

Effects of Barbiturate Anesthesia on Functional Residual Capacity and Ribcage/Diaphragm Contributions to Ventilation

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The effect of iv methohexital infusion anesthesia on functional residual capacity (FRC) (helium dilution) in 14 surgical patients (age 23 to 59 years) was determined. Eight subjects were studied wearing an inflatable mask, sealed with surgical lubricant. They showed a mean \pm SD $3.5 \pm 6.4\%$ FRC decrease (no significance). Six subjects studied *via* mouthpiece awake and *via* endotracheal tube during anesthesia showed a mean $22 \pm 19\%$ reduction in FRC, significantly greater than face mask studies ($P < 0.05$). The greatest FRC decrease occurred in subjects with repetitive or protracted coughing after intubation. The serum methohexital level was 6.6 ± 3.6 $\mu\text{g/ml}$ for intubated patients, and 6.0 ± 1.1 $\mu\text{g/ml}$ in those with face mask (no significance). The depth of anesthesia was sufficient to produce a 50% reduction in ventilatory response to CO_2 rebreathing, from 15.8 to 8.7 l/min/% CO_2 . RespiTrace[®] plethysmography indicated a $38 \pm 12\%$ ribcage contribution to tidal volume during quiet breathing, which increased to $47 \pm 14\%$ with CO_2 breathing (end-tidal FCO_2 , 9–10%). There was no diminution of ribcage contribution during anesthesia in either group, irrespective of CO_2 concentration. The authors interpret their findings to indicate that iv methohexital anesthesia does not produce FRC reduction, in contrast to an inhaled anesthetic such as halothane. It is proposed that this difference may be related to maintenance of coordinated ribcage/diaphragm muscle activity, because ribcage activity is markedly suppressed by halothane. In addition, it is proposed that FRC reduction in intubated subjects was the result of a confounding variable, namely coughing in response to the endotracheal tube. (Key words: Anesthetics, intravenous: barbiturate; methohexital. Muscle: diaphragm; intercostals. Ventilation: functional residual capacity; pattern of breathing.)

REDUCTION IN FUNCTIONAL residual capacity (FRC) is a commonly reported feature of general anesthesia.^{1,2} Despite numerous studies, the cause of the reduction in FRC has remained elusive. Most explanations have centered on changes in the properties of the ventilatory muscles. Froese and Bryan proposed that this was due to a cephalad displacement of the diaphragm, seen both with and without muscle paralysis.³ Tusiewicz *et al.*⁴ and Jones *et al.*⁵ suggested that loss of phasic and tonic ribcage muscle activity probably contributes to the fall in lung volume during spontaneous ventilation with halothane anesthesia. This was supported by Hedenstierna *et al.* who showed reduction in the transverse area of the thorax as well as

cephalad displacement of the diaphragm.⁶ Diaphragm activity, however, has been shown to be minimally affected during 1 MAC halothane anesthesia, when intercostal muscle response to CO_2 was suppressed by 89%.⁴ This finding led Tusiewicz *et al.* to speculate that halothane causes preferential suppression of the spinal intercostal neurons pool rather than only brain stem respiratory depression.⁴

A recent observation in sheep studies led us to speculate that such preferential suppression of ribcage activity may not be a feature of all general anesthetics. Barbiturate anesthesia, in contrast to halothane, did not produce a significant decrease in FRC.⁷ A review of the anesthesia literature shows that Colgan and Whang also reported no change in FRC with barbiturate anesthesia in spontaneously breathing human subjects.⁸ Howell and Peckett⁹ and Bergman¹⁰ found reductions of less than 200 ml in end-expiratory position (closed-circuit spirometry *via* face mask) following administration of thiopental. In contrast, studies employing endotracheal intubation, muscle paralysis, and mechanical ventilation, or inhalation anesthetic agents have all shown FRC reduction on the order of 400 ml (*i.e.*, 15–20% reduction in awake control FRC). We therefore wished to address the specific question: does iv barbiturate anesthesia cause FRC reduction and loss of ribcage contribution to tidal ventilation?

