The Prone Positioning During General Anesthesia Minimally Affects Respiratory Mechanics While Improving Functional Residual Capacity and Increasing Oxygen Tension

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We investigated the effects of the prone position on the mechanical properties (compliance and resistance) of the total respiratory system, the lung, and the chest wall, and the functional residual capacity (FRC) and gas exchange in 17 normal, anesthetized, and paralyzed patients undergoing elective surgery. We used the esophageal balloon technique together with rapid airway occlusions during constant inspiratory flow to partition the mechanics of the respiratory system into its pulmonary and chest wall components. FRC was measured by the helium dilution technique. Measurements were taken in the supine position and after 20 min in the prone position maintaining the same respiratory pattern (tidal volume 10 mL/kg, respiratory rate 14 breaths/min, F_{IO_2} 0.4). We found that the prone position did not significantly affect the respiratory system compliance (80.9 ± 16.6 vs 75.9 ± 13.2 mL/cm H_2O) or the lung and chest wall compliance. Respiratory resistance slightly increased in the prone position (4.8 ± 2.5 vs 5.4 ± 2.7 cm $H_2O \cdot L^{-1} \cdot s$, $P < 0.05$), mainly due to the chest wall resistance (1.3 ± 0.6 vs 1.9 ± 0.8 cm $H_2O \cdot L^{-1} \cdot s$, $P < 0.05$). Both FRC and Pao_2 markedly ($P < 0.01$) increased from the supine to the prone position (1.9 ± 0.6 vs 2.9 ± 0.7 L, $P < 0.01$, and 160 ± 37 vs 199 ± 16 mm Hg, $P < 0.01$, respectively), whereas $Paco_2$ was unchanged. In conclusion, the prone position during general anesthesia does not negatively affect respiratory mechanics and improves lung volumes and oxygenation.

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The prone position is used widely in specific surgical indications (1) and to improve oxygenation in patients with acute respiratory failure (2). Despite this wide use in mechanically ventilated patients, the modifications in respiratory mechanics and gas exchange during anesthesia in the prone position have not been extensively investigated.

Lynch et al. (3) found that anesthetized patients in the prone position, breathing spontaneously, were unable to maintain an adequate minute volume and oxygenation. Moreover, a reduction of 20%–30% in the compliance of the respiratory system and an increase in peak airway pressure has been found in anesthetized and paralyzed patients when they were turned to the prone position (3,4). These authors concluded that the reduction in compliance was mainly due to a decrease in chest wall elasticity, and that the prone position appeared to have an adverse effect on the mechanics of breathing in anesthetized patients.

In a recent study performed in normal awake subjects, Lumb and Nunn (5) found that the prone position did not markedly affect respiratory function and may increase functional residual capacity (FRC). The authors suggested that the reduction in FRC and closing capacity with anesthesia is similar to that seen with patients supine, and with less disturbance of gas exchange compared to the supine position.

The aim of this study was to investigate changes in respiratory mechanics (partitioned into its lung and chest wall components), gas exchange, and lung volume modifications between the supine and prone positions in a group of anesthetized and paralyzed patients.

Methods

We studied a group of 17 consecutive patients (9 male, age 43 ± 14 yr, body mass index 23.2 ± 2.6 kg/m^{-2}) receiving general anesthesia for elective surgery requiring the prone position (removal of a herniated

