RIB CAGE MOVEMENT DURING HALOTHANE ANAESTHESIA IN MAN

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SUMMARY
Chest wall movement, partitioned into rib cage and abdomen/diaphragm contributions, was measured using four mercury-in-rubber strain gauges and an analog computer. The relative volume contribution of the rib cage and the abdomen/diaphragm to tidal volume was measured in 13 subjects before and during anaesthesia with thiopentone and halothane. In awake subjects, movement of the abdomen/diaphragm contributed more than 70% of the tidal volume, the smaller rib cage contribution ranged from 5 to 30%. Manual ventilation during induction of anaesthesia showed that the rib cage was less compliant than the abdomen/diaphragm, but when suxamethonium was given there was a disproportionate increase in rib cage compliance. In nine out of 12 subjects, halothane anaesthesia resulted in a large decrease in the fractional contribution of the rib cage. In two of these subjects there was paradoxical breathing, the rib cage and the abdomen/diaphragm movement being 180° out of phase. This effect was produced easily in three other subjects by inserting a resistance (1 kPa litre⁻¹ s⁻¹) into the anaesthetic circuit. The technique also produced information about changes in volume of the trunk induced by anaesthesia. Eleven subjects showed an increase in end-expiratory abdominal volume (mean increase 120 ml) during halothane anaesthesia while there was a mean reduction in end-expiratory rib cage volume of 29 ml. We concluded that halothane depressed both phasic and tonic postural reflex activity, which affected predominantly the rib cage musculature. This reduced the amplitude of phasic rib cage movement, impaired stability of the rib cage and predisposed to paradoxical ventilation. The results also suggested that the reduction in lung volume during anaesthesia may result from a loss of postural control of the chest wall and a central shift of blood volume.

Since the pioneering work of Snow in 1858 and of Miller (1925) and Burstein (1942), it has been known that intercostal muscle paralysis accompanies inhalation anaesthesia. Despite recent advances in the understanding of rib cage and abdomen/diaphragm function, described in the important papers of Mead (1974) and Goldman (1974), and with the exception of the recent study of Tusiewicz, Bryan and Froese (1977), there has been a paucity of new information on the effect of inhalation anaesthetics on chest wall function in man.

The chest wall is subdivided into the rib cage and abdomen/diaphragm, which have both supportive and ventilatory functions. These two functions are closely integrated by the central nervous system and their efficacy depends upon two types of motor neuron input, tonic and phasic. The importance of the tonic, non-ventilatory input is less widely appreciated than the phasic input, but it is this type of motor activity which plays a fundamental role in stabilizing the shape of the rib cage so that the function of the diaphragm is optimized (DaSilva et al., 1977). This study was designed to measure the relative volume contributions of the rib cage and abdomen/diaphragm to tidal breathing and to demonstrate that the selective effect of halothane on rib cage movement had important consequences on rib cage stability, the loss of which was an important factor explaining the decrease in lung volume which follows induction of anaesthesia.

METHODS
Movement of the chest wall was measured using four mercury-in-rubber strain gauges, two around the rib cage and two around the abdomen. From measurements of the circumference of the trunk at these four locations, the height of each section and the change in circumference, it was possible to compute the relative volume contributions to tidal breathing of the rib cage and abdomen/diaphragm using an analog computer method described previously (Faithfull, Jones and Jordan, 1979). Measurements of tidal breathing at the mouth were made...
using a heated Fleisch II pneumotachograph connected to either a mouthpiece or an endotracheal tube. The pressure decrease across the pneumotachograph was measured with a Validyne transducer, Model MP 45, pressure range ± 0.25 kPa (Northridge, California). Data from the analog computer included signals proportional to the tidal volume contribution of rib cage movement, abdominal wall movement and sum of rib cage and abdomen. These signals, together with flow from the pneumotachograph, were recorded using a modified Akai audio tape recorder, the input and output signals being connected via an eight-channel multiplexer unit. Data were replayed off-line onto a Devices chart recorder, tidal volume at the mouth being obtained by electronic integration of the tape-recorded flow signal.

