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Ventilatory Pattern and Chest Wall Mechanics during Ketamine Anesthesia in Humans

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The effects of anesthetic doses of ketamine (iv bolus of 3 $mg \cdot kg^{-1}$ followed by a continuous infusion of 20 $\mu g \cdot kg^{-1} \cdot min^{-1}$) on functional residual capacity (FRC) measured by the helium dilution method and on the breathing pattern recorded by a noninvasive method (NIM) based on chest wall circumference changes were studied in 14 ASA P.S. I patients. Ketamine anesthesia was associated with: 1) the maintenance of FRC, minute ventilation, and tidal volume; 2) an increase in rib cage contribution to tidal breathing; and 3) an alteration of volume–motion relationships of the chest wall compartments. It is concluded that: 1) in contrast to volatile anesthetic agents, ketamine anesthesia has a sparing effect on intercostal muscle activity, which may explain the maintenance of FRC; and 2) changes in chest wall geometry and compliance induced by anesthetic agents must be taken into account for NIM to be valid. (Key words: Anesthetics, intravenous: ketamine. Ventilation: functional residual capacity; pattern of breathing; thoraco–abdominal partitioning.)

SEVERAL STUDIES have demonstrated that the use of a mouthpiece plus nose clip or face mask for ventilatory measurements induces important changes in breathing pattern.[1–3] Therefore, external or noninvasive measurements of chest wall movements are used to assess lung volume changes and their time components, without disturbing spontaneous breathing. In addition, these noninvasive methods provide qualitative information on the partitioning of tidal volume (V_t) between abdominal and rib cage compartments and allow measurements in resting chest wall volumes under different conditions.

The use of such methods in anesthesia, however, raises a methodologic problem. Change in chest wall geometry induced by anesthetic agents may modify the relation between external chest wall dimensions and lung volumes (volume–displacement relationship). This point, neglected in most previous studies,[4–6] is important for two reasons. First, the accuracy in V_t and partitioning measurements obtained by noninvasive methods depends on the accuracy of the volume–motion relationship. Second, it may partly explain why no change in chest wall external dimensions has been detected when thoracic gas volume was reduced.[5,6] Thoraco–abdominal blood pooling and lack of sensitivity in techniques used are the remaining hypotheses explaining this phenomenon.

The aim of this study was to investigate, by a noninvasive method, the respiratory effects of anesthetic doses of ketamine, with particular attention to possible change in the volume–displacement relationship between awake and ketamine periods.

Materials and Methods

PATIENTS

Fourteen unpremedicated ASA P.S. I male subjects (mean age [±SD] 28 yr [±6.4]) were studied before and during ketamine anesthesia, prior to minor orthopedic surgery. None of the subjects was overweight. Mean height and weight (±SD) were, respectively, 1.69 m (±0.11) and 66 kg (±9.7). This study was approved by our institutional ethics committee, and subjects gave informed consent. Anesthesia was induced with 3 $mg \cdot kg^{-1}$ ketamine injected intravenously over 1 min. Succinylcholine (1 $mg \cdot kg^{-1}$) was injected to facilitate orotracheal intubation after lidocaine 5% had been sprayed into the larynx and trachea. All patients were intubated with a Portex® 8.5 mm cuffed endotracheal tube. Anesthesia was maintained with a continuous infusion of ketamine (20 $\mu g \cdot kg^{-1} \cdot min^{-1}$). After recovery of spontaneous breathing, patients breathed a 40% O_2/air gas mixture.

At the end of the study, before surgery, general anesthesia was continued as required with diazepam, fentanyl, and N_2O.

METHODS

Thoracic and Abdominal Dimensions. Changes in rib cage (RC) and abdominal (AB) circumferences were simultaneously measured using two differential linear transformers (DLT) connected to belts. DLT is an electromechanical sensor (Shaewitz, Orgeval, France) comprised of two parts: 1) a hollow, cylindrical support with one primary and two secondary internal, symetrically mounted coils; and 2) a ferromagnetic core that moves freely inside the support and thus creates a magnetic flux variation between the coils.

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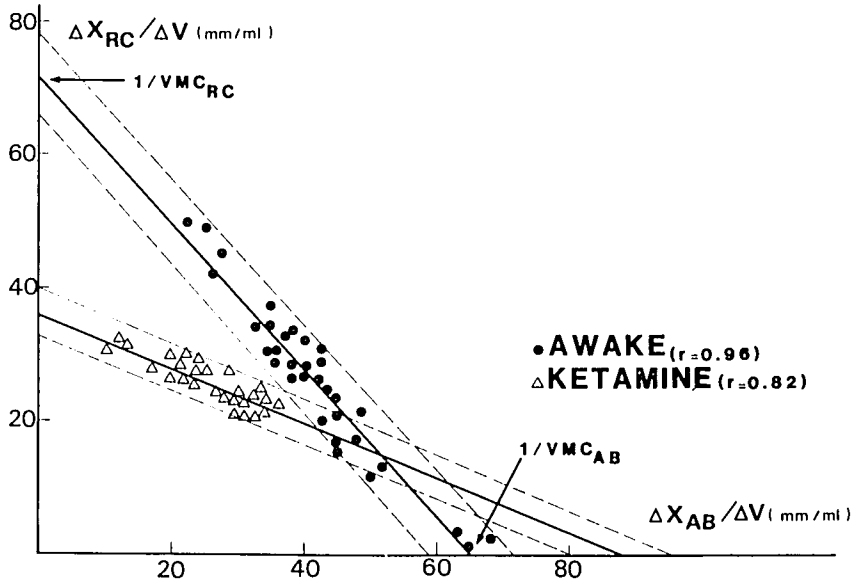