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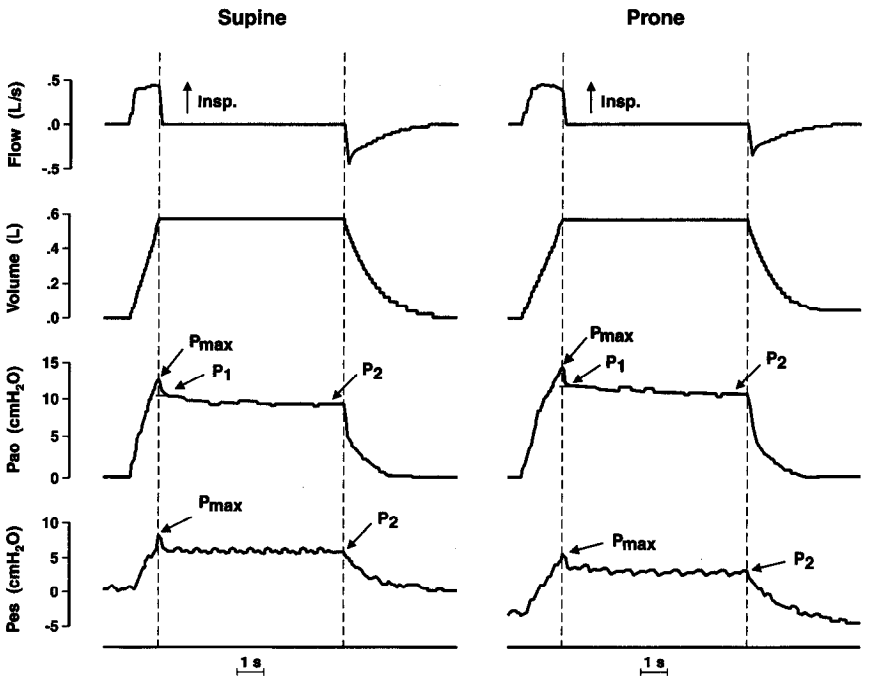

Figure 1. Tracings (top to bottom) of flow, volume, pressure at the airway opening (Pao), and esophageal pressure (Pes) from a representative patient in supine and prone position. During an end-inspiratory occlusion there was an immediate decrease in Pao from a maximum pressure value (P_{max}) to a lower value (P_1), followed by a slow decay to a plateau pressure (P_2), that represented an end-inspiratory elastic recoil of the respiratory system. In Pes no immediate decrease was appreciable from P_{max} to P_1. Plateau pressure in Pes represents the end-inspiratory elastic recoil of the chest wall. Insp. = inspiration.

disk); all were free from cardiorespiratory disease. The research was approved by our internal ethics committee, and verbal informed consent was obtained from all patients.

All patients were premedicated with diazepam 10 mg and atropine 0.5 mg. Anesthesia was induced with fentanyl 0.10 mg and propofol 2 mg/kg intravenously; muscle relaxation was obtained with vecuronium bromide 0.1 mg/kg. Patients were orotracheally intubated with a reinforced cuffed tube (7.5–8.0 mm internal diameter) positioned under direct laringoscopy and then connected to a mechanical ventilator (Servo 900 C; Siemens, Berlin, Germany), using the control mode ventilation with constant inspiratory flow; the ventilator setting consisted of a fixed respiratory rate of 14 breaths/min, an inspiratory to expiratory time ratio of 1:2, a tidal volume (V_T) of 10 mL/kg, and an inspired oxygen fraction (F_{IO_2}) of 40%. Anesthesia was maintained with continuous intravenous infusion of propofol (6–12 $mg \cdot kg^{-1} \cdot h^{-1}$) and fentanyl as needed. Electrocardiogram and blood pressure were clinically monitored throughout the procedure.

The FRC was measured at end-expiration using a simplified closed-circuit helium dilution method (6). Briefly, an anesthesia bag filled with 2 L of a known gas mixture (13% helium in oxygen) was connected to the airway opening at end-expiration and 10 deep manual breaths were performed. The helium concentration in the anesthesia bag was then measured with a helium analyzer (PK Morgan Ltd., Chatham, Kent, England) and FRC was computed according to the following formula:

$$FRC = V_i[He]_i/[He]_{fin} - V_i,$$

where V_i is the initial gas volume in the anesthesia bag and $[He]_i$ and $[He]_{fin}$ are the initial and final helium concentrations, respectively, in the anesthesia bag.

Airway pressure (Pao) was measured proximal to the endotracheal tube by means of a polyethylene catheter (2 mm internal diameter, 120 cm long), connected to a Bentley Trantec® pressure transducer (Bentley Lab., Irvine, CA). Esophageal pressure (Pes) was measured with an esophageal balloon (Bicore CP-100, Irvine, CA) modified to allow connection to a Bentley Trantec transducer; during measurements the balloon was inflated with 0.5–1 mL of air. Before induction of anesthesia the validity of Pes was verified using the "occlusion test" method proposed by Baydur et al. (7), and the balloon fixed in that position. The occlusion test was repeated in the prone position at the end of surgery, when patients resumed spontaneous breathing.