Procedure
Thirteen patients (mean age 40.4 yr) undergoing routine surgery gave informed consent for the study. Pre-anaesthetic medication comprised atropine alone. All patients were studied in the supine position. Gauges were applied to the trunk and baseline measurements of circumference and trunk height were set up on the potentiometers of the analog computer as described previously (Faithfull, Jones and Jordan, 1979). The patients breathed through a pneumotachograph for control measurements of tidal volume, and for partitioning tidal breathing into the rib cage and the abdomen/diaphragm contributions.

Anaesthesia comprised a standard technique with thiopentone, suxamethonium, nitrous oxide, oxygen and halothane. Measurements of chest wall movements were made continuously and end-tidal halothane concentrations were measured using a Hook and Tucker ultraviolet absorption meter modified to give a 90% rise time of 100 ms to a square-wave input of halothane. The inspired concentration of halothane was adjusted to yield end-tidal concentrations of 0.5, 1.0 and 2.0%. The fractional contribution of the rib cage to tidal breathing at each alveolar halothane concentration was obtained from X–Y plots of rib cage against the abdomen/diaphragm (AD) contribution obtained at different stages of the anaesthetic procedure.

RESULTS

Rib cage and abdomen/diaphragm movement before and immediately after induction

The mean rib cage contribution to tidal breathing before induction was 14.6% (n = 13). Following induction of anaesthesia, rib cage contribution was measured during manual inflation of the lungs. In nine patients measurements were made after the administration of thiopentone alone. In five of these patients measurements were repeated after the administration of suxamethonium. In the remaining four patients measurements were made only after the combined administration of thiopentone and suxamethonium. Changes in rib cage contribution are shown in figure 1. The rib cage contribution to tidal volume increased with manual inflation during thiopentone anaesthesia and increased further following neuromuscular blockade with suxamethonium (table I).

These changes may also be expressed as relative compliance (RC/AD) during manual inflation. This ratio increased after suxamethonium (fig. 2). The

![Graph showing fractional volume contribution of the rib cage to tidal volume in 13 patients before and after induction of anaesthesia. All data obtained following induction of anaesthesia were during manual inflation of the lungs. ■ = awake; ○ = anaesthetized; □ = anaesthetized and paralysed.](https://academic.oup.com/bja/article-abstract/51/5/399/248065)

![Table I. Mean (+1 SEM) differences in rib cage contribution, shown in figure 1, on transition from spontaneous ventilation awake to manual ventilation anaesthetized with thiopentone (column 1), thiopentone followed by suxamethonium (column 2) and from awake to anaesthetized with thiopentone and suxamethonium given in rapid succession (column 3).](https://academic.oup.com/bja/article-abstract/51/5/399/248065)
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FIG. 2. Relative rib cage compliance derived from the data in figure 1 during manual inflation. • = anaesthetized; O = anaesthetized and paralysed.

Elimination of suxamethonium was accompanied by a decrease in compliance of the rib cage and thus returned to the values obtained previously with thiopentone alone. Manual ventilation was usually accompanied by an increase in lung volume which was seen as a shift in baseline of the rib cage and abdominal traces. If the lungs were inflated without permitting the pressure in the airway to decrease to atmospheric, a characteristic series of changes were observed. After an initial increase in rib cage and abdominal girth, the rib cage girth decreased to its original value and the abdomen continued to increase in size. Within a few seconds the circulation had adjusted so that both rib cage and abdomen changed in circumference together.

Phase relationships at onset of and recovery from anaesthesia

Nearly all subjects showed a narrow loop and thus a close-phase relationship between rib cage and abdominal movement when awake and when anaesthetized. An example of this pattern is shown in one subject in figure 3. A few subjects showed larger phase differences with quite wide loops while awake, but during anaesthesia the loop area in every subject always became much less (fig. 4). Immediately following administration of thiopentone there was either a small reduction in rib cage contribution, the rib cage–abdomen loop resembling that seen with low concentrations of halothane, or there was a transient large phase difference with wide loops progressing rapidly to apnoea. Onset of spontaneous respiration with halothane showed a reduction in the area of the rib cage–abdomen loop, a diminished rib cage contribution and in most instances the inspiratory and expiratory parts of the loop were superimposed. On recovery from anaesthesia (fig. 3) there was usually gross phase lag between the two signals which often lasted as long as 1 min before reappearance of the phase relationship that characterized the pre-anaesthetic control pattern.