Methods

SUBJECTS

Fourteen male surgery patients (age 23–59 yr) were selected on the basis of a normal pulmonary history (no previous or current respiratory disease, no recent upper respiratory infections), and no history of cardiovascular disease or major medical illnesses. None were obese, as defined by a body mass index (BMI) of more than 29 (BMI = weight (kg)/height (m)²). Four of the subjects were cigarette smokers (10–20 pack-year histories). Approval of the protocol was obtained from the University Human Subjects Committee, and written informed consent was obtained from each subject.

The subjects received no preoperative medications. On the morning of surgery spirometric pulmonary function tests were administered to 13 of the 14 subjects. A Steadwells 9 l spirometer was used to measure tidal volume, vital capacity, forced expired volume at 1 and 3 s, maximum ventilatory ventilation and forced expiratory flow ($\text{FEF}_{25-75\%}$).

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MEASUREMENT TECHNIQUES

FRC was measured by closed-circuit helium dilution. Oxygen was metered into the closed circuit at a rate achieving constant volume of the test circuit. Thorough mixing of gas in the spirometer and connecting tubing was insured by a mixing fan. The fractional helium content (F_{He}) was checked periodically, and the final F_{He} was not recorded until it had reached a constant level (5–7 min). Total closed-circuit volume measurements were corrected for the helium lost with each gas sample.

Because increased flow resistance due to upper airway relaxation and collapse during anesthesia could have dynamically increased FRC by trapping air in the lungs, ventilatory flow rates were measured in three subjects. A large-bore pneumotachograph (Fleisch #00) was included just distal to the face mask. Ventilatory flow rates were then evaluated during the FRC determinations to rule out flow interruption prior to the end of expiratory ribcage or abdominal motion. The pneumotachograph was calibrated against a rotameter flowmeter by passing compressed air through both simultaneously. The response was linear over the range of flows encountered *in vivo* (0–5 l/s).

The individual contributions of the ribcage (primarily intercostal muscles) and abdominal (primarily diaphragm) compartments to tidal ventilation were measured with an inductance plethysmograph (Respirace Corp., Ardsley, NY). The elastic transducer bands were calibrated while the patient was awake and breathing normally into the 9-l spirometer in both seated and supine position.^{11,12} Ink marks on the skin were used to reposition the bands if they moved. The separate signals from the ribcage, abdomen, and a sum channel (ribcage + abdomen) were displayed on a strip chart recorder. During FRC determinations and during the CO₂ rebreathing tests, tidal volume was simultaneously measured with the spirometer and plethysmograph. In a report by Zimmerman *et al.* the accuracy of the Respirace[®] in determining tidal volume was called into question.¹³ We therefore compared the independent and simultaneously measured tidal volumes determined by Respirace[®] sum channel with that of the spirometer for a series of 40 breaths spanning a large range of volume in nine awake subjects during FRC determination and in another series of 40 breaths in eight awake subjects during CO₂ stimulated ventilation. An equal number of measurements (total of 160 breaths) were made on the same subjects when anesthetized.

Compartmental contributions to ventilation were also measured during CO₂ stimulated ventilation using the modified Read rebreathing method.¹⁴ Each subject was studied while supine breathing through a rubber mouthpiece with snug nose-clip ($n = 6$), or through a plastic face mask ($n = 8$) sealed tightly around both the nose and mouth with an inflatable cuff and surgical lubricant. In-

spiratory and expiratory volume and end-tidal CO₂ at the mouthpiece were measured continuously with the subject rebreathing from the 9 l spirometer filled initially with 6.5% CO₂ in O₂. The helium dilution mixing fan insured rapid system equilibration, but the baralyme canister was bypassed. The CO₂ level in the rebreathed gas steadily increased from 6.5 to a maximum of 10% end-tidal CO₂. A Perkin-Elmer MGA-100[®] Mass Spectrometer was used to measure the fractional concentrations of He, CO₂, and O₂.

SUBJECT PREPARATION

Catheters were placed in a peripheral vein for anesthetic administration and in a radial artery for blood pressure and blood gas analysis using an IL-813[®] blood gas analyzer. ECG electrodes were attached to each subject and in several cases, a transcutaneous PO₂ monitor was employed.