Gas flow was recorded with a heated pneumotachograph (Fleish no. 2) connected to a Validyne MP 45-1® differential pressure transducer (Validyne Corp., Northridge, CA). Volume was obtained by digital integration of the flow signal. Both flow and pressure

Table 1. Total Respiratory System, Lung, and Chest Wall Mechanics in the Supine and Prone Positions

	Supine	Prone	*P*
Cst,rs (mL/cm H_2O)	80.9 ± 16.6	75.9 ± 13.2	NS
Cst,w (mL/cm H_2O)	203.2 ± 72.4	184.3 ± 77.1	NS
Cst,L (mL/cm H_2O)	150.0 ± 52.3	142.5 ± 36.7	NS
R_{max},rs (cm $H_2O \cdot L^{-1} \cdot s$)	4.8 ± 2.5	5.4 ± 2.7	<0.05
DR,rs (cm $H_2O \cdot L^{-1} \cdot s$)	2.7 ± 1.1	3.2 ± 0.9	<0.01
R_{max},L (cm $H_2O \cdot L^{-1} \cdot s$)	3.5 ± 2.3	3.5 ± 2.5	NS
R_{min},L (cm $H_2O \cdot L^{-1} \cdot s$)	2.1 ± 1.8	2.2 ± 1.8	NS
DR,L (cm $H_2O \cdot L^{-1} \cdot s$)	1.3 ± 1.1	1.3 ± 1.0	NS
R_{max},w (cm $H_2O \cdot L^{-1} \cdot s$)	1.3 ± 0.6	1.9 ± 0.8	<0.05

Data are expressed as mean ± SD.
Cst,rs = respiratory system compliance; Cst,w = chest wall compliance; Cst,L = lung compliance; R_{max},rs = maximum resistance of the respiratory system; DR,rs = "additional" resistance of the respiratory system; R_{max},L = maximum resistance of the lung; R_{min},L = airway resistance; DR,L = "additional" resistance of the lung; R_{max},w = resistance of the chest wall; C = compliance; R = resistance; st = static; rs = respiratory system; L = lung; w = chest wall; NS = not significant.

signals were recorded on a four-pen channel recorder and processed via an analog-to-digital converter by computer for storage and calculations. The response of the pneumotachograph, which was calibrated with the same gas mixture used during the experiment, was linear over the whole experimental range of flows. The pressure-flow relationships of the endotracheal tubes were determined after each experiment with the use of the experimental gas mixture. These relationships were used to determine the resistive pressure decrease due to the endotracheal tubes for any given flow during tests (8). As shown in Figure 1, we used the esophageal balloon technique together with transient airway occlusions during constant inspiratory flow to partition the mechanics of the respiratory system into its pulmonary and chest wall components (9). The end-inspiratory hold button of the Servo 900 C was pressed for brief (3–4 s) airway occlusions. Occlusion was maintained until both Pao and Pes decreased from a maximum value (P_{max}) to an apparent plateau (P_2). After the occlusion, an immediate drop from P_{max} to a lower value (P_1), at flow 0, was appreciable in Pao but not in Pes. The plateau pressure (P_2) of Pao and Pes were taken to represent the static end-inspiratory recoil pressures of the respiratory system (Pst,rs) and the chest wall (Pst,w), respectively. The static respiratory system (Cst,rs) and chest wall (Cst,w) compliances were obtained by dividing V_T by the difference between Pst,rs-Pao at end-expiration and Pst,w-Pes at end-expiration, respectively. The static lung compliance (Cst,L) was obtained from Cst,rs and Cst,w according to the following equation: Cst,L = (Cst,rs × Cst,w)/(Cst,w − Cst,rs). An end-expiratory occlusion maneuver was always performed to exclude the possible presence of intrinsic positive end-expiratory pressure. Maximum (R_{max},rs) and minimum (R_{min},rs) resistance of the respiratory system were computed from Pao as $(P'_{max}-P_2)/V'_i$ and $(P'_{max}-P_1)/V'_i$, where P'_{max} represents the new P_{max} value obtained correcting Pao for tube resistance (see above) and V'_i is the flow immediately preceding