FIG. 3. X–Y plots of volume contributions of rib cage and abdomen/diaphragm. The gain for each signal was the same. This patient showed a close phase relationship both before anaesthesia (control) and with 0.5% alveolar halothane. There was a much larger asynchrony of rib cage and abdomen during induction of anaesthesia and particularly during recovery from anaesthesia. When awake at the end of the anaesthetic the loops were similar to control.

Control
Thio
Hal 0.5%
Recovery
Awake

FIG. 4. Loops of larger areas were seen in control breaths of some patients. An example of this pattern is shown in this patient in whom, on induction with thiopentone (Thio), the rib cage and abdomen/diaphragm became 90° out of phase just before ventilation ceased. Also shown are the relative contributions of rib cage and abdomen during manual ventilation (MV) and during halothane anaesthesia. Note that the change in slope of the loop with halothane anaesthesia indicates a reduction in rib cage activity.

Effect of "steady-state" halothane anaesthesia on chest wall movement

In nine of 12 subjects there was a reduction in rib cage contribution with halothane anaesthesia. This was related to the alveolar halothane concentration in such a way that 1% alveolar halothane produced a mean reduction of 50% in rib cage contribution compared with awake controls (fig. 5). Two per cent alveolar halothane resulted in a further reduction in rib cage contribution. In two subjects the rib cage and abdomen became opposite in phase, so that there was a negative contribution of the rib cage.
and it was pulled inwards by the contracting diaphragm.

There was also a reduction in tidal volume (fig. 6) and an increase in breathing frequency in all subjects. The three subjects showing an increase in rib cage contribution had particularly small tidal volumes (<100 ml) and larger breathing frequencies (>60 b.p.m.); one of these subjects had the largest pre-anaesthetic tidal volume (1400 ml) and the smallest tidal volume (85 ml) with 2% alveolar halothane.

**Effect of resistive loading**

In three subjects a resistive load of 1 kPa litre\(^{-1}\) s was added to the breathing circuit during the control period and then during anaesthesia. No change in tidal volume or in the relative contribution of the rib cage was noted during the control period. During anaesthesia there was an immediate decrease in tidal volume and the rib cage contribution became opposite in phase to movement of the abdomen/diaphragm. As carbon dioxide drive to respiration increased there was an increase in abdominal excursion and a further indrawing of the rib cage during inspiration.

In these subjects the mean rib cage contribution before the load was 2% of the tidal volume. Immediately after addition of the load there was an immediate decrease in tidal volume with a negative rib cage contribution of 3% of the tidal volume which, with increasing carbon dioxide drive showed further paradoxical movement, negative rib cage contribution increasing to a mean of 10%. This implied an emptying of alveoli in the upper part of the chest during diaphragmatic inspiration, thus adding alveolar gas to the inspired deadspace.

**Effect of anaesthesia on the end-expiratory chest wall position**

The end-expiratory level of the rib cage and abdomen/diaphragm signals was found by a line of best fit drawn through a 10-min control period before anaesthesia and a similar period before the end of the study. Only one subject showed a reduction of rib cage and abdominal volume. Eleven subjects had an increase in abdominal volume in the range 25–250 ml (mean increase 120 ml) (fig. 7). The mean change in chest volume for all the subjects was a decrease of 29 ml. An example of a typical trace is shown in figure 8 where the changes in trunk, rib cage and abdominal volume before, during and after the induction of anaesthesia are illustrated. An increase in end-expiratory rib cage and abdominal volume was seen during manual ventilation following apnoea induced by thiopentone. Subsequently there was a decrease in end-expiratory lung volume to just below control value and then, by the time that the study was discontinued, there was an increase in
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**DISCUSSION**

