
<td>4.6</td>
<td>23.9</td>
<td>0.14</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>12.4</td>
<td>4.2</td>
<td>8.2</td>
<td>40.4</td>
<td>0.10</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>18</td>
<td>8.7</td>
<td>9.3</td>
<td>29.9</td>
<td>0.29</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>17.7</td>
<td>5.2</td>
<td>12.5</td>
<td>27.3</td>
<td>0.29</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>12.3</td>
<td>6.7</td>
<td>5.6</td>
<td>25.9</td>
<td>0.26</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>12.6</td>
<td>7.5</td>
<td>5.1</td>
<td>26.9</td>
<td>0.28</td>
<td></td>
</tr>
<tr>
<td>Mean ± SD</td>
<td>12.3±3.1</td>
<td>5.9±2.4</td>
<td>6.4±1.6</td>
<td>29.3±5.0</td>
<td>0.21±0.09</td>
<td></td>
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</tbody>
</table>
Increasing pressure recruits the atelectasis due to the displacement of the diaphragm, i.e., counteracting the previously unopposed intra-abdominal pressure (16, 24, 29). This recruitment of atelectatic regions may sharply change elastance along the inflection zone, after which the increase in inflation volume does not really decrease $E_{st,rs}$, as the recruitment is very likely complete.

$R_{int,rs}$. In obese subjects, $R_{int,rs}$ was three times that in normal subjects ($5.9 \pm 2.4$ and $2.3 \pm 0.5$ cmH$_2$O·l$^{-1}$·s$^{-1}$, respectively, at comparable $V$ and $\Delta V$; Table 1), as previously reported in awake, seated, obese patients using body plethysmography ($5 \pm 0.05$ cmH$_2$O·l$^{-1}$·s$^{-1}$) (41), and sharply decreased with increasing volume ($P < 0.01$, Fig. 4A). Consequently, the conductance (G$_{int,rs}$), i.e., $1/R_{int,rs}$, increased with volume, according to the following equation

$$G_{int,rs} = a + b \cdot \Delta V$$

(5)

where $a$ is the conductance at FRC and $b$ is the change in conductance per unit change in $V$. The average values of $a$ and $b$ in obese patients were $-0.005 \pm 0.11$ l·cmH$_2$O$^{-1}$·s$^{-1}$ and $0.35 \pm 0.34$ cmH$_2$O$^{-1}$·s$^{-1}$, respectively, which are significantly ($P < 0.01$) different from normal values ($0.39 \pm 0.09$ l·cmH$_2$O$^{-1}$·s$^{-1}$ and $0.15 \pm 0.09$ cmH$_2$O$^{-1}$·s$^{-1}$).

While $R_{int,rs}$ decreased with increasing volume, at the same tidal volume (iso-$\Delta V$) it rose dramatically with flow, compared with normal (Fig. 4B). Expressing the $R_{int,rs}$-flow relationship with Rohrer’s equation

$$R_{int,rs} = K_1 + K_2 \cdot \dot{V}$$

(6)

we found an average $K_1$ (“laminar” component) of $1.08 \pm 3.24$ cmH$_2$O·l$^{-1}$·s$^{-1}$, which is similar to normal ($K_1 = 1.94 \pm 0.51$ cmH$_2$O·l$^{-1}$·s$^{-1}$), whereas $K_2$, the “turbulent” component, was significantly ($P < 0.01$) increased ($8.1 \pm 6.3$ and $0.52 \pm 0.08$ cmH$_2$O·l$^{-1}$·s$^{-2}$, respectively).

The $R_{int,rs}$ in anesthetized humans usually reflects airway resistance, although it may include an “ohmic” component of the chest wall, $-27\%$ according to D’Angelo et al. (12). $R_{int,rs}$ not only represents the interrupter resistance of the lung and chest wall in series but also parallel “gaps” in homogeneity within the lungs. With these limitations in mind, our data suggest that the lung volume reduction plays a major role in the increase in $R_{int,rs}$. Airway caliber is closely related to lung volume (39), and a large reduction in caliber, due to the smaller lung volume, could account
for an increased turbulent component, i.e., higher K₂, observed at a rate that only minimally affects airway resistance in normal persons. Other possible factors, such as recruitment of previously collapsed lung regions, during tidal volume may be involved in the increase in Rint,rs, but, at the moment, this remains a matter of speculation.