STUDY PROTOCOL

In supine awake subjects two or three determinations of FRC were made and arterial blood gases were measured while the subjects breathed room air or 30% O₂ in air. Blood samples were also obtained for later analysis of barbiturate levels (control). The CO₂ rebreathing test was then administered. In awake studies the FRC and CO₂ rebreathing tests were administered *via* a mouthpiece and snug nose-clip (first six subjects) or *via* a mask (last eight subjects).

Intravenous anesthesia was then administered by 50 to 100 mg incremental injections as well as by continuous infusion (IVAC[®]) of 15–30 mg/min of methohexital (Brevital[®]). The rate was adjusted to achieve stable breathing (regular rate and rhythm, minimal coughing or hiccupping), heart rate, and blood pressure. One patient in the tracheally intubated group (see following) received only thiopental, but with the same parameters for depth of anesthesia. After induction, the first six subjects were intubated with a cuffed tracheal tube placed with the aid of topical lidocaine. Eight subjects were not intubated but continued to breathe *via* the mask used for the awake portion of the study. In several of these latter subjects a latex nasopharyngeal or oral airway was inserted to improve upper airway patency. Then, measurements of FRC, CO₂ ventilatory response, and blood gas samples were obtained during periods of stable respiratory and circulatory conditions. At the end of the CO₂ rebreathing test, a blood sample was obtained for later analysis of serum barbiturate levels by nitrogen-phosphorus flame ionization detection (NP-FID) gas chromatography, using Bond Elut[®] extraction columns (Analytchem International, Inc., Harbor City, CA), 3-foot glass chromatography columns packed with 3% poly A103 on Gas Chrom Q[®] 100/120 mesh (Applied Science Laboratories, Deerfield, IL) and an amobarbital internal standard.

STATISTICAL TESTS

Unless specified, paired or standard *t* tests were used to compare the means of data from face mask or intubated groups. Linear regressions were calculated by the method of least squares. Analysis of co-variance was applied to the comparison of tidal volume measurements from spirometer and RespiTrace®. The degree of significance of increase in per cent contribution of the ribcage compartment to tidal volume was assessed by analysis of variance. *P* < 0.05 was required for significance in all cases.

Results

SUBJECT CHARACTERISTICS

Anthropometric data on the study subjects were: (means ± SD) age 37.1 ± 10.1 (range 23–59), height 1.78 ± 0.07 m, weight 77.8 ± 10.7 kg, BMI 24.4 ± 2.4 kg/m², and cigarette use 13.3 ± 8.2 pack-years (four subjects). Thirteen of the 14 subjects were administered pulmonary function tests prior to beginning the study. In these 13 subjects the tidal volume, vital capacity, forced vital capacity, forced expiratory volume at 1 and 3 s, FEF_{25–75%}, and maximum ventilation volume were all within normal limits.¹⁵ There were no significant differences in age, pulmonary function tests, or anthropomorphic data between the group of patients tracheally intubated for the study and the group studied without intubation (inflatable mask).

FRC

Figure 1 presents a comparison of FRC in subjects before and after receiving methohexital. The mean awake FRC value for all subjects (mouthpiece and face mask) was 2.43 ± 0.50 (SD) l. There was no significant difference in awake control FRC of the eight face mask and six mouthpiece subjects (*P* > 0.1). Anesthesia produced a mean 22.3 ± 19.6% reduction in FRC compared with control in the six patients with tracheal intubation (range 2.9–54%) (*P* < 0.05, paired *t* test), compared with only a 3.5 ± 6.4% reduction in face mask subjects (not different from 0% reduction). The difference in FRC reduction between intubated and face mask groups was statistically significant (*P* < 0.05). Protracted coughing (10–30 s duration) in response to endotracheal tube placement was observed in five of the six intubated subjects, but no coughing was seen in those studied with a face mask.

Pneumotachograph recordings in three face-masked subjects showed no evidence of interruption in expiratory or inspiratory gas flow (due to airway obstruction) during the anesthesia FRC determinations. There was also a period of zero flow just preceding inspiration in essentially all breaths of the three subjects studied, suggesting that the lungs were free to reach their normal end tidal volume.