This study has shown that halothane anaesthesia depressed the amplitude of rib cage and diaphragm movements in all the patients investigated. In nine of the 12 patients this effect was most marked on the rib cage contribution, there being a disproportionately large degree of depression of phasic intercostal movement compared with that of the diaphragm. Halothane not only affected the amplitude of phasic movements of the rib cage, but also influenced the tone of the intercostal muscles, impairing rib cage stability and predisposing to paradoxical ventilation. These results are similar to those found by Tusiewicz, Bryan and Froese (1977); however, three of our patients did not show a disproportionate depression of phasic rib cage contribution, which was a consistent finding in all the subjects described by Tusiewicz. This lack of selective depression on rib cage could not be attributed to the much greater range of rib cage contribution in our subjects before anaesthesia. Patients with very small pre-anaesthetic contributions of the rib cage also showed profound depression of phasic intercostal activity with halothane. Goldman (1974) has pointed out that in the upright position a large part of the rib movement results from the mechanical coupling between the diaphragm and the rib cage, so that movement of the latter is simply a result of contraction of the diaphragm alone. As discussed by Tusiewicz, Bryan and Froese (1977), this coupling is less effective in the supine position, but there may be anatomical variations in the attachment of the diaphragm to the rib cage which may explain the unusual features seen in our three patients. These patients also had the largest reduction in tidal volumes and correspondingly large increases in frequency. Wade (1954) and Agostoni and Torri (1967) showed that increasing the frequency of breathing in conscious man produced either no change or a slight reduction in rib cage amplitude, although this may have little relevance to rib cage movement during anaesthesia.

The depressant effects of volatile anaesthetic agents on phasic movements of the chest wall are well recognized (Miller, 1925; Burstein, 1942) and have hitherto been believed to result from supraspinal effects of anaesthetics involving depression of medullary control. Tusiewicz, Bryan and Froese (1977) suggested that the major component of ventilatory depression resulted from preferential suppression of intercostals similar to that seen in the present study. The sensitivity of the "respiratory system" to the depressant effects of volatile anaesthetic agents varies widely.

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**FIG. 7.** The change in end-expiratory volume of rib cage and abdomen/diaphragm is the difference between the awake control value and the value obtained during steady-state halothane anaesthesia in the same posture.

<table>
<thead>
<tr>
<th>Time (min)</th>
<th>Trunk volume</th>
<th>Rib cage</th>
<th>Abdomen (litre)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>-1</td>
<td>-1</td>
<td>1</td>
</tr>
<tr>
<td>1</td>
<td>0</td>
<td>0</td>
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**FIG. 8.** After 20 min of anaesthesia this patient showed an increase in abdominal volume of 150 ml and an increase in rib cage volume of less than 50 ml while halothane anaesthesia was maintained at an alveolar concentration of 1.0%. Note the change in end-expiratory lung volume just before and during induction of anaesthesia. The change in rib cage position is less than that of the abdomen/diaphragm during these manoeuvres, indicating the differences in elastic properties of these two components of the chest wall.

volume of the abdomen of 150 ml while the rib cage showed a small increase of about 50 ml. The study terminated just before the commencement of surgery.
For example, 3 MAC of ether is required to give the same degree of carbon dioxide retention as 1 MAC of halothane (Larson et al., 1969). The magnitude of the selective effect of different anaesthetic agents on the rib cage has not been studied and it would be of considerable interest to see if depression of rib cage movement has a greater range of susceptibility to different anaesthetics as does depression of movement of the whole chest wall.