△Rs and Rs. △Rs at the baseline ventilator setting was significantly (P < 0.01) higher than normal (6.4 ± 1.6 and 2.8 ± 0.6 cmH₂O·l⁻¹·s, respectively; Table 1), and it was markedly dependent on V and △V, decreasing with V and increasing with △V (Figs. 5A and 6A). Consequently, Rs was significantly (P < 0.01) increased in obese subjects (12.3 ± 3.1 compared with 5.1 ± 0.9 cmH₂O·l⁻¹·s for normal patients; Table 1), but it was neither V nor △V dependent (Figs. 5B and 6B).

The increase in △Rs in our patients might reflect higher time-constant inequalities within the respiratory system and/or altered stress-adaptation properties of the thoracic tissues (lung and chest wall). The reduction in lung volume itself may increase △Rs (11). Unfortunately, the end-inspiratory occlusion method does not distinguish between stress-adaptation phenomena and time-constant differences. However, during mechanical ventilation, in obese patients the distribution of ventilation is markedly uneven (22) and, although the structures responsible for the viscoelastic behavior of the respiratory system are not known, mechanical alterations of both lung and chest wall may contribute to the increase in △Rs (13).

Spring and dashpot model of respiratory mechanics. We found that the spring and dashpot model presented in Fig. 1 can be applied not only to normal subjects (11, 13) but also to morbidly obese, sedated, paralyzed patients. In all patients, the relationship between △Rs and Ti closely fitted (P < 0.01) the theoretical exponential function (Eq. 2, Fig. 7). The individual values for the constants in Eq. 2 in obese subjects are listed in Table 2, which also provides the E₂ (R₂/τ₂) values. Both R₂ and τ₂ were significantly (P < 0.01) higher than in normal anesthetized paralyzed persons (18.3 ± 6.9 vs. 5.9 ± 1.8 cmH₂O·l⁻¹·s and 4.4 ± 2.9 vs. 1.3 ± 0.3 s, respectively). E₂, however, was close to normal (5.6 ± 3.0 vs. 4.5 ± 0.9 cmH₂O/l). The values of A were small and not significantly different from zero. Moreover, according to the model in Fig. 1, △Rs increased exponentially with △V and decreased with V, as expected from Eq. 3. In obese patients, however, the standard time constant of the respiratory system (= Rint,rs/Est,rs) was 0.21 ± 0.09 s (Table 1), which is slightly

![Graph](image)

**Table 2. Viscoelastic constants of morbidly obese patients**

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>A₂ (cmH₂O·l⁻¹·s⁻¹)</th>
<th>R₂ (cmH₂O·l⁻¹·s)</th>
<th>E₂ (cmH₂O/l)</th>
<th>τ₂ (s)</th>
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<td>1</td>
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<td>5.25</td>
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<td>15.3</td>
<td>9.0</td>
<td>1.70</td>
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<td>2.7</td>
<td>8.65</td>
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<td>8.55</td>
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<td>0.9</td>
<td>11.2</td>
<td>4.8</td>
<td>2.36</td>
</tr>
<tr>
<td>Mean ± SD</td>
<td>1.5 ± 1.3</td>
<td>18.3 ± 6.9</td>
<td>5.6 ± 3.0</td>
<td>4.38 ± 2.92</td>
</tr>
</tbody>
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A, R₂, and τ₂, constants computed according to Eq. 2 by using all iso-volume and iso-flow data; E₂, R₂/τ₂. Relationships were highly significant for all patients, r > 0.99, P < 0.01.
higher than normal (0.14 – 0.17 s) (11). Although the nature of the increase in $R_2$ and $t_2$ in obese patients is not known and the anatomic correlates of $R_2$ and $E_2$ have yet to be determined, up to the volume investigated $\Delta R_{rs}$ was consistent with the linear viscoelastic model shown in Fig. 1.

Conclusions. In our patients, the lung structure itself is very likely healthy, preoperative pulmonary function tests being substantially normal and there being no evidence of previous cardiac diseases. During sedation and paralysis, this healthy lung presumably undergoes atelectasis and/or airway closure in the dependent part because of displacement of the diaphragm caused by the elevated unopposed intra-abdominal pressure.

The reduction in lung volume, due to atelectasis formation and/or airway closure, may explain the increased elastance of the respiratory system. Moreover, the lung volume reduction is associated with a reduction in airway caliber, resulting in increased ohmic resistance. At higher tidal volumes, the inflation pressures rise too, at least partly counteracting the elevated intra-abdominal pressure. This probably causes increasing recruitment of previously collapsed areas and/or a reopening of closed airways, a decrease in dynamic and static elastance of the respiratory system, and a reduction in the ohmic resistance.

Overall, our data suggest that the main cause of the mechanical alterations of the respiratory system in obese subjects during sedation and paralysis is a reduction in lung volume due to excessive unopposed intra-abdominal pressure.

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