FIG. 1. Calibration represented in a function plane. This graphic representation enables improved visualization of the large different partitions between RC and AB during the calibration procedure. The regression line (*continuous line*) is obtained by a least-square method. For simplicity, all data have not been represented.

The belts are made of a 2-cm wide, nonextensible tissue with a 4-cm long piece of rubber fixed in the middle, thus enabling elastic recoil with respiratory movements. The electrical output signal induced by the displacement of the core inside the transformer during respiratory movements is linearly related to chest wall circumference changes. Its principle properties are high sensitivity (525 mV/mm) and high stability without time drift (1 mV/24 h).[7,8]

Two levels of measurement were used, each corresponding to a compartment, at the level of the nipples for the RC and 2 cm above the umbilicus for the AB. Signals were recorded on a four-channel Gould® Brush 2400 strip chart recorder.

Calibration Procedure. A calibration was performed in each state (awake and ketamine) to calculate volume–motion coefficients for RC and AB. According to the hypotheses of Konno and Mead,[9] the chest wall may be considered a physical system with two independent moving parts, the RC and the AB. Pulmonary volume change (ΔV) can be related to dimension changes (ΔX) for the RC and AB compartments by:

$$\Delta V = VMC_{RC} \cdot \Delta X_{RC} + VMC_{AB} \cdot \Delta X_{AB} \quad \text{(eq. 1)}$$

where VMC_{RC} and VMC_{AB} are the volume–motion coefficients.

Dividing equation 1 by ΔV, it becomes:

$$1 = VMC_{RC} \cdot \frac{\Delta X_{RC}}{\Delta V} + VMC_{AB} \cdot \frac{\Delta X_{AB}}{\Delta V} \quad \text{(eq. 2)}$$

To obtain different values of ΔV, $\frac{\Delta X_{AB}}{\Delta V}$, and $\frac{\Delta X_{RC}}{\Delta V}$, awake patients were asked to alternate predominantly abdominal and thoracic breathing while circumference changes were matched to simultaneously measured spirometric volumes (9-l water-filled spirometer). During ketamine anesthesia, a new calibration was performed using: 1) spontaneous variations in the RC/AB partitioning and V_t that were larger than during the control period; and 2) the transitory increase in RC contribution and V_t that followed the release of short occlusions of the endotracheal tube.

From experimental recordings, $\frac{\Delta X_{AB}}{\Delta V}$ was then plotted on the x axis and $\frac{\Delta X_{RC}}{\Delta V}$ on the y axis, and a regression line was found by the least-square technique.[10,11] The intercepts of the regression line (equation 2) with the x and y axes produced, respectively, $1/VMC_{AB}$ and $1/VMC_{RC}$ (fig. 1). Furthermore, the slope of this line is the VMC_{AB}/VMC_{RC} ratio. From the obtained correlation coefficient (r), the standard deviation of the slope is calculated. Calibrations were always performed in the same supine position.

Validity of the Calibration. In this study, calibrations were performed during each period using a mean of 30 respiratory cycles and were considered satisfactory because, for the whole group of patients:

1. The ranges of volumes and RC contributions to tidal breathing ($\Delta V_{RC}/V_t$) used for the calculation of VMCs were sufficiently large (values given are mean ± SD). In awake conditions, volumes studied ranged from 410 ml (±168) to 1,370 ml (±336), and $\Delta V_{RC}/V_t$ from 15% (±11) to 72% (±7). During ketamine anesthesia, volume ranged from 300 ml (±97) to 870 ml (±250) and V_{RC}/V_t from 35% (±16) to 71% (±13).

2. The correlation coefficient between $\frac{\Delta X_{AB}}{\Delta V}$ and

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$\frac{\Delta X_{RC}}{\Delta V}$ was sufficiently high for a linear regression analysis. The mean correlation coefficient was 0.925 ± 0.033 awake and 0.862 ± 0.064 during ketamine anesthesia.

3. Volumes were accurately measured using the noninvasive method. The mean correlation coefficient for all subjects between DLTs and spirometric volumes (V_S) was 0.988 (±0.007) in awake conditions and 0.973 (±0.022) with ketamine. The relative difference between V_{DLT} and simultaneously obtained V_S was calculated in each patient as: $[(V_{DLT} - V_S)/V_S] \times 100$. The mean relative value (%) was 5.6 ± 1.4 awake and 4.6 ± 1.3 with ketamine.

