The Effects of Thiopental-Meperidine Anesthesia with Succinylcholine Paralysis on Functional Residual Capacity and Dynamic Lung Compliance in Normal Sitting Man

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Functional residual capacity (FRC) and dynamic lung compliance \( [C_{FRC}] \) were measured in ten normal sitting subjects while conscious and again during thiopental-meperidine anesthesia with succinylcholine paralysis and mechanical ventilation. At similar tidal volumes and respiratory frequencies, no significant changes in FRC and \( C_{FRC} \) were observed. Mean FRC's were 3.26 ± 0.26 (SE) liters and 3.29 ± 0.24 (SE) liters and mean values for \( C_{FRC} \) were 182 ml/cm H₂O (SE ± 25) and 133 ml/cm H₂O (SE ± 34) in conscious and anesthetized paralyzed subjects, respectively. The results suggest that the reductions in FRC and \( C_{FRC} \) previously observed in supine anesthetized subjects are not the result of a direct pharmacologic effect of thiopental-meperidine anesthesia on the lung. (Key words: Dynamic lung compliance; Functional residual capacity; Mechanical ventilation; Paralysis; Sitting position; Succinylcholine; Thiopental-meperidine anesthesia.)

Several authors have reported that thiopental anesthesia results in reductions of both functional residual capacity (FRC)¹-³ and lung compliance \( [C_{FRC}] \)⁴,⁶,⁷ in supine subjects. The underlying mechanisms of these changes are not completely understood. Recently, it has been suggested that the reduction in FRC observed in supine subjects anesthetized with thiopental-meperidine and paralyzed with succinylcholine is the result of a change in the pressure-volume characteristics of the chest wall. The low lung compliance is believed to be secondary to breathing at lung volumes below normal FRC.⁸

Recognizing that the changes in the pressure-volume characteristics of the chest wall during anesthesia may be dependent on position, and that changes with anesthesia may not occur except in the supine position, we decided to measure lung compliance and FRC in anesthetized sitting man.

\( C_{FRC} \) and FRC were determined in ten normal subjects in the sitting position, first conscious and then during thiopental-meperidine anesthesia with succinylcholine paralysis and mechanical ventilation. No significant changes in FRC and \( C_{FRC} \) were demonstrated after the subjects were placed in the sitting position following induction of anesthesia. This study suggests that the reductions of FRC and lung compliance found in supine subjects are not a direct pharmacologic effect of the anesthetic agent on the lung.

**Methods**

Ten healthy young adult volunteers participated in this study. Each subject was interviewed and told of the nature of the study, the manner in which it would be conducted, and the possible risks involved. Written consent to participate was accepted only after suitable time for consideration had been given. Each subject was allowed to rest for about 30 minutes prior to the study. An indwelling arterial needle was inserted percutaneously under local anesthesia into a radial artery, and both legs were wrapped with elastic bandages. The subject then assumed the sitting position. The control study in the awake state was carried out by connecting the subject through a mouthpiece (nasal) orotracheal intubation.
### Table 1. Functional Residual Capacity, Dynamic Lung Compliance, Ventilation Indices, and Blood-Gas Tensions

<table>
<thead>
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<th>Index</th>
<th>Conscious, Breathing Spontaneously</th>
<th>Anesthetized, Paralyzed, Mechanically Ventilated</th>
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<tbody>
<tr>
<td></td>
<td>N</td>
<td>Mean</td>
</tr>
<tr>
<td>FRC (BTPS), liters</td>
<td>9</td>
<td>3.26</td>
</tr>
<tr>
<td>Dynamic lung compliance, ml/cm H₂O</td>
<td>9</td>
<td>182</td>
</tr>
<tr>
<td>Vt (BTPS), ml</td>
<td>9</td>
<td>697</td>
</tr>
<tr>
<td>Respiratory frequency, breaths/min</td>
<td>9</td>
<td>11</td>
</tr>
<tr>
<td>Pao₂ (FiO₂ = 0.21), torr</td>
<td>7</td>
<td>96</td>
</tr>
<tr>
<td>Pao₂ (FiO₂ = 1.0), torr</td>
<td>6</td>
<td>510</td>
</tr>
<tr>
<td>Paco₂ (FiO₂ = 0.21), torr</td>
<td>7</td>
<td>37</td>
</tr>
<tr>
<td>Paco₂ (FiO₂ = 1.0), torr</td>
<td>6</td>
<td>36</td>
</tr>
</tbody>
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clip) to a nonrebreathing system (end-expiratory pressure less than 1 cm H₂O).