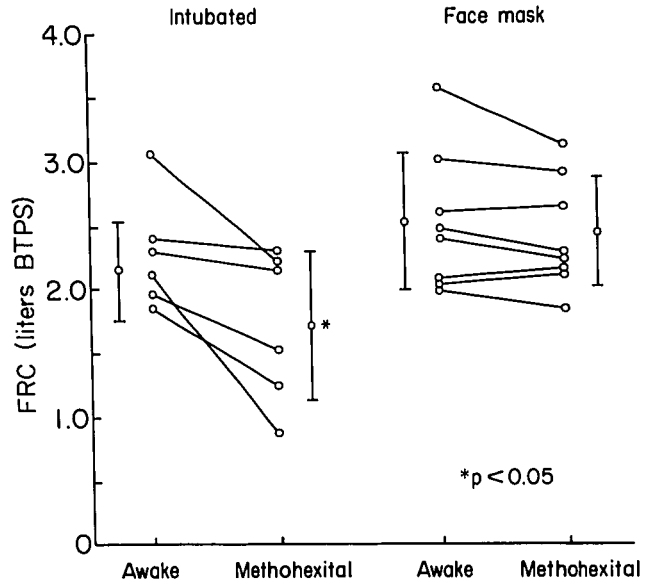


FIG. 1. FRC values (mean ± SD) in individual subjects when awake (supine) and following methohexital. In face mask subjects FRC decreased 3.5 ± 6.4% (NS) and in intubated subjects 22.3 ± 19.6% (*P* < 0.05).

USE OF RIBCAGE AND ABDOMINAL MUSCLES AT REST

In the supine position, while breathing 30% O₂, 0% CO₂ in air, the ribcage compartment contributed an average 38 ± 12% of total tidal volume and the abdominal/diaphragm compartment the remaining 62 ± 9% of tidal volume. During anesthesia and quiet breathing corresponding values were 40 ± 15% and 60 ± 10% of tidal volume, respectively (awake anesthesia difference not significant).

SERUM BARBITURATE LEVELS

The total dose of methohexital received up to the start of the CO₂ rebreathing test was 15.0 ± 8.2 mg/kg (mean ± SD). Serum methohexital levels were 6.6 ± 3.6 µg/ml in intubated subjects and 6.0 ± 1.1 µg/ml in subjects studied with face masks (difference was not significant).

VENTILATORY RESPONSE DURING CARBON DIOXIDE REBREATHING TESTS

The ventilatory response to rebreathing CO₂ in awake supine subjects was described overall by the least-squares regression:

$$\dot{V}_E = 155.4(\%CO_2 ET) - 917.5 (r = 0.96)$$

and that during anesthesia by the least-squares regression:

$$\dot{V}_E = 87.2(\%CO_2 ET) - 498.2 (r = 0.97)$$

where \dot{V}_E is in ml BTPS · kg⁻¹ · min⁻¹ and ET = end-tidal. The slopes and intercepts are significantly different at the 0.001 level. These ventilation data are plotted in figure 2 in increments of 0.5% CO₂.

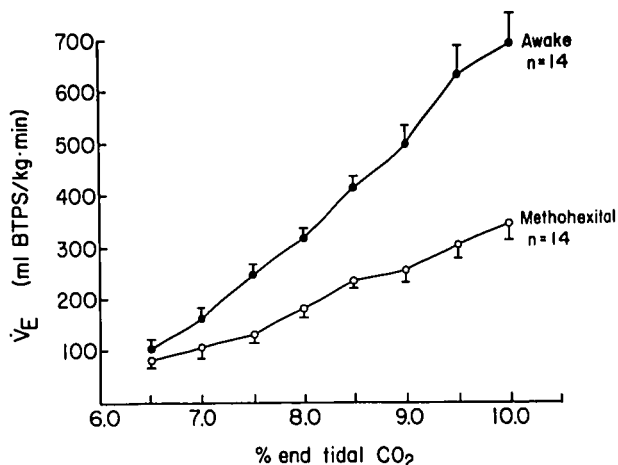


FIG. 2. Minute ventilation (\dot{V}_E) during the CO₂ rebreathing test in awake and anesthetized subjects. Values shown are mean \pm SE. The slope of the response changed from 15.8 to 8.7 l/min/%CO₂ with anesthesia. See text for least-squares regression formulae describing these relationships.