Measured Ventilatory Variables. Circumference changes and corresponding VMCs permitted calculation of V_t and RC contribution to V_t ($\Delta V_{RC}/V_t$). Minute ventilation ($\dot{V}_E$) was the product of V_t and respiratory rate (RR). Changes in end-expiratory levels (ΔEEL) of RC and AB compartments between awake and ketamine anesthesia were measured.

Functional residual capacity: FRC was measured by the closed-circuit helium dilution method in eight patients using a 9-l water-filled spirometer (Mijnhardt, Odijk, Holland) with a CO_2 absorber and a helium analyzer (Mijnhardt UG-45). A single measurement was performed both awake and during anesthesia. The helium analyzer was calibrated before each measurement. By monitoring chest wall circumference changes, the patient could be connected to the circuit at the end of expiration. Oxygen was continuously added to obtain a constant end-expiratory circuit volume. A 2-min plateau in helium concentration was obtained before measurement. The interval between two measurements was 30 min. Values were corrected for BTPS conditions.

Experimental Procedure

Circumference changes of the chest wall were continuously recorded during the study while the patients remained supine. While awake, after FRC measurement and calibration of the noninvasive method, the patients were disconnected from the spirometer and left in a quiet atmosphere to allow recording of breathing pattern in steady-state conditions. Anesthesia was then induced as previously described. The calibration procedure was repeated in all patients during ketamine anesthesia after spontaneous breathing had resumed. Therefore patients were reconnected to the spirometer every 2 min. For the last eight patients, FRC was measured during this period.

For each awake subject, respiratory variables were expressed as the mean of all cycles for a 5-min period of stable breathing. In anesthetized subjects, the period studied began 5 min after spontaneous breathing had resumed and lasted 2 min. Furthermore, in eight of our 14 patients, a second set of measurements, lasting 2 min, was obtained 10 min after spontaneous ventilation had resumed.

To assess the incidence of the load offered by an 8.5 mm endotracheal tube on resting breathing, we performed the following trial in six healthy supine volunteers (mean age [±SD] 32 yr ± 2.8, weight 68.5 kg ± 2.5, height 1.73 m ± 0.03). RC and AB circumference changes were measured using two DLTs in two successive conditions: 1) with nose clip and mouthpiece; and 2) with nose clip and an 8.5 mm ID endotracheal tube externally connected to the mouthpiece to reproduce a resistance close to that of ketamine-intubated patients.

Volume–motion coefficients for RC and AB were obtained in both conditions. For each subject, respiratory variables were calculated as the mean (±SD) of all respiratory cycles during a 5-min period of quiet breathing.

Statistical Analysis

All values are reported as mean and SD. Changes in the slope of the calibration line and in VMCs between awake and ketamine states were analyzed for each subject using a Student's *t* test. Respiratory variables obtained at different periods were compared using an analysis of variance and a modified *t* test.

Results

Effects of Ketamine Anesthesia on the Slope of the Calibration Line and Volume–Motion Coefficients

Between the awake state and ketamine anesthesia, for all but one patient, there was a significant change ($P < 0.01$) in the slope of the calibration line (fig. 2) and in VMCs of the RC and the AB (fig. 3). In ten patients we observed a decrease in the slope with an increase in VMC_{RC} and a decrease in VMC_{AB}, while in four patients the slope increased with a decrease in VMC_{RC} and an increase in VMC_{AB}. The mean slope decreased significantly ($P < 0.05$) from 1.540 ± 0.372 to 1.152 ± 0.670, with a significant increase in the mean VMC_{RC} ($P < 0.02$), while the mean VMC_{AB} remained unchanged. A typical graphic representation of a change in calibration line is given in figure 1.

Effects of Ketamine on the Breathing Pattern and Partitioning

For the entire group (n = 14), when compared with control values, V_t, RR and $\dot{V}_E$ remained unchanged 5 min following recovery of spontaneous breathing (table 1). $\Delta V_{RC}/V_t$ increased significantly.

Measurements performed in eight patients at 10 min after the resumption of spontaneous breathing (table 2)

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showed a significant increase in $\dot{V}_E$ when compared with 5-min values and control values. No further significant changes were observed in the other respiratory variables when compared with 5-min values. This group of eight patients did not differ from the whole group in the respiratory variables obtained in awake conditions.

FRC (N = 8) and Chest Wall End-Expiratory Levels

Ketamine produced a significant increase ($P < 0.01$) in the end-expiratory level of the two compartments (tables 1 and 2), while the mean increase in simultaneously measured FRC (+195 ml ± 265) was not significant (table 3).

Effects of the Endotracheal Tube in Awake Subjects (Table 4)

Comparing the two conditions, with and without an 8.5 mm endotracheal tube connected to the mouthpiece:volume–motion coefficients for RC and AB, V_t, RR, $\dot{V}_E$, and end-expiratory levels remained unchanged.