After the control study, the subject was anesthetized in the supine position. The larynx and trachea were sprayed with 4 per cent lidocaine, and the trachea was intubated with a cuffed oral endotracheal tube. Anesthesia was induced and maintained with an intravenous infusion of thiopental (8 to 20 mg/kg) and supplemented with intravenously administered meperidine hydrochloride (1 to 2 mg/kg).

Depth of anesthesia and degree of muscle paralysis were judged by use of clinical signs only. Muscle paralysis was achieved with succinylcholine chloride (6 to 14 mg/kg), and each subject received an intravenous injection of scopolamine hydrobromide (0.43 mg) after induction of anesthesia. The frequency of mechanical ventilation with room air was adjusted to result in a Paco₂ of approximately 40 torr (Bird Mark 4 driven by a Bird Mark 7) at tidal volumes similar to those observed during the study of conscious subjects. Approximately 30 minutes after the induction of anesthesia, the subject was positioned and secured in a sitting posture similar to that used in the control study. The subject was then connected through the endotracheal tube to the nonrebreathing system (end-expiratory pressure less than 1 cm H₂O). The second part of the study was carried out after the lungs had been inflated three times to an end-inspiratory airway pressure of 30 cm H₂O. Total anesthesia time was 1 to 1.5 hours.

FRC was determined by the open-circuit method of nitrogen washout, with pure oxygen being used as the inhaled diluent gas. Expired gas was collected for 7 minutes in a neoprene bag, which was evacuated just prior to gas collection. Expired gas volume was measured in a 120-liter gasometer. Nitrogen concentration of alveolar gas was determined by a nitrogen meter. Oxygen and carbon dioxide concentrations of the expired gas were determined by duplicate Haldane analyses, and the nitrogen concentration was obtained by difference. All FRC values were corrected for the switching error and for nitrogen eliminated from blood and tissue. C_drn(t) was estimated from volume change and transpulmonary pressure change. Volume was obtained from the integrated flow signal of a pneumotachograph. Transpulmonary pressure was determined from the difference between pleural pressure and oral pressure (differential strain gauge, Statham PM131). Pleural pressure was estimated with an esophageal balloon (12 cm long, 3.5 cm perimeter), containing 0.6 ml of air. The balloon was placed in the middle third of the esophagus (usually 42 cm from the nares), where there was minimal interference with the pressure signal by cardiac activity. The position of the balloon was not changed throughout the study. In each subject, lung compliance was derived from the mean value of several consecutive breaths (7 to 29).

Arterial blood was sampled immediately before the determination of FRC during breathing of room air (FiO₂ = 0.21) and again at the end of the 7-minute period of breathing oxygen (FiO₂ = 1.0). Arterial oxygen and carbon dioxide tensions were determined with
appropriate electrodes (temperature 37°C) and corrected for body temperature (rectal thermistor).

Results

Ten volunteers whose ages ranged from 21 to 30 years participated in the study. Data from one subject have been excluded because of technical difficulties.

Mean arterial blood-gas tensions and their standard errors in conscious subjects and during anesthesia are shown in Table 1. No significant changes in $P_{O_2}$ and $P_{CO_2}$ were observed. Mean values for FRC did not change significantly ($P > 0.05$) after the induction of general anesthesia, muscle paralysis, and mechanical ventilation (Table 1); FRC's were 3.26 l in conscious subjects and 3.29 l in anesthetized paralyzed subjects. Similarly, mean values for $C_{d鞲r(1)}$ measured at similar tidal volumes and respiratory frequencies did not change significantly; they were 182 ml/cm H$_2$O in conscious subjects and 133 ml/cm H$_2$O in anesthetized paralyzed subjects.

