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# Human Chest Wall Function while Awake and during Halothane Anesthesia

## I. Quiet Breathing

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***Background:*** **Data concerning chest wall configuration and the activities of the major respiratory muscles that determine this configuration during anesthesia in humans are limited. The aim of this study was to determine the effects of halothane anesthesia on respiratory muscle activity and chest wall shape and motion during spontaneous breathing.**

***Methods:*** **Six human subjects were studied while awake and during 1 MAC halothane anesthesia. Respiratory muscle activity was measured using fine-wire electromyography electrodes. Chest wall configuration was determined using images of the thorax obtained by three-dimensional fast computed tomography. Tidal changes in gas volume were measured by integrating respiratory gas flow, and the functional residual capacity was measured by a nitrogen dilution technique.**

***Results:*** **While awake, ribcage expansion was responsible for 25 ± 4% (mean ± SE) of the total change in thoracic volume ($\Delta V_{th}$) during inspiration. Phasic inspiratory activity was regularly present in the diaphragm and parasternal intercostal muscles. Halothane anesthesia (1 MAC) abolished activity in the parasternal intercostal muscles and increased phasic expiratory activity in the abdominal muscles and lateral ribcage muscles. However, halothane did not significantly change the ribcage contribution to $\Delta V_{th}$ (18 ± 4%). Intrathoracic blood volume, measured by comparing changes in total thoracic volume and gas volume, increased significantly during inspiration both while awake and while anesthetized (by approximately 20% of $\Delta V_{th}$, $P < 0.05$). Halothane anesthesia significantly reduced the functional residual capacity (by 258 ± 78 ml), primarily *via* an inward motion of the end-expiratory position of the ribcage. Although the diaphragm consistently changed shape, with a cephalad displacement of posterior regions and a caudad displacement of anterior regions, the diaphragm did not consistently contribute to the reduction in the functional residual capacity. Halothane anesthesia consistently increased the curvature of the thoracic spine measured in the sagittal plane.**

***Conclusions:*** **The authors conclude that (1) ribcage expansion is relatively well preserved during halothane anesthesia despite the loss of parasternal intercostal muscle activity; (2) an inward displacement of the ribcage accounts for most of the decrease in functional residual capacity caused by halothane anesthesia, accompanied by changes in diaphragm shape that may be related to motion of its insertions on the thoracoabdominal wall; and (3) changes in intrathoracic blood volume constitute a significant fraction of $\Delta V_{th}$ during tidal breathing. (Key words: Anesthetics, volatile: halothane. Lung: breathing pattern; diaphragm; functional residual capacity; intrathoracic blood volume; ribcage. Measurement technique: dynamic spatial reconstructor; electromyography; fast computed tomography; respiratory impedance plethysmography. Muscle: diaphragm; external oblique; parasternal intercostal; respiratory; transversus abdominis.)**

IT has long been appreciated that anesthetic drugs affect chest wall function.[1] Observations of the pattern of breathing were employed for many years as a clinically useful guide to the proper administration of anesthesia. Intercostal muscle paralysis, defined by delayed or diminished inspiratory expansion of the ribcage, delineated the third plane of surgical ether anesthesia.[2] A similar pattern of breathing has been observed during halothane anesthesia.[3,4] However, there are major limitations in our current understanding of chest wall function during anesthesia.

Exploration of mechanisms of anesthetic effects on the chest wall requires knowledge of the activity of the respiratory muscles. Many studies use chest wall motion to infer anesthetic actions on the activities of individual respiratory muscles. Because the motion of chest wall structures depends on the complex, coordinated activity of many respiratory muscles, this approach has significant limitations. For example, impaired inspiratory ribcage expansion during halothane anesthesia has been attributed to an attenuation of nor-

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**Table 1. Patient Characteristics**

| Patient No. | Age (yr) | Height (cm) | Weight (kg) | $FEV_1/FVC$ (%) | VC (1) | FRC (1) |
|---|---|---|---|---|---|---|
| 1 | 32 | 195 | 83 | 75 | 6.3 | 3.80 |
| 2 | 38 | 173 | 81 | 81 | 4.1 | 1.93 |
| 3 | 34 | 179 | 84 | 82 | 4.3 | 1.70 |
| 4 | 39 | 183 | 81 | 79 | 5.7 | 2.50 |
| 5 | 28 | 175 | 68 | 85 | 4.9 | 2.92 |
| 6 | 40 | 176 | 68 | 76 | 4.4 | 2.37 |

$FEV_1$ = forced expiratory volume in 1 s; FVC = forced vital capacity; VC = vital capacity; FRC = functional residual capacity.