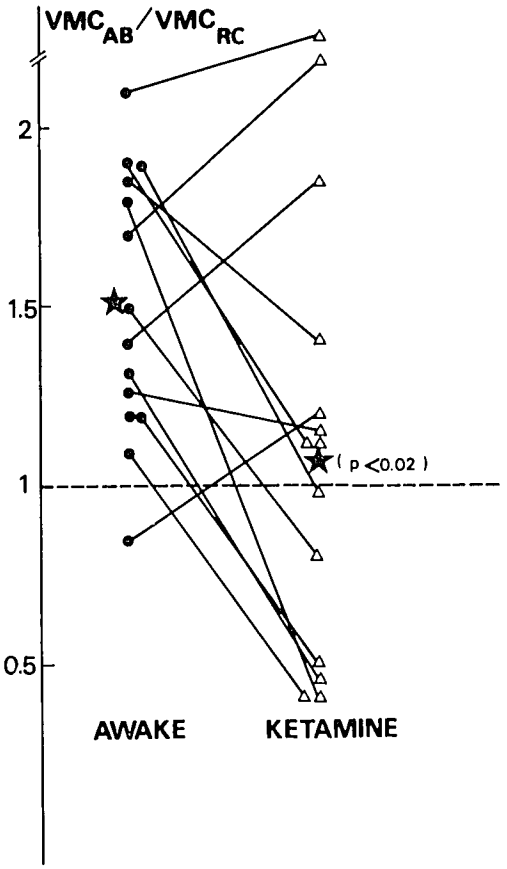

FIG. 2. Changes in individual values of the slope VMC_{AB}/VMC_{RC}, in awake patients and under ketamine anesthesia. *Stars* indicate mean values.

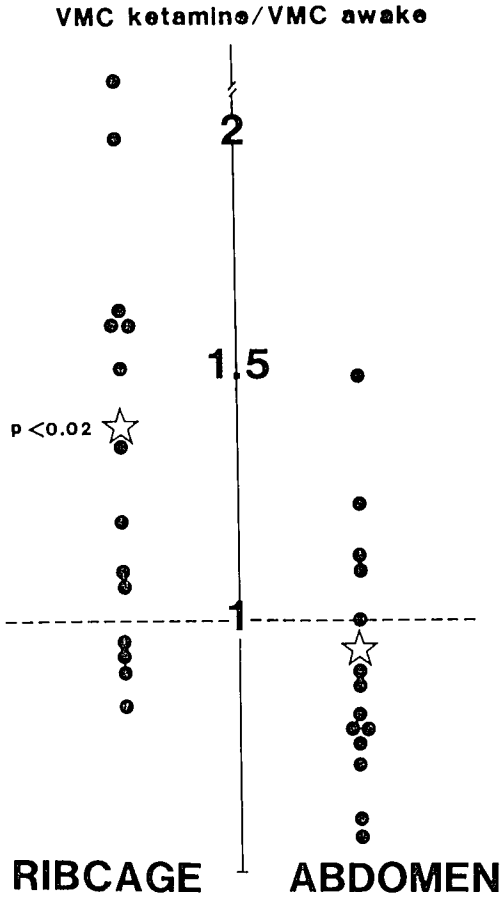

FIG. 3. Changes in volume–motion coefficients for rib cage (*left*) and abdomen (*right*) compartments expressed as a ratio of ketamine/awake coefficients. *Stars* indicate mean changes.

$\Delta V_{RC}/V_t$ was significantly ($P < 0.05$) decreased from 32% to 26%.

Discussion

Methodologic Aspects

Noninvasive measurement of ventilation using chest wall movements is based on the assumptions of Konno and Mead.[9] In their model, the chest wall may be considered a simplified physical system, with two independent moving parts during spontaneous breathing: the RC and the AB. Each compartment is characterized by a single degree of freedom and a linear relationship between volume and motion changes. Different calibration procedures[9,12] have been used to obtain the VMCs that define this relationship.

A set of VMCs is representative of a definite and stable geometric state of the respiratory system. Any change in resting chest wall geometry would affect the volume–motion relationship. Zimmerman *et al.*[13] demonstrated the

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TABLE 1. Results of the Different Ventilatory Variables in 14 Patients in Awake Conditions and under Ketamine Anesthesia 5 Min after Recovery of Spontaneous Breathing

	V_t (ml)	RR (c·min^{-1})	V_E (l·min^{-1})	$\Delta V_{RC}/V_t$ (%)	ΔEEL_{RC} (mm)	ΔEEL_{AB} (mm)
Awake	358 ± 140	16 ± 4	5.3 ± 1.5	34 ± 17	—	—
Ketamine 5 min	437 ± 180	18 ± 4	7.9 ± 4.7	52 ± 17	6 ± 4	4 ± 4
	NS	NS	NS	$P < 0.01$	$P < 0.001$	$P < 0.001$

Values are expressed as mean ± SD. NS = not significant.
V_t = tidal volume; RR = respiratory rate; $\dot{V}_E$ = minute ventilation; $\Delta V_{RC}/V_t$ = rib cage contribution to tidal volume; ΔEEL_{RC} and ΔEEL_{AB} = change in end-expiratory levels for rib cage and abdomen.

alteration in VMCs with changes in posture (from standing to semirecumbent and from semirecumbent to supine). In the same way, anesthesia may alter chest wall shape and, consequently, the volume–motion relationship of chest wall compartments. Recently, Krayer *et al.*,[14] using computed tomography and three-dimensional reconstruction of the thorax, showed that the decrease in RC volume with intravenous anesthesia was nonuniform, more pronounced in the cranial than caudal portion. Tusiewicz *et al.*[4] reported that halothane induces a preferential suppression of both tonic and phasic intercostal electromyographic activity, with a loss in RC stability characterized by an inward inspiratory movement. This effect suggests a change in chest wall shape because parasternal intercostals are essential in maintaining RC shape.[15] A change in the slope of isovolume lines, which is the VMC_{AB}/VMC_{RC} ratio, is reported in their study and suggests that the VMCs had changed between control and posthalothane conditions. The magnitude and direction of this change was not provided.