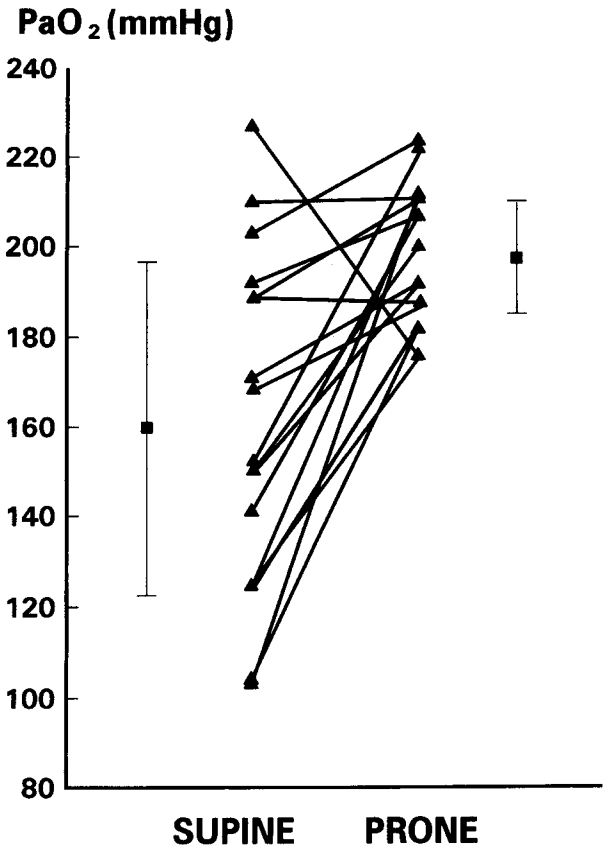

Figure 2. Individual changes in Pao_2 from the supine to the prone position. Pao_2 in the prone position was significantly ($P < 0.01$) increased compared to the supine position.

the occlusion. R_{min},rs represents the flow resistance of airways, and R_{max},rs includes R_{min},rs plus the "additional" respiratory resistance caused by stress relaxation and/or time constant inequalities within the respiratory tissues (9,10). The difference between R_{max},rs and R_{min},rs was termed DR,rs. Since there was no appreciable drop in Pes (i.e., P_1 in the esophageal tracings was not identificable) immediately after the occlusion, R_{min},rs essentially reflects airway resistance

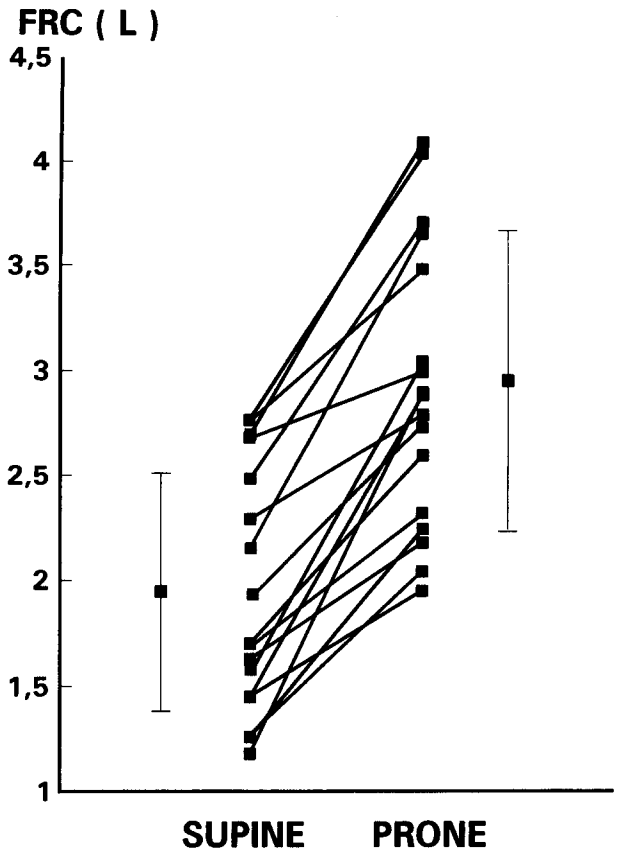

Figure 3. Individual changes in functional residual capacity (FRC) from the supine to the prone position. FRC in the prone position was significantly ($P < 0.01$) higher than in the supine position.