All pulmonary function tests were performed with subjects in the supine position.

mal phasic inspiratory activation of the parasternal intercostal muscles.[3,4] However, halothane anesthesia also produces phasic expiratory activity in abdominal muscles, which may affect chest wall motion.[5–7] Data concerning respiratory muscle activation during anesthesia in humans are scarce,[3,5,6,8] and no studies have examined the relationship between chest wall motion and the activity of the major respiratory muscles that determine this motion.

Furthermore, available techniques to measure the motion of the chest wall have significant limitations. Current methods use changes in the external dimensions of the ribcage and abdomen to estimate the volume displaced by the ribcage and diaphragm during inspiration.[9] Although the volume displaced by the abdomen often is considered to reflect diaphragm displacement, these two quantities are not identical.[10] Thus, basic information, such as the relative volumes displaced by the ribcage and diaphragm during tidal breathing, is unknown, either during awake or anesthetized states. Assessment of the end-expiratory position of the diaphragm, thought to contribute to reductions in the functional residual capacity (FRC) caused by anesthesia,[11] remains problematic. Measurements of external dimensions also do not provide information regarding possible changes in intrathoracic blood volume during breathing[12,13] that may affect lung gas volumes during anesthesia.[14]

The overall objective of this study was to determine the effects of halothane anesthesia on both respiratory muscle activity and chest wall motion in human subjects breathing spontaneously, combining measurements of the respiratory muscle electromyograms (EMGs) with imaging of the thorax using three-dimensional fast computed tomography. Results of these experiments are presented in two papers. Issues addressed by the current report include (1) the effects of halothane anesthesia on chest wall motion as determined directly from thoracic images during quiet breathing, (2) the relationship between chest wall motion and respiratory muscle activation during halothane anesthesia, and (3) mechanisms reducing the FRC during halothane anesthesia. Results obtained during carbon dioxide rebreathing, providing a more detailed analysis of halothane's effects on neural drive to individual respiratory muscles, are presented in an accompanying communication.[15]

## Materials and Methods

This study was approved by the Institutional Review Board. Six healthy males were studied after informed consent. Each subject had a complete physical examination, including pulmonary function testing (table 1), and was brought to the laboratory the day before the actual experiment for familiarization with experimental procedures.

### *Instrumentation*

All studies were performed with subjects supine. Respiratory impedance plethysmography (RIP) belts (Respitrace) were placed around the upper ribcage and midabdomen. An intravenous catheter was inserted, and the radial artery was cannulated to obtain samples for blood gas analysis (IL 1302) and to monitor blood pressure.

Bipolar EMG electrodes were inserted into several respiratory muscles. The electrodes were fashioned by removing 1 mm of insulation from the end of 0.005-cm Teflon-insulated wires. Two wires were passed through an insulated 30-G needle and then bent 1 mm from the end to form hooks. The electrodes were inserted under ultrasonic guidance[16] during monitoring of electrical activity. After placement in the desired location, the needle was removed, leaving the wires in the desired muscle. Electrodes were placed in the transversus abdominis and external oblique muscles at the anterior axillary line approximately 4 cm inferior to the costal margin, the parasternal intercostal muscle at the third right interspace, approximately 3 cm lateral to the midline, and the diaphragm. To insert the diaphragm electrode, ultrasound was first used to define the caudad extent of the area of apposition at full inspiration. The needle was inserted at the next most inferior interspace in the left midaxillary line and ad-

vanced until phasic inspiratory electrical activity was detected.[17] This procedure minimized the possibility of pneumothorax, placing the electrode through the intercostal muscles into the diaphragm close to its insertion on the ribcage. After removal of the introducing needles, the subjects noted no sensation from the electrodes, other than some mild discomfort during full inspiration at the site of the diaphragmatic electrode in some subjects. EMG signals were amplified (Grass P511), bandpass -filtered between 30 and 3,000 Hz, and recorded both on a strip chart recorder (Astromed MT9500) and on digital audio tape (TEAC RT100) for later processing.