Our study clearly demonstrates the individual changes in VMCs that occur during ketamine anesthesia. For all but one patient, the calibrations obtained in the two conditions were different, because the slopes of the regression lines (VMC_{AB}/VMC_{RC}) were significantly modified and the correlation coefficients between $\Delta X_{AB}/\Delta V$ and $\Delta X_{RC}/\Delta V$ were high enough to validate the linear regressions. Moreover, in each condition and using corresponding VMCs, volumes obtained from circumference changes were always accurate when compared with volume simultaneously measured by direct spirometry (see validity of the calibration in "Methods"). In addition, the calibration with ketamine may be considered stable and representative of the period studied, because the respiratory cycles used to obtain it are taken at different times of the ketamine period and are on the same regression line for each patient.

Hypotheses that can be advanced to explain changes in VMC are related to the model of Konno and Mead[9] of two linear variables. First, two variables are insufficient to describe all the geometric behavior of the chest wall, especially during anesthesia. Second, chest wall distortion induced by anesthesia may introduce nonlinear effects in the volume–motion relationship. Consequently, the magnitude and direction of VMC changes are probably nonpredictable. Morel *et al.*[16] reported that 1 mg·kg^{-1} ketamine did not modify VMCs. However, in their study, inability to detect any change may be related to the chosen timing because the second calibration was performed 1 h after ketamine injection, when chest wall geometry had probably returned to its baseline.

The change in VMCs following anesthesia has two major consequences.

First, the accuracy of V_t measurement and its partitioning, assessed by these indirect methods, can be greatly diminished when this change is not taken into account. In our study when awake VMCs are used for the ketamine period, the error in V_t is large (11% ± 8) compared with that obtained when correct VMCs are used (4.6 ± 1.3). The resulting difference in V_t partitioning ($\Delta V_{RC}/V_t$) is

TABLE 2. Results of the Different Ventilatory Variables in 8 Patients in Awake Conditions and under Ketamine Anesthesia 5 and 10 Min after Recovery of Spontaneous Breathing

	V_t (ml)	RR (c·min^{-1})	V_E (l·min^{-1})	$\Delta V_{RC}/V_t$ (%)	ΔEEL_{RC} (mm)	ΔEEL_{AB} (mm)
Awake	300 ± 98	18 ± 4	5.2 ± 1.8	34 ± 20	—	—
Ketamine						
5 min	331 ± 115	17 ± 5	5.5 ± 2.2	53 ± 18*	6 ± 4†	3 ± 3†
10 min	407 ± 180	19 ± 4	7.3 ± 1.9‡	59 ± 15†	4 ± 4*	3 ± 3*

See footnote to table 1 for abbreviations.
Values are expressed as mean ± SD.
* $P < 0.05$.
† $P < 0.01$ *vs.* control.
‡ $P < 0.05$ ketamine 10 min *vs.* ketamine 5 min.

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much greater (22% ± 15). Indeed, the opposite change in the two VMCs induced by ketamine partially cancels the consequence of this change on V_t calculation.

The second consequence is related to the translation of ΔEEL in terms of chest wall end-expiratory volume changes.

The two-variable model of Konno and Mead is only valid when estimating volume changes in stable geometric states. This is the case for awake supine patients or during ketamine anesthesia, but the change in chest wall end-expiratory volume between the two states cannot be accurately inferred from ΔEEL_{AB} and ΔEEL_{RC} because VMCs have changed. In such circumstances, the two-variable model is methodologically insufficient in translating changes in external dimensions in terms of volume. In order to obtain a calibration independent of geometric changes, additional independent variables are required to characterize the chest wall.

The alteration in chest wall shape associated with an increase in central blood volume[14,17] may explain the lack of parallelism between changes in chest wall end-expiratory volumes obtained from chest wall end-expiratory levels and changes in FRC induced by anesthesia.[5,6]

In this study, an 8.5 mm cuffed endotracheal tube was used in all patients and might have contributed to changes observed during the ketamine period because this apparatus results in inspiratory and expiratory resistances.[18] Our trial, performed in awake subjects to assess the incidence of the load offered by the tube, shows no change in resting breathing and VMCs. The slight decrease in $\Delta V_{RC}/V_t$ observed is opposite to the large increase in $\Delta V_{RC}/V_t$ during ketamine anesthesia. In addition, changes in breathing pattern, partitioning, and end-expiratory levels observed with 1 $mg \cdot kg^{-1}$ ketamine and without an endotracheal tube[16] followed the same pattern as in the present study. The degree of change in respiratory variables between the two studies is different either because doses of ketamine used were different and/or the change in VMCs had not been taken into account. Changes in breathing pattern, RC/AB partitioning, and end-expiratory volumes in ketamine-intubated patients are exactly the opposite of changes observed in halothane-intubated patients,[4] suggesting important differences in action on respiratory muscles between the two anesthetics despite the fact that an endotracheal tube was present in both studies.