COMPARTMENTAL VENTILATION DURING CARBON DIOXIDE REBREATHING TESTS

Ribcage and abdomen compartment contributions to tidal volume are shown in figure 3. On average, during the entire CO₂ rebreathing test the ribcage contributed $47 \pm 14\%$ of tidal volume in awake subjects and $46 \pm 15\%$ of tidal volume (mean \pm SD) during anesthesia. In figure 4 the percentage contribution of the ribcage compartment to tidal ventilation has been plotted against end-tidal CO₂

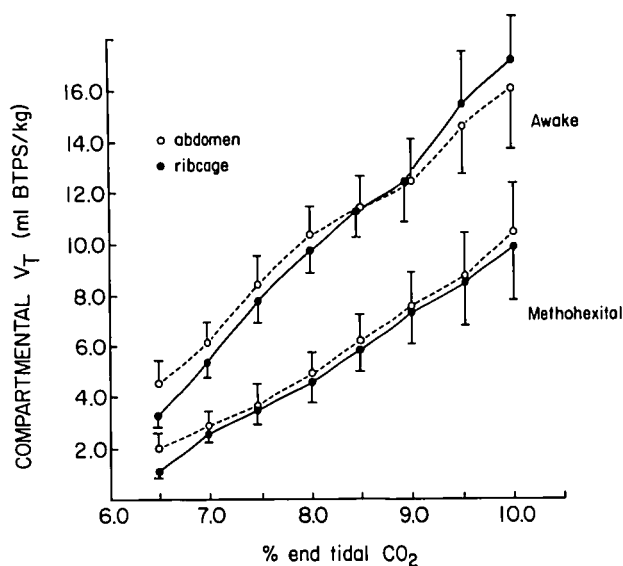


FIG. 3. Separate contributions of the ribcage (RC) and abdomen/diaphragm (ABD) compartments to tidal volume in awake and anesthetized subjects during the CO₂ rebreathing test. Values are mean \pm SE. There was no significant change in relative compartmental contributions, awake *versus* anesthetized.

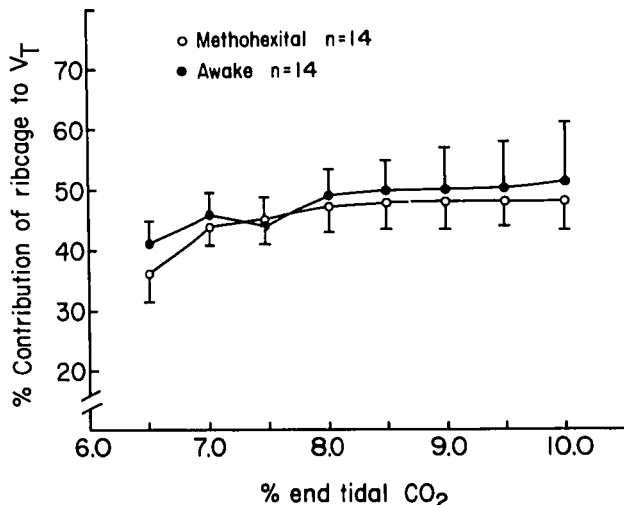


FIG. 4. Percentage contribution of the ribcage compartment to tidal volume (V_T) in awake and anesthetized subjects during the CO₂ rebreathing test. Values are mean \pm SE. Note that the characteristic increase in ribcage contribution with CO₂ stimulation is essentially the same for awake and anesthesia conditions. See text for overall mean values and for values during 0% CO₂ breathing.

concentration. There was a significant increase in the contribution of the ribcage to tidal volume as end-tidal CO₂ increased from 6.5 to 10% in both awake and anesthetized subjects ($P < 0.05$). At a given level of end-tidal CO₂, the percentage contribution of the ribcage compartment to ventilation was not changed by methohexital anesthesia.