The RIP bands were calibrated using the method of Mankikian *et al.*[18] In this method, changes in dimensions of the ribcage and abdomen measured with the RIP bands are related to the volumes displaced by the ribcage and diaphragm-abdomen (referred to hereafter as abdominal) compartments during tidal breathing. These relationships are expressed as volume-motion coefficients of the ribcage and abdomen. These coefficients are calculated from data obtained by asking the subjects to alternate predominately abdominal and thoracic breathing. Calibrations were performed at the beginning and end of each experimental condition (awake and anesthetized), and average values were calculated for each condition.

Each subject breathed quietly through a mouthpiece with a nose clip until the breathing pattern was stable (see Appendix for discussion of the effects of this equipment on breathing). A gas mixture of 30% $O_2$, balance nitrogen, was inspired *via* an open circuit throughout the experiment. Inspiratory and expiratory gas flows were measured using a pneumotachograph (Fleisch 3) connected to a differential pressure transducer (Validyne MP-45). Gas flows were integrated to obtain changes in lung volume and corrected to body temperature, standard pressure conditions. The electrocardiogram, blood pressure, and arterial oxygen saturation were monitored throughout the study.

*Procedure*

Each subject was placed in the dynamic spatial reconstructor (DSR), a high-speed x-ray scanner that used the computed tomography principle to provide three-dimensional images of the thorax. This technique has been described in detail elsewhere.[12–14,19,20] The DSR has sufficient temporal resolution to image thoracic structures during quiet breathing and sufficient volume resolution to determine a known volume to within 2%.

DSR images were obtained while the subject breathed quietly through a mouthpiece and nose clip. Scans of 300 ms duration were triggered manually at end-expiration and end-inspiration. Scans at the same point in the breathing cycle were recorded during three consecutive breaths and gated together during later analysis to provide end-expiratory and end-inspiratory images.[14] Because the cephalocaudal height of the imaging field was not sufficient to include the entire thorax, these initial scans included only the superior half of the thorax. The subject next was shifted cephalad, and a similar sequence of scans was obtained to image the inferior portion of the thorax.[14] RIP measurements, tidal volume, and EMGs were recorded simultaneously to ensure stability of the breathing pattern. During later analysis, the superior and inferior images were joined to produce end-inspiratory and end-expiratory images of the entire thorax.

Immediately after scanning, the FRC was measured in duplicate using a nitrogen dilution technique.[21,22] Each subject performed six vital capacity maneuvers into a 4-l bag initially filled with 100% $O_2$ after the bag was connected to the mouthpiece at end-expiration. Nitrogen concentrations in the bag were determined before and after this maneuver by a mass spectrometer (Perkin-Elmer MGA 1100). Preliminary measurements in three subjects showed a coefficient of variation ranging from 3–7% over five consecutive FRC measurements and agreement to within 8% with a standard 7-min nitrogen washout technique.[23] The FRC values measured with this method while the subjects were awake during the study did not differ significantly from values measured by prestudy pulmonary function testing, which used a helium dilution technique (2.50 ± 0.35 (mean ± SE) and 2.54 ± 0.31 l, respectively).