An endotracheal tube may also be seen as an irritant factor upon tracheal mucosa. In our patients, the upper airways were probably adequately anesthetized after lidocaine 5% spray, because neither cough nor irritation was noted during and after positioning of the tube. In addition, airway anesthesia with lidocaine aerosol did not change breathing patterns in normal subjects.[19]

We conclude from both the literature and our own trial that the flow resistance offered by an 8.5 mm endotracheal tube cannot be incriminated in changes observed in V_t, RR, partitioning, end-expiratory levels, and VMCs in our ketamine-intubated patients, probably because the mean inspiratory flow of our anesthetized subjects was relatively low (range 170–300 $ml \cdot s^{-1}$).

TABLE 3. Individual and Mean Values of Functional Residual Capacity (FRC)

Patient No.	FRC (ml)		
	Awake	Ketamine	ΔFRC
7	2,160	2,340	+180
8	2,775	2,730	−45
9	1,740	2,030	+290
10	1,870	2,050	+180
11	2,030	2,090	+60
12	3,010	3,140	+130
13	2,280	2,210	−70
14	2,215	3,050	+835
Mean	2,260	2,455	+195
SD	405	425	265

FUNCTIONAL RESIDUAL CAPACITY

This study showed that FRC, measured by the helium dilution method, remained unchanged during ketamine anesthesia in adults. This is consistent with a previous study in children.[20] While there were large individual variations (from −3% to +37%), the mean FRC increase (+10%) is in contrast with the classic decrease (−18%) reported either after intravenous or inhalation anesthesia.[21] This mean 10% increase in FRC is probably un-

TABLE 4. Effect of 8.5 Endotracheal Tube Externally Connected to the Mouthpiece (six supine subjects) on Respiratory Variables and Volume–Motion Coefficients (mean ± SD)

	Nose Clip + Mouthpiece	Nose Clip + Mouthpiece + 8.5 Tube	P
V_t (ml)	570 ± 108	583 ± 117	NS
RR ($c \cdot min^{-1}$)	11.5 ± 3	10 ± 3	NS
$\dot{V}_E$ ($l \cdot min^{-1}$)	6.4 ± 1.4	5.8 ± 1.3	NS
$\Delta V_{RC}/V_t$ (%)	32 ± 9	26 ± 7*	<0.05
ΔEEL_{AB} (mm)	0.0 ± 0.3	0.0 ± 0.3	NS
ΔEEL_{RC} (mm)	0.5 ± 0.68	0.4 ± 0.5	NS
VMC_{AB} (ml/mm)	81 ± 10	80 ± 12	NS
VMC_{RC} (ml/mm)	94 ± 43	92 ± 49	NS
VMC_{AB}/VMC_{RC}	1.01 ± 0.42	1.04 ± 0.43	NS
Correlation coefficient (ΔXAB/ΔV, ΔXRC/ΔV)	0.95 ± 0.06	0.95 ± 0.05	NS
Correlation coefficient (V_{DLT}, V_S)	0.99 ± 0.01	0.98 ± 0.01	NS

See table 1 and text for abbreviations.
* $P < 0.05$.

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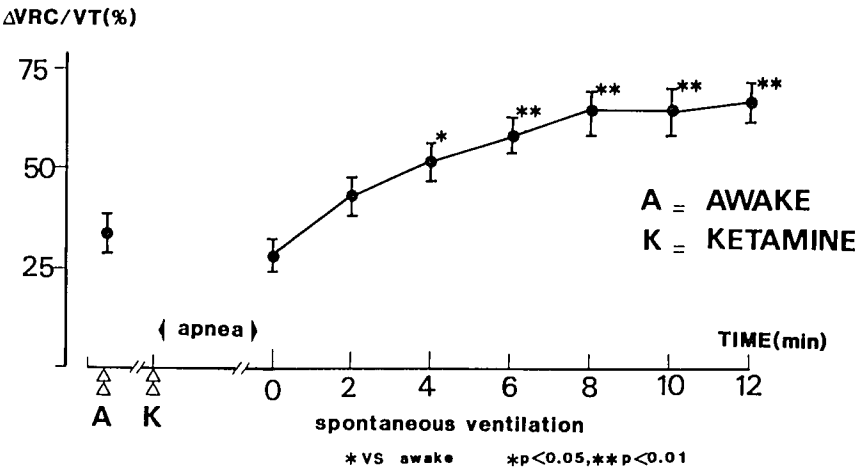

FIG. 4. Time course of rib cage contribution to tidal volume during the study in 8 subjects. *Stars* indicate significant difference compared with awake conditions.

derestimated, because the spirometer during the ketamine period was filled with 21% O_2 while the subject breathed a 40% O_2/air gas mixture. Values given in this study were not corrected. The influence of O_2 concentration on our helium analyzer was measured, and the difference between fractional inspired oxygen content (FI_{O_2}) 0.21 and 0.40 was 110 ml (5%).