Simultaneous measurements of RespiTrace® and spirometer tidal volumes showed a correlation coefficient of 0.993 ($n = 160$) for both awake and anesthesia conditions. Further analysis demonstrated an average $4.2 \pm 5.0\%$ underestimate by the RespiTrace® in awake subjects during FRC determinations ($n = 40$ breaths, six subjects) and a $5.0 \pm 4.0\%$ underestimate during the CO₂ test. During anesthesia, the comparative figures were a $2.7 \pm 6.1\%$ overestimate during FRC measurement and $3.8 \pm 6.4\%$ underestimate during CO₂ rebreathing (no significance).

ARTERIAL BLOOD GAS VALUES

Arterial pH, P_{CO₂}, and P_{O₂} in subjects breathing 30% O₂, 0% CO₂ in air for at least 10 min before and after induction of anesthesia are given in table 1. Values are as measured at 37° C and not corrected to patient temperature. The differences between awake and anesthesia values were significant ($P < 0.05$). Table 1 also shows that the alveolar-arterial PO₂ difference [$P(A - a)_{O_2}$, alveolar P_{O₂} calculated with the alveolar gas equation:

$$PA_{O_2} = PI_{O_2} - Pa_{CO_2}/R$$

where R is the respiratory exchange ratio (0.8)], showed a modest but not statistically significant increase in either

TABLE 1. Arterial Blood Gas Values and $P(A - a)_{O_2}$ (mean \pm SD) (n in parentheses) while Breathing 30% O_2 , 70% N_2

	Awake		Anesthesia	
	Mouthpiece	Face Mask	Endotracheal Tube	Face Mask
pH	7.39 \pm 0.02 (6)	7.37 \pm 0.03 (8)	7.32 \pm 0.01 (6) *	7.29 \pm 0.06 (6) *
P_{aCO_2} mmHg	40.7 \pm 1.8 (6)	43.1 \pm 2.4 (8)	48.7 \pm 1.6 (6) *	50.0 \pm 4.0 (6) *
P_{aO_2} mmHg	122.5 \pm 21.4 (4)	133.8 \pm 11.0 (8)	99.8 \pm 19.1 (5)	119.7 \pm 20.8 (7) *
$P(A - a)_{O_2}$ mmHg	36.6 \pm 21.4 (4)	20.6 \pm 13.0 (8)	49.9 \pm 19.1 (5)	31.3 \pm 22.1 (7)

Temperature of blood gas electrodes was 37° C.

* Significant difference between anesthesia and awake values. Dif-

ferences between awake and anesthesia $P(A - a)_{O_2}$ values were not statistically significant.

anesthetic group, with a trend ($P = 0.20$) toward increasing more in the intubated subjects. There was a significant correlation between reduction in FRC during anesthesia and increase in $P(A - a)_{O_2}$ as expressed by the following relationship:

$$\text{Awake } P(A - a)_{O_2} - \text{Anesthesia } P(A - a)_{O_2} = -1.02(\% \text{ FRC change}) - 4.12$$

($r = -0.74$, $P = 0.01$, $n = 10$) but not between FRC reduction and P_{aO_2} ($r = 0.4$).

Discussion

FRC DURING METHOHEXITAL ANESTHESIA

The effects of methohexital anesthesia on the mechanics of breathing are different from those of inhalational anesthetics. The 3.3% reduction in FRC in nonintubated subjects is comparable to the 6.1% decrement in FRC in normal sleep.¹⁶ Our findings are in agreement with several other studies employing barbiturates alone in nonintubated subjects.⁸⁻¹⁰ For example, Don *et al.* found no change in spirometer position (closed-circuit) during thiopental-induced apnea.¹⁷

Our findings contrast with the large reduction in FRC produced by inhalational anesthesia in both human subjects and experimental animals (for review, see ref. 1). Prutow *et al.* found a 15% reduction in FRC with halothane in surgical patients.¹⁸ Hickey *et al.* found a 19% decrease in FRC with mask breathing during halothane anesthesia maintenance following iv thiopental induction.¹⁹ Don *et al.* found a 31% reduction with halothane in subjects who showed no FRC reduction with thiopental.¹⁷