Changes in tone of the rib cage musculature were deduced from the changes observed with manual ventilation and from the observation of paradoxical ventilation where the rib cage contribution became negative in sign. During passive ventilation there was a much larger fractional movement of the rib cage than during spontaneous ventilation, although the abdomen/diaphragm movement was always greater than rib cage movement. This increase in rib cage movement, which was noted with thiopentone anaesthesia, showed a further increase following neuromuscular blockade with suxamethonium and this indicated a greater degree of background tone in the rib cage musculature than in the musculature of the abdomen/diaphragm. The relative movements of the rib cage and abdomen/diaphragm under these circumstances were indicative of the relative compliances of these two parts of the chest wall, although regional differences in lung recoil pressure reduced the evenness of distending pressures on the rib cage and abdomen/diaphragm. It is of interest that, whereas the lowest compliances were found in the two oldest subjects, there was no correlation between age and relative rib cage compliance. These results are similar to those described by Grimby, Hedenstierna and Löfström (1975), although these workers reported a larger mean rib cage contribution of 40% of tidal volume awake and, following anaesthesia with thiopentone, fentanyl and mechanical ventilation, there was a much larger rib cage contribution (72%). These workers used the magnetometer technique to record the changes in antero-posterior diameter of the rib cage and abdomen at two points and we have previously suggested (Faithfull, Jones and Jordan, 1979) that this technique may be inaccurate during anaesthesia because of the loss of stability of chest wall shape. The concept of the rib cage as a shell-like structure stabilized by the postural activity of its musculature has been developed by DaSilva and others (1977). They showed in the lightly anaesthetized, prone cat that transition from spontaneous respiration to mechanical ventilation following administration of gallamine was accompanied by a marked change in the shape of the rib cage and abdomen. Furthermore, the range of movement of the rib cage during mechanical ventilation was less than that during spontaneous ventilation, indicating a reduction in rib cage compliance in this posture. While our results, indicating a relative increase in rib cage compliance with neuromuscular blockade, are in agreement with those of Grimby, Hedenstierna and Löfström (1975), the difference in magnitude may be methodological in origin, since the reduction in rib cage compliance in the prone, paralysed cat (DaSilva et al., 1977) may be a result of the greater traction on the rib by the poorly supported abdominal contents. Underestimation of rib cage compliance may also have been produced by displacement of blood from the chest as airway pressure was increased. In the present study, this was observed in the rib cage trace when manual ventilation at a maintained positive airway pressure gave an initial increase followed by a decrease in rib cage volume as blood was displaced. This was followed by an increase in volume of the rib cage to a stable position as the circulation readjusted.

The onset of and recovery from anaesthesia was usually associated with transient and often large phase differences between rib cage and abdominal movement and these disappeared with either return of consciousness or the maintenance of "steady-state" anaesthesia. During the latter stage the consistent diminution in rib cage contribution in most patients was accompanied by a normal in-phase relationship between rib cage and abdomen. However, in two subjects, movements of the rib cage and abdomen were 180° out of phase during deep anaesthesia, producing a negative rib cage contribution. This phase reversal was accentuated by resistive loading in three other patients who had shown neither phase shift nor reduction in rib cage contribution while breathing against the same load before induction of anaesthesia. In the study of Tusiewicz, Bryan and Froese (1977), while none of the five young tracheally intubated patients showed any tendency to a negative rib cage contribution, all three of the non-tracheal intubated adult patients had paradoxical ventilation despite careful adjustment of the lower jaw.

This study has demonstrated that there are two effects of anaesthesia on the function of the rib cage: diminution of ventilatory movement and loss of postural control leading to instability of movement. The mechanism of the selective effect of volatile anaesthetic agents on these two functions of the rib cage is unknown. A helpful analysis of the effects of
anaesthesia on these functions of the intercostal muscles has been made by Duron (1973). He classified the chest wall musculature into two groups: (i) the internal and external intercostals, (ii) the diaphragm, interchondral and abdominal muscles. Tonal postural activity predominates in the former group and phasic ventilatory activity in the latter. Movements of these groups are produced by signals fed directly through the alpha motor neurone pathway and indirectly via the gamma loop system, and there is probably a close \( \alpha-\gamma \) linkage which is used in the regulation of muscle function (Sears, 1973). Nathan and Sears (1960) described a temporary paralysis of the diaphragm and intercostal muscles following section of the dorsal cervical and thoracic roots in man and suggested that this resulted from opening of the \( \gamma \) loop. Susceptibility of this \( \alpha-\gamma \) pathway to the effects of halothane has been reported by de Jong, Hershey and Wagman (1967) and it is of interest to note that Duron (1973) showed that muscles with the fewest muscle spindles, and thus least \( \alpha-\gamma \) linkage, are those with the most marked phasic activity. There are few spindles in the diaphragm and interchondral muscles, and their relative lack of inhibition by halothane may result from their independence from \( \gamma \)-loop control. We propose that halothane anaesthesia produces an attenuation of both phasic and tonic activity in the intercostal musculature by its predominant effect both on \( \gamma \) efferents and on more central control of \( \alpha-\gamma \) linkage. This leads to reduction in amplitude of phasic movements and loss of tone and also to instability in the shape of the rib cage with paradoxical movements between the rib cage and abdomen/diaphragm, particularly under conditions of increased respiratory loading (T. Sears, personal communication).