A decrease in outward recoil of the chest wall induced by the suppression of respiratory muscle tonic activity has been suggested as a hypothesis to explain the decrease in FRC observed with general anesthesia.[22] Thus, loss of tonic electromyographic activity of the diaphragm[23] and intercostal muscles[4] has been demonstrated with halothane. Other studies have shown a cephalad shift of the diaphragm[14,17,24] and a decrease in internal diameters[14] of the RC with intravenous anesthesia. In our study, the important increase in RC contribution to tidal breathing and the increase in resting end-expiratory position of the RC compartment suggest at least a sparing effect on the RC stability and intercostal function. This may, in part, explain the maintained FRC.

FRC stability should have a beneficial effect on gas exchange because a correlation has been found between FRC during anesthesia and alveolar–arterial oxygen difference.[25] The absence of change in Pa_{O_2}, which has been found in ketamine-anesthetized subjects,[26,27] is probably related to FRC stability.

Thoraco–Abdominal Partitioning

Ketamine anesthesia was associated with an important increase in RC motion index, $\Delta V_{RC}/V_t$ (mean increase +55%). Changes in respiratory muscle phasic activity however, cannot directly be inferred from changes in compartmental contributions. Indeed, chest wall movements are not only dependent on the phasic activity of respiratory muscles (movement function), but also on the relative compliance and stability of the two compartments. These are, in part, determined by the tonic activity of these respiratory muscles (postural function). For example, in seated patients, RC motion becomes predominant (approximately two-thirds of V_t), while the diaphragm remains the main inspiratory muscle.[28,29] The change in chest wall motion is partly related to the decreased AB compliance with increased tonic activity of AB muscles.[29] In our study, the changes in VMCs and VMC ratio induced by ketamine indicate a change in chest wall geometry. Therefore, different mechanisms may account for the increased RC motion: 1) recruitment of RC inspiratory muscles; 2) increased AB wall tone with decreased AB compliance; and/or 3) decreased phasic diaphragmatic activity.

The important increase in RC contribution associated with increased V_t did not support the hypotheses of decreased phasic diaphragmatic activity and/or decreased AB compliance as main determinants of this ventilatory mode. Although simultaneous electromyographic studies were not performed, these results strongly suggest recruitment of intercostal muscles following ketamine anesthesia. Consequently, ketamine effects on chest wall motion are contrary to those reported with volatile anesthetics,[4,6,30] which are characterized by marked impairment of intercostal muscle function.

Because succinylcholine was used, the relationship between the residual effect of this drug and the respiratory effects of ketamine reported in our study may be questioned. Indeed, after induction of anesthesia, apnea lasted for an average of 4.5 min and was followed by a progressive increase in V_t and $\Delta V_{RC}/V_t$ until a plateau was reached 5 min later. Thereafter, $\Delta V_{RC}/V_t$ remained stable (fig. 4). This initial ascending phase in RC contribution to tidal breathing may be related to the residual effect of succinylcholine. However, the respiratory effect of ketamine was obtained 5 and 10 min after spontaneous breathing had resumed and during a stable period. Moreover, partial relaxation cannot account for increased V_t

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and $\Delta V_{RC}/V_t$, because it has effectively been shown to produce an opposite effect.[31] In conclusion, this study demonstrates consistent changes in RC and AB VMCs during ketamine anesthesia. These changes must be taken into account in order to measure accurately V_t and partitioning during anesthesia and impede the calculation of chest wall end-expiratory volume changes.

The principal respiratory effects of ketamine are the maintenance of $\dot{V}_E$, RC stability, and FRC. These effects oppose those reported with volatile anesthetic agents and are of interest because ketamine is frequently used without mechanical ventilation.