We suggest that one reason why FRC did not fall appreciably with methohexital anesthesia is that the coordinated action of the intercostal and abdominal/diaphragm muscles is preserved. This contrasts with an average 91% inhibition of intercostal/ribcage muscle contribution to ventilation during 1 MAC halothane anesthesia.^{4,5} A recent study found that halothane anesthesia produced reductions in the transverse area of the thorax as well as cephalad displacement of the diaphragm;

both changes are consistent with inhibition of normal respiratory muscle performance.⁶ A common feature of other studies on FRC during anesthesia is the use of muscle relaxants and/or opiates and sedatives. Because muscle relaxants could reduce both the phasic and tonic performance of the respiratory muscles, it seems likely that muscle relaxants could directly contribute to reduced FRC. In the few studies in which lung volume did not decrease following induction of anesthesia, no muscle relaxants or paralyzing agents were used.^{8,9}

Tracheal intubation has been used in almost all studies on the effects of anesthesia on FRC in humans. It is of interest that the only studies in which lung volume has been reported to show little change due to anesthesia involved subjects fitted with a face mask and not intubated.⁸⁻¹⁰ We are aware of only one study (that of Hickey *et al.*,¹⁹ with halothane anesthesia) that showed a significant FRC reduction (19%) in subjects studied with a face mask and not intubated.

In our study there appeared to be a relationship between intubation and anesthesia-induced decrease in FRC despite sustained ribcage muscle activity. We suggest that coughing, which occurred much more frequently and intensely in intubated subjects and rarely, if ever, in those fitted with a mask, may have been responsible. Five of 6 subjects coughed sufficiently to produce a temporary shift in the RespiTrace® baseline recording following endotracheal intubation. The mechanism by which coughing may have influenced FRC reduction is unknown, but may involve airway irritant reflex changes in both inspiratory and expiratory muscle activity.²⁰

STUDY DESIGN

Several methodologic points require mention. It has been shown that mouthpiece and nose-clip can change tidal volume and inspiratory flow.²¹ The effect was minimal during CO_2 breathing. No data are available on the effect of this apparatus on FRC, as FRC measurements cannot be made without a mask or mouthpiece of some sort. However, a comparison of RespiTrace® tidal volume data in our patients with and without mouthpiece and nose-clip or face mask revealed at most a 5-10% increase in tidal volume in those with the apparatus.

We believe that our calibration and use of the RespiTrace® apparatus gave accurate measures of compartmental ventilation. Some question does remain concerning the magnitude of the change in volume-motion coefficients with induction of anesthesia. In view of the excellent correlation between spirometer and RespiTrace® tidal volumes, we believe that any errors in relative compartmental contribution are small.

The depth of anesthesia achieved in the present study was defined by serum methohexital levels of 6.6 µg/ml (*i.e.*, within the "therapeutic window" of 3.4–10.7 µg/ml).²² Second, the level of ventilatory depression with methohexital was about the same as seen with halothane at 1 MAC (50% reduction in slope of CO₂ response curve).^{2,4} Finally, varying depth of methohexital anesthesia as assessed by varying tidal volume during induction and after supplemental bolus injections, did not produce any significant change in per cent ribcage contribution to tidal volume. Total dose of methohexital similarly had no relationship to per cent ribcage contribution. This supports our impression that even if anesthetic depth varied between patients or in an individual patient our conclusion that ribcage activity is preserved would not be altered.

IMPACT ON GAS EXCHANGE

The significant inverse correlation between changes in $P(A - a)_{O_2}$ and FRC supports the hypothesis that there is a significant cause and effect relationship between FRC reduction and impaired gas exchange. This argument is weakened, however, by the absence of a statistically significant difference in P_{aO_2} or $P(A - a)_{O_2}$ between intubated and face mask groups. We suspect that this was due to both variability in respiratory depression and to the relatively small FRC reduction in one-half the intubated subjects. In any event, the average 12 mmHg increase in $P(A - a)_{O_2}$ was substantially less than the 30 mmHg increase reported by Hewlett *et al.* during spontaneous respiration breathing halothane/air at an $F_{I_{O_2}}$ of 0.35.²³

We conclude that methohexital anesthesia used without endotracheal intubation does not produce clinically significant FRC reduction. This may be related to the observation that methohexital does not alter the functional coordination of the ribcage and diaphragm muscles.

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