(R_{min},L) and minimum chest wall resistance (R_{min},w) can be considered negligible (9). As a consequence, maximum chest wall resistance (R_{max},w) is entirely due to the viscoelastic properties of the chest wall tissues (i.e., R_{max},w = DR,w). "Additional" resistance of the lung (DR,L) was obtained as DR,rs-DR,w while the sum of R_{min},L + DR,L gives the maximum lung resistance (R_{max},L). DR,L and DR,w (i.e., R_{max},w) were due to stress relaxation and/or time constant inequalities within the lung and chest wall, respectively. In calculation of R_{min},L the errors caused by the closing time of the ventilator were corrected as described (11).

All the measurements were obtained in triplicate prior to surgery. A sample of arterial blood from a puncture of the radial artery was obtained after 15 min by starting the mechanical ventilation and analysis analyzing of blood gases was performed.

Measurements of respiratory mechanics and FRC were also taken. Then the patients were positioned prone, assuring free abdominal movements with upper chest and pelvic supports as suggested by Smith (12). Measurements of gas exchange, respiratory mechanics, and FRC were repeated after 20 min of the prone position. Ventilatory setting (VT and respiratory rate) and F_{IO_2} were unchanged during the protocol.

Data are expressed as mean ± SD. Statistical analysis was made using a Student's paired *t*-test comparing data obtained in supine and prone positions. The least-squares regression method was used to evaluate relationships between variables (13). $P < 0.05$ was accepted as statistically significant.

Results

The delivered VT and V'_i were similar in the supine and prone position (0.662 ± 0.092 L vs 0.665 ± 0.090 L and 0.468 ± 0.065 L/s vs 0.470 ± 0.063 L/s, respectively). Both Cst,rs, Cst,L, and Cst,w were unchanged from supine to prone position (Table 1). On the contrary, changing position, R_{max},rs slightly increased (approximately 20%), mainly due to DR,rs. However, the modifications in respiratory and chest wall resistance were statistically significant but clinically unimportant (Table 1). On the other hand, oxygenation was markedly improved from supine to prone (160 ± 37 mm Hg vs 199 ± 15.7 mm Hg, $P < 0.01$). An increase in Pao_2 less than 20 mm Hg was observed in four patients (24%) as shown in Figure 2. Both $Paco_2$ and pHa did not change significantly from supine to prone (33.8 ± 3.9 mm Hg vs 33.6 ± 3.7 mm Hg and 7.45 ± 0.03 vs 7.46 ± 0.04, respectively). The improvement in oxygenation was paralleled by a marked increase in FRC from the supine to the prone position (1.935 ± 0.576 L vs 2.921 ± 0.681 L, $P < 0.01$) (Figure 3). Changes in FRC between the supine and the prone position were not significantly correlated with respiratory mechanics or Pao_2 changes.

Discussion

In anesthetized and paralyzed patients, we demonstrated that the prone position, if correctly performed, does not significantly alter either lung or chest wall mechanics, while it markedly improves lung volume and oxygenation. Thus, it does not seem to have adverse effects on the mechanics of breathing and gas exchange.

In our supine anesthetized, paralyzed subjects, we obtained an average value of respiratory compliance of 80.9 ± 16.6 mL/cm H_2O. This value is slightly higher than those reported by Bherakis et al. (8) and D'Angelo et al. (9), with the same method of measurement. However, the VT used in these studies was lower than in the present investigation (0.34 L and 0.47 L, respectively vs 0.67 L) and, since compliance varies with VT during anesthesia, this may account for the observed difference (14). When patients were turned prone, we did not observe any significant change in respiratory compliance. Few previous studies investigated changes in respiratory system compliance between the supine and the prone position during anesthesia and paralysis. Lynch et al. (3) observed a 30%–

35% decrease in respiratory compliance and an increase in peak airway pressure when patients were positioned prone; however, they used parallel, hard rubber rolls to support shoulders and hips. This could have impaired chest and abdominal movements with consequent alterations in respiratory mechanics. Also, Safar and Agusto-Escarraga (4) found similar results, although it is not clear how their patients were placed in the prone position. The decrease in respiratory compliance was mainly ascribed to a decrease in chest wall compliance, although it was not really measured. Both authors concluded that the prone position appeared to have adverse effects on the mechanics of breathing in anesthetized subjects.