Following induction of anaesthesia in man, there is a reduction in functional residual capacity amounting to several hundred millilitre and this is of similar magnitude either with spontaneous respiration or after neuromuscular block (Hewlett et al., 1974a, b). This remarkable finding is so far unexplained. Part of the present study was designed to partition this change in FRC between the rib cage and the abdomen and it was expected that there would be a reduction in rib cage and abdominal volume. We were surprised to find only one subject who showed a reduction of both rib cage and abdominal volume, the majority of subjects showing an increase in abdominal volume (mean change +130 ml) and a small mean decrease in rib cage volume (−29 ml). This discrepancy between the widely reported reduction in internal volume and the net increase in external volume of the trunk found in this study, suggested that part of the mechanism for the reduction in FRC was an increase in the volume of the abdominal contents which displaces the abdominal wall outwards and the diaphragm upwards into the chest cavity. Such a distortion of the diaphragm into the chest cavity has been reported by Froese and Bryan (1974) from radiographic studies of the position of the diaphragm in awake and anaesthetized man. The only explanation for the increase in abdominal contents is an increase in either abdominal gas or blood volume. An increase in gas volume might occur from diffusion of nitrous oxide into intestinal air pockets and would be quite slow to reach a new equilibrium. A more likely explanation is an increase in vascular volume in the abdomen resulting from either a direct effect of halothane on vascular smooth muscle or impairment of autonomic regulation during anaesthesia. This leads to an augmentation of the normal central shift of blood volume from the legs that produces a gradual reduction of functional residual capacity (FRC) after assuming the supine position (Tenney, 1959). Tenney showed that in conscious subjects this reduction in FRC was accompanied by an increase in abdominal and rib cage circumference, but these changes were absent in four subjects with congenital or traumatic loss of the lower extremities. Lung volume is well controlled in conscious man by adjustments in the tone of the rib cage and abdominal wall musculature, the diaphragm position being controlled to a greater extent by fine control of the musculature of the rib cage and abdominal musculature than by fine adjustments within the diaphragm itself. Thus, following induction of anaesthesia, there is loss of postural reflexes in the anti-gravity muscles (which include those of the chest wall) and if abdominal pressure increases because of vasodilatation in the viscera, the diaphragm and abdominal wall are displaced in accordance with their respective compliances.

In conclusion, the present study has demonstrated a number of interacting factors that may contribute to the gas exchange abnormality that occurs during anaesthesia. Reduction in amplitude of rib cage movement results in a redistribution of \( \dot{V}/Q \) ratios within the lung. Loss of chest wall stability enhances this effect by increasing the likelihood of paradoxical ventilation, especially in the presence of obstruction. Increased respiratory frequency and reduction in tidal volume further exacerbates the \( \dot{V}/Q \) abnormality because of reduced time for diffusive mixing in
the airways (Cumming et al., 1967) and for the increased deadspace fraction of inspired gas (Cumming, Jones and Horsfield, 1969; West, 1971). Loss of postural reflexes impedes control of functional residual capacity, leading to a reduction in lung volume and narrowing or closure of dependent airways. These effects can only be ameliorated partially by combining positive end-expiratory pressure with a low inspiratory resistance breathing circuit. Further research into the effects of different anaesthetic agents on the chest wall is required to see if there is a wide difference in selective rib cage depression.