Discussion

The medical literature contains conflicting results concerning the effects of general anesthesia on FRC and lung compliance. Most authors have reported reductions in FRC during thiopental anesthesia, but Howell and Peckett found FRC unchanged. Some investigators found no change in lung compliance during thiopental anesthesia and succinylcholine paralysis, but others, demonstrated reductions in lung compliance. Recently, Westbrook et al. from our institution, reported a marked reduction in static lung compliance during thiopental–meperidine anesthesia. Using similar anesthetic management and similar doses of thiopental and meperidine and succinylcholine, we were unable to demonstrate in the present study decreases in FRC and $C_{d鞲r(1)}$ in anesthetized sitting subjects. The findings of this study, therefore, suggest that it was not a direct pharmacologic effect of the thiopental–meperidine on the lung which caused the decreases in lung compliance and FRC in anesthetized, supine subjects. This conclusion is in agreement with the hypothesis of Westbrook et al. who suggested that a change of the pressure–volume characteristics of the chest wall results in a reduction of FRC and that the reduced lung compliance was secondary to breathing at a lung volume below normal FRC. Changes in the pressure–volume characteristics of the chest wall during thiopental–meperidine anesthesia, therefore, appear to be dependent on position.

The validity of lung compliance measurements in supine subjects might be questioned, since changes in esophageal pressure are not identical to changes in pleural pressure, and falsely-low values for lung compliance may be obtained when the patient is in the supine position. However, comparative measurements of esophageal pressures should give an accurate estimate of changes in lung compliance if the position of the esophageal balloon has not been changed.

Reduction of lung volume to levels approaching residual volume has been reported to result in increased closure of airways. Airway closure during tidal breathing can result in the cessation of ventilation of lung regions distal to the airway closure. If perfusion of these nonventilated regions continues, the alveolar–arterial oxygen tension gradient should increase. Therefore, it is of interest that in the presence of unchanged lung volume we were unable to demonstrate a significant decrease in $P_{O_2}$ during breathing of either room air or oxygen after the induction of anesthesia.

In summary, no decrease in FRC or $C_{d鞲r(1)}$ was observed in sitting subjects when measurements were made before and after thiopental–meperidine anesthesia with succinylcholine paralysis and mechanical ventilation.

References

Drugs and Their Actions

CARIOVASCULAR EFFECTS OF LEVODOPA The cardiovascular effects of levodopa are potentially dangerous and are due primarily to dopamine, which is released by decarboxylation of the amino acid. Dopamine acts on β-adrenergic, α-adrenergic, and "dopaminergic" receptors. The last cause renal and mesenteric vasodilation. In normal man and the dog moderate doses produce increases in cardiac output and renal blood flow, a decrease in total peripheral resistance, and no significant change in heart rate. The present study determined the cardiovascular effects of orally-administered levodopa during treatment of parkinsonism. A non-invasive technique of measuring the pre-ejection period by recording simultaneously the phonocardiogram, carotid pulse contour, and ECG was used. Essentially, this is an indirect measurement of myocardial contractility. Following 1.0 and 1.5 g of levodopa given by mouth to six patients, the pre-ejection period was shortened by 11 ± 2.4 msec and 20 ± 3.2 msec, respectively. The effect began in 20 minutes and lasted approximately two hours. Heart rate and arterial blood pressure in the supine position did not change. The effect of levodopa was blocked by 10 mg of propranolol given orally. Tolerance to the cardiovascular effects appeared after periods of at least three months of treatment with levodopa. The investigations show a definite β-adrenergic effect of clinical doses of levodopa and suggest caution in starting therapy in the presence of coronary-artery insufficiency or atrial, nodal, or ventricular arrhythmias. Therapy should be started where intensive care facilities are available. There may be a summation of effects of drugs in patients being treated for asthma. Orthostatic hypotension is not uncommon and should be considered in patients with coronary and cerebral insufficiency. Propranolol and phentolamine should be effective in treating an overdose of levodopa. Halothane or cyclopropane can be administered safely to patients when levodopa is discontinued the night before surgery. (Goldberg, L. I., and Whitsett, T. L.: The Cardiovascular Effects of Levodopa. J.A.M.A. 218: 1921-1923, 1971.)