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References

1. Gilbert RJ, Auchincloss JH, Brodsky J, Boden W: Changes in tidal volume, frequency and ventilation induced by their measurement. J Appl Physiol 33:252–254, 1972
2. Askanazi JP, Silverberg A, Foster RJ, Hyman AI, Milic-Emili J, Kinney JM: Effects of respiratory apparatus on breathing pattern. J Appl Physiol 48:577–580, 1980
3. Weissman C, Askanazi J, Milic-Emili J, Kinney JM: Effects of respiratory apparatus on respiration. J Appl Physiol 57:475–480, 1984
4. Tusiewicz K, Bryan AC, Froese AB: Contributions of changing rib cage–diaphragm interactions to the ventilatory depression of halothane anesthesia. ANESTHESIOLOGY 47:327–337, 1977
5. Hedenstierna G, Lofstrom B, Lundh R: Thoracic gas volume and chest–abdomen dimensions during anesthesia and muscle paralysis. ANESTHESIOLOGY 55:499–506, 1981
6. Jones JG, Faithfull D, Jordan C: Rib cage movement during halothane anesthesia in man. Br J Anaesth 51:399–407, 1979
7. Simonneau G, Vivien A, Sartene R, Kunstlinger F, Samii K, Noviant Y, Duroux P: Diaphragmatic dysfunction induced by upper abdominal surgery. Role of postoperative pain. Am Rev Respir Dis 128:899–903, 1983
8. Rouby JJ, Simonneau G, Benhamou D, Sartene P, Sardnal F, Deriaz H, Duroux P, Viars P: Factors influencing pulmonary volumes and CO_2 elimination during high-frequency jet ventilation. ANESTHESIOLOGY 63:473–482, 1985
9. Konno K, Mead J: Measurement of the separate volume changes of rib cage and abdomen during breathing. J Appl Physiol 22: 407–422, 1967
10. Chada TS, Watson H, Birch S, Jenouri GA, Schneider AN, Cohn MA, Sackner MA: Validation of respiratory inductive plethysmography using different calibration procedures. Am Rev Respir Dis 125:644–649, 1982
11. Dolfin T, Duffty P, Wilkes DL, Bryan H: Calibration of respiratory induction plethysmography in infants. Am Rev Respir Dis 126: 577–579, 1982
12. Stagg D, Goldman MD, Newson-Davis J: Computer-aided measurement of breath volume and time components using magnetometers. J Appl Physiol 44:623–633, 1978
13. Zimmerman PV, Connellan SJ, Middleton HC, Tabona MV, Goldman MD, Pride N: Postural changes in rib cage and abdominal volume–motion coefficients and their effect on the calibration of a respiratory inductance plethysmograph. Am Rev Respir Dis 127:209–214, 1983
14. Krayer S, Rehder K, Beck KC, Hoffman EA, Didier EP, Ritman EL: Why is the functional residual capacity reduced with general anesthesia (abstract)? ANESTHESIOLOGY 63:A557, 1985
15. De Troyer A, Estenne M: Coordination between rib cage muscles and diaphragm during quiet breathing in humans. J Appl Physiol 57:899–906, 1984
16. Morel D, Forster A, Gemperle M: Noninvasive evaluation of breathing pattern under infusion of ketamine and droperidol in volunteers (abstract). ANESTHESIOLOGY 59:A493, 1983
17. Hedenstierna G, Strandberg A, Brismar B, Lundquist H, Svensson L, Tokics L: Functional residual capacity, thoracoabdominal dimensions and central blood volume during general anesthesia with muscle paralysis and mechanical ventilation. ANESTHESIOLOGY 62:247–254, 1985
18. Zin WA, Behrakis PK, Wijendijk SCM, Higgs BD, Baydur A, Boddener A, Milic-Emili J: Immediate response to resistive loading in anesthetized human. J Appl Physiol 60:506–512, 1986
19. Cross BA, Guz A, Jain SK, Archer S, Stevens J, Reynolds F: The effect of anesthesia on the airway in dog and man: A study of respiratory reflexes, sensations and lung mechanics. Clin Sci Mol Med 50:439–454, 1970
20. Shulman D, Beardsmore CS, Aronson HB, Godfrey S: The effect of ketamine on the functional residual capacity in young children. ANESTHESIOLOGY 62:551–556, 1985
21. Rehder K, Sessler AD, Marsh HM: General anesthesia and the lung. Am Rev Respir Dis 112:541–563, 1975
22. Westbrook PR, Stubbs SE, Sessler AD, Rehder K, Hyatt RE: Effects of anesthesia and muscle paralysis on respiratory mechanics in normal man. J Appl Physiol 34:81–86, 1973
23. Muller N, Volgyesi G, Becker L, Bryan MH, Bryan AC: Diaphragmatic muscle tone. J Appl Physiol 47:279–284, 1979
24. Froese AB, Bryan Ch: Effects of anesthesia and paralysis on diaphragmatic mechanics in man. ANESTHESIOLOGY 41:242–255, 1974
25. Bergman NA, Tien YK: Contribution of the closure of pulmonary units to impaired oxygenation during anesthesia. ANESTHESIOLOGY 59:395–401, 1983
26. Gooding JM, Dimick AR, Tavakoli M, Corssen G: A physiologic analysis of cardio pulmonary responses to ketamine anesthesia in non cardiac patients. Anesth Analg 55:813–816, 1977
27. Tweed WA, Minuck M, Mymin D: Circulatory responses to ketamine anesthesia. ANESTHESIOLOGY 37:613–619, 1972
28. Vellody VP, Nassery M, Druz WS, Sharp JT: Effects of body position change on thoracoabdominal motion. J Appl Physiol 45: 581–589, 1978
29. Druz WS, Sharp JT: Activity of respiratory muscles in upright and recumbent humans. J Appl Physiol 51:1552–1561, 1981
30. Cantineau JP, Mankikian B, Poete P, Sartene R, Clergue F, Viars P: Ventilatory pattern and chest wall mechanics during isoflurane anesthesia (abstract). ANESTHESIOLOGY 63:A551, 1985
31. De Troyer A, Bastenier J, Delhez L: Function of respiratory muscles during partial curarization in humans. J Appl Physiol 49: 1049–1056, 1980

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