While breathing quietly with a mouthpiece and nose clip, the subject was sedated with halothane at 0.2 MAC end-tidal concentration (Nellcor N-2500) and the pattern of breathing recorded after approximately 10 min. The mouthpiece was removed, and a face mask was applied. An inhalation induction with halothane was performed, using an oral airway to maintain airway patency when necessary. The trachea was intubated with a 9.0 mm ID endotracheal tube during deep halothane anesthesia, then the inspired halothane concentration was adjusted to maintain approximately 1 MAC end-tidal concentration. Esophageal and gastric balloons were placed in the midesophagus and stomach, respectively, and these measurements were validated by standard techniques.[24] After the pattern of breathing

had stabilized, the subject was placed in the DSR and the thorax was scanned as before. Measurements of the FRC were repeated, using a 4-l syringe to passively inflate the lungs.

After these measurements, the endotracheal tube was removed, inspired halothane was discontinued, and the subject was allowed to recover from anesthesia.

*Data Analysis*

Details of image processing to define chest wall boundaries and validation of the DSR in measuring chest wall motion have been described previously.[12–14,19,20] To summarize, each scan produced a three-dimensional volume image of the thorax composed of cubic volume elements (voxels) with edge lengths of 1.3 mm (fig. 1A). Images were processed to define each voxel in the image as being in the thoracic cavity, the abdominal cavity, or the background. Thoracic volume ($V_{th}$) was determined by counting the number of voxels in the thoracic cavity above the diaphragm. Changes in thoracic liquid volume during inspiration ($\Delta V_{liq}$), presumably representing changes in thoracic blood volume,[14] were calculated as the difference between changes in $V_{th}$ from the beginning to end of inspiration ($\Delta V_{th}$) and $V_T$ measured by the integration of gas flow ($\Delta V_{liq} = \Delta V_{th} - V_T$). Changes in $V_{th}$ between any two scans were partitioned into volumes displaced by the motion of the diaphragmatic and ribcage surfaces as previously described.[12]

Changes in the volume of the heart and major vessels were estimated directly from these images.[21] The heart and other mediastinal structures were isolated in each image using a combination of computer thresholding and operator interaction (fig. 1B). The pulmonary vessels were truncated at approximately the same location in all scans from each subject. The total volume of these mediastinal structures ($V_{ms}$) was measured by counting the number of voxels within these structures and multiplying by the volume of one voxel.

Statistical comparisons were made by paired *t* tests, and a $P < 0.05$ was taken as significant.