In contrast to previous studies, we positioned our patients as described by Smith (12), assuring free abdominal movements, with upper chest and pelvic supports. This kind of prone position could explain our different results, since the chest wall is less constricted compared to other proposed prone positions (15).

Moreover, we partitioned total respiratory system mechanics into lung and chest wall components in order to define their relative modifications with position changes. We adopted the end-inspiratory occlusion technique with constant inspiratory flow together with an esophageal balloon (9). One could question the comparison of esophageal balloon measurements in the supine and the prone positions. However, this method has been considered adequate and was previously adopted by Milic-Emili et al. (16) to perform lung-volume curves in awake subjects in different positions, including the prone position. Furthermore, we performed the "occlusion test" (7) in both the supine and the prone position, assuring an accurate measurement of esophageal pressure in both positions.

We found that not only total respiratory compliance but also lung and chest wall compliance remained unchanged when patients were positioned prone. We are not aware of other studies that have partitioned lung and chest wall mechanics in the supine and the prone positions. As discussed above, our patients were positioned to assure free abdominal and chest movements, and this could have contributed to the unaltered chest wall compliance.

We also found a slight and clinically unimportant increase in respiratory resistance, mainly due to an increase in the "additional" resistance of the respiratory system (DR,rs), while airway resistance (R_{min},L) was unchanged in the prone position. DR,rs represents the viscoelastic properties of the respiratory system, including those of the lung and the chest wall (9,10). The increase in DR,rs that we observed was mainly due to an increase in the chest wall component (DR,w). A previous study showed moderate modifications in respiratory and chest wall resistance in different positions in awake subjects, and these changes were probably due to different adaptability of the chest wall mechanical behavior in different postures (17). Overall, our findings do not support the idea that the prone position significantly alters respiratory resistance; and although the prone position did not significantly impair compliance and resistance, it markedly improved oxygenation and FRC (maintaining unchanged $Paco_2$).

Moreno and Lyons (18) found that subjects in the prone position failed to show a significant change in FRC as compared with the supine position, but Lumb and Nunn (5) found an increase in FRC of 0.350 L in the prone position in a group of normal, awake subjects. A previous work (19), in anesthetized prone humans, reported a mean FRC no different from our mean value (3.020 ± 0.900 L vs 2.921 ± 0.681 L). It has been clearly shown that, during anesthesia and muscle paralysis in the supine position, there is a tendency for alveolar collapse, particularly in the dependent lung regions, and that the amount of atelectatic areas is well correlated with the impairment in oxygenation (shunt fraction) (20,21). The increase in FRC in the prone position may be explained both by a reduction of cephalad pressure on the diaphragm and/or a reopening of atelectatic segments, although the former hypothesis is the most likely.

Oxygenation changes in the prone position during anesthesia have been poorly investigated (5). Our study demonstrated that patients in the prone position had better oxygenation compared to those in the supine position. A possible explanation for this may be the relative improvement in the ventilation-perfusion ratio within the lungs. The heart occupies the anterior mediastinum, and therefore there is less lung volume anteriorly than posteriorly. Consequently, there is more ventilatable lung in nondependent regions in the prone position. This hypothesis was confirmed by a recent computed tomographic study showing a predominant motion of nondependent diaphragm regions during mechanical ventilation in the prone position (22). In addition, a more uniform distribution of perfusion has been shown, at least in animals, in the prone compared to the supine position (23). The diminished atelectasis probably present in the dependent lung regions (since they are occupied by the heart) and the nongravitational distribution of blood flow in the prone position may explain the improvement in the relative ventilation-perfusion ratio and oxygenation.

In conclusion, we have demonstrated that when the prone position is used correctly it does not alter respiratory mechanics and it improves oxygenation and lung volume.

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