REFERENCES


MOUVEMENT DE LA CAGE THORACIQUE PENDANT UNE ANESTHÉSIE A L'HALOTHANE CHEZ L'HOMME

RESUME
Le mouvement de la paroi de la poitrine, divisé entre les contributions de la cage thoracique et de l'abdomen/diaphragme, a été mesuré à l'aide de quatre extensiomètres à mercure dans du caoutchouc et d'un ordinateur analytique. La contribution relative du volume de la cage thoracique et de l'abdomen/diaphragme par rapport à la quantité d'air expiré à chaque respiration a été mesurée sur 13 sujets avant et pendant l'anesthésie anesthésie au thiopentone et à l'halothane. Chez les sujets éveillés, le mouvement de l'abdomen/diaphragme a contribué à plus de 70% de la quantité d'air expiré à chaque respiration, la contribution de la cage thoracique à été moindre et s'est située entre 5 et 30%. La ventilation manuelle pendant l'induction de l'anesthésie a montré que la cage thoracique était moins compliquée que l'abdomen/diaphragme, mais après l'administration de suxaméthonium il y a eu une augmentation disproportionnée de la compliance de la cage thoracique. L'anesthésie par l'halothane a résulé, chez neuf sujets sur 12, en une diminution importante de la contribution fractionnelle de la cage thoracique. Chez deux de ces sujets, la respiration a été paradoxale, le mouvement de la cage thoracique et celui de l'abdomen/diaphragme étant déphasés de 180°. Cet effet a facilement été reproduit sur trois autres sujets en insérant une résistance (1 kPa litre⁻¹ s) dans le circuit de l'anesthésie. Cette technique a aussi donné des informations sur les variations du volume du tronc causées par l'anesthésie. Onze sujets ont accusé une augmentation du volume abdominal expiratoire final (augmentation moyenne de 120 ml) pendant l'anesthésie par l'halothane,
alors qu’il y avait une réduction moyenne de 29 ml dans le volume expiratoire final de la cage thoracique. Nous en avons conclu que l’halothane déprime à la fois l’activité tonique et l’activité de phase du réflexe postural, ce qui a surtout affecté la musculature de la cage thoracique. Ceci a réduit l’amplitude du mouvement de phase de la cage thoracique, a affecté la stabilité de la cage thoracique et l’a prédéposé à la ventilation paradoxale. Les résultats laissent aussi penser que la réduction du volume pulmonaire pendant l’anesthésie peut provenir de la perte de contrôle postural de la paroi de la poitrine et un déplacement central du volume de sang.

MOVIMIENTO DEL COSTILLAJE DURANTE ANESTESIA DE HALOTANO EN EL HOMBRE

SUMARIO
Se midió el movimiento de las paredes del pecho, distinguiéndose las contribuciones del costillaje y abdomen/diafragma, empleándose para tal fin cuatro deformímetros de "mercurio en caucho" y un computador analógico. La contribución de volumen relativa del costillaje y del abdomen/diafragma al volumen de respiración corriente fue medida en 13 sujetos antes y durante anestesia con tiopentona y halotano. En los sujetos despiertos, el movimiento del abdomen/diafragma contribuyó más del 70% del volumen de la respiración y la contribución menor del costillaje varió entre 5 y 30%. La ventilación manual durante la inducción de anestesia indicó que el costillaje era menos elástico que el abdomen/diafragma, pero cuando se administró suxametonio, se produjo un aumento desproporcionado en la elasticidad del costillaje. En nueve de 12 sujetos, la anestesia de halotano causó una gran disminución de la contribución fraccional del costillaje. En dos de estos sujetos se produjo una respiración paradójica, estando el movimiento del costillaje y del abdomen/diafragma desfasado en 180°. Este efecto fue producido con facilidad en tres sujetos más mediante la inserción de una resistencia (1 kPa litro⁻¹) en el circuito anestésico. La técnica también aportó información acerca de los cambios en el volumen del tronco inducidos por anestesia. Once sujetos acusaron un aumento en el volumen abdominal al final de la expiración (aumento medio de 120 ml) durante la anestesia de halotano, mientras que se produjo una reducción media en el volumen del costillaje al final de la expiración de 29 ml. Concluimos que el halotano deprimió la actividad refleja, tanto fásica como tónica, que afectó predominantemente a la musculatura del costillaje. Esto redujo la amplitud del movimiento fásico del costillaje, prejudició la estabilidad del costillaje y predispuso la ventilación paradójica. Los resultados también sugirieron que la reducción en el volumen de los pulmones durante la anestesia puede ser el resultado de una pérdida de control de la postura en la pared del pecho y un desplazamiento central del volumen sanguíneo.