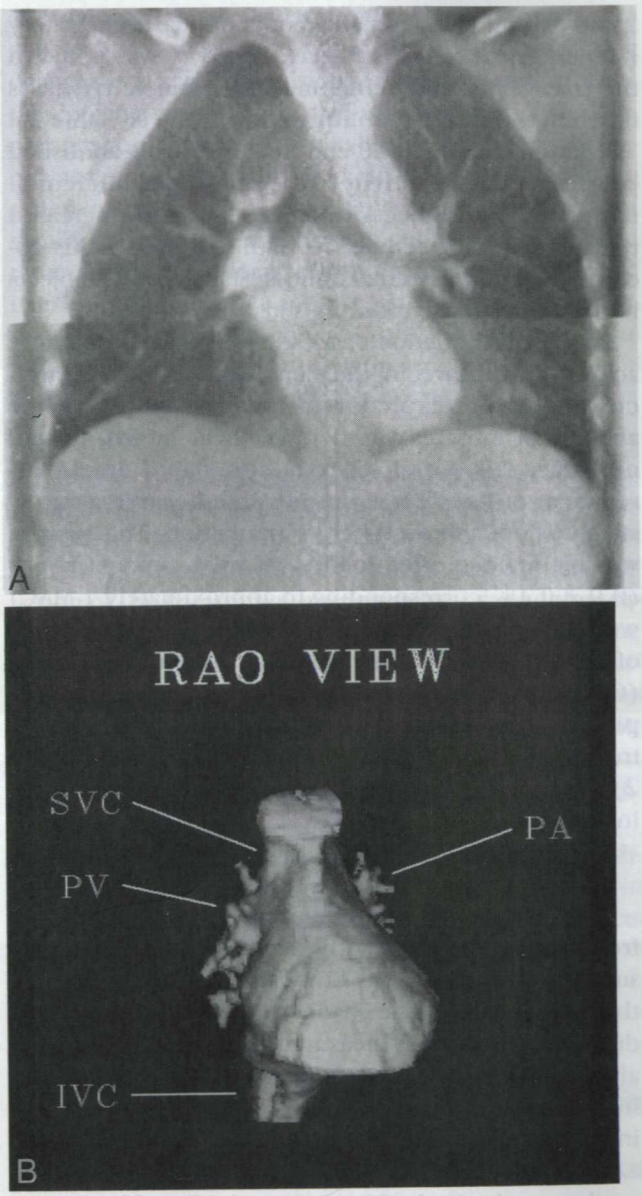



**Fig. 1. Representative dynamic spatial reconstructor images. (*A*) Coronal section of the thorax. (*B*) Image of the heart and mediastinal structures, right anterior oblique (RAO) view. IVC = inferior vena cava; PA = pulmonary artery; PV = pulmonary vein; SVC = superior vena cava.**

## Results

The duration of anesthesia averaged 171 ± 11 min; no untoward reactions were observed.

*EMG Activity*

Consistent phasic inspiratory activation was observed in the diaphragm and parasternal intercostal muscles during quiet breathing while awake (fig. 2 and table 2). Both muscles exhibited consistent postinspiratory inspiratory activity, defined as EMG activity that persisted into the early part of expiration. Phasic expiratory activity was detected by the diaphragm electrode in four subjects (figs. 2 and 3). No consistent tonic activity was observed in the parasternal intercostal muscle (fig.

2). In two subjects, a small amount of phasic expiratory (subject 5) or tonic (subject 6) activity was noted in the transversus abdominis muscle; tonic activity was noted in the external oblique of one subject (table 2).

Sedation with 0.2 MAC end-tidal halothane abolished phasic inspiratory activity in the parasternal intercostal muscle of two subjects (table 2). Sedation increased the incidence of activity in both the transversus abdominus and the external oblique (table 2). All subjects remained responsive during sedation.

During the induction of anesthesia, phasic inspiratory activity disappeared in the parasternal intercostal muscle, and phasic expiratory activity developed in the abdominal muscles (fig. 2). Tracheal intubation did not affect this overall pattern, although a precise assessment of its effect is not possible because a steady state was not achieved before intubation. This pattern was maintained after stable end-tidal concentrations ($0.9 \pm 0.1\%$, corresponding to approximately 1 MAC) were achieved (figs. 2 and 3 and table 2). The incidence of activity was greater in the transversus abdominus (6/6) than in the external oblique (1/5). Phasic expiratory activity was detected by the diaphragm electrode in all subjects during anesthesia (fig. 3 and table 2), probably reflecting activity in the adjacent internal intercostal muscles.

*Chest Wall Motion*

Halothane anesthesia significantly increased breathing frequency and significantly decreased both $V_T$, measured by integrating gas flow, and $\Delta V_{th}$, measured by the DSR (table 3). The change in intrathoracic volume during inspiration, as measured by the DSR ($\Delta V_{th}$), was always greater than the tidal volume ($V_T$) measured by integrating gas flow, indicating that $V_{liq}$ increased during inspiration (table 3). This increase in $V_{liq}$ comprised a substantial fraction of $\Delta V_{th}$ both while awake and while anesthetized ($20 \pm 4\%$ and $18 \pm 3\%$, respectively) and corresponded closely to the change in $V_{ms}$ during inspiration, measured directly from DSR images of the mediastinal structures (table 3).

While awake, ribcage expansion was responsible for $25 \pm 4\%$ of $\Delta V_{th}$, as measured by the DSR, with the balance of thoracic expansion produced by caudad displacement of the diaphragm (table 3). Halothane anesthesia reduced the absolute volumes displaced by the ribcage and diaphragm but did not significantly change the relative contribution of the ribcage to $\Delta V_{th}$ ($18 \pm 2\%$). A similar pattern was observed for the RIP measurements of chest wall motion, as halothane had no

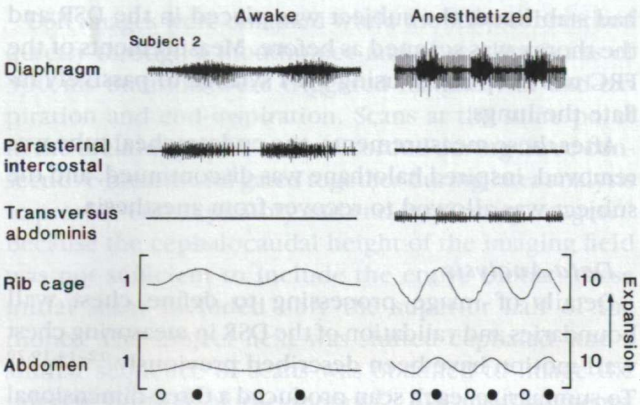



**Fig. 2. Representative record from one subject while awake and during halothane anesthesia. Upper three tracings are electromyograms obtained using fine-wire electrodes; lower tracings represent ribcage and abdominal dimensions measured by respiratory impedance plethysmography, expressed in arbitrary units. Open and closed circles denote the beginning and end of inspiration, respectively. Note that, although the amplitude of both ribcage and abdominal excursions is diminished during halothane anesthesia, the relationship between their amplitudes is preserved.**

significant effect on the ribcage contribution to $V_T$ (table 3). During both conditions, RIP measurements significantly overestimated the ribcage contribution and underestimated the diaphragm contribution to $V_T$ (table 3). Halothane anesthesia did not significantly affect RIP calibration (data not shown).

*Functional Residual Capacity*

Halothane anesthesia significantly decreased both the FRC, measured by nitrogen dilution, and $V_{th}$ at end-expiration, measured with the DSR (table 4). In all subjects, the cross-sectional area of the ribcage at end-expiration decreased, contributing to the fall in $V_{th}$ (table 4). The pattern of this motion was variable among subjects (fig. 4). In all subjects, the most dependent regions of the diaphragm moved cephalad (fig. 5). However, in five subjects, the most nondependent regions of the diaphragm moved caudad. As a result, there was a net cephalad displacement of the diaphragm in only two subjects; in the other four subjects, there was a net caudad diaphragm displacement. Thus, changes in end-expiratory diaphragm position did not consistently contribute to the reduction in $V_{th}$ caused by halothane (table 4).

These changes in ribcage and diaphragm shape were accompanied by changes in the position of other thoracic structures. In five subjects, the end-expiratory

**Table 2. Incidence of Electromyogram Activity**

| Patient No. | Parasternal Intercostal | Diaphragm | Transverse Abdominus | External Oblique |
|---|---|---|---|---|
| Awake | | | | |
| 1 | P, I | P, I | — | — |
| 2 | P, I | P, I | — | — |
| 3 | P, I | P, I, and E | — | — |
| 4 | P, I | P, I, and E | — | NA |
| 5 | P, I | P, I, and E | P, E | — |
| 6 | P, I | P, I, and E | T | T |
| Sedated (0.2 MAC) | | | | |
| 1 | P, I | P, I | P, E | — |
| 2 | P, I | P, I | T | T |
| 3 | — | P, I, and E | T | T |
| 4 | P, I | P, I, and E | — | NA |
| 5 | P, I | P, I, and E | P, E | — |
| 6 | — | P, I, and E | P, E | — |
| Anesthetized (1 MAC) | | | | |
| 1 | — | P, I, and E | P, E | P, E |
| 2 | — | P, I, and E | P, E | — |
| 3 | — | P, I, and E | P, E | — |
| 4 | — | P, I, and E | P, E | NA |
| 5 | — | P, I, and E | P, E | — |
| 6 | — | P, I, and E | P, E | — |

— = no activity; P = phasic activity; T = tonic activity; NA = measurements not available. For physically active muscles: I = activity predominantly during inspiratory gas flow; E = activity predominantly during expiratory gas flow.

Nomenclature shows the type of activity (none, phasic, or tonic) detected by each electrode and when phasic activity predominates, if present (inspiration or expiration).

position of the sternal angle moved caudad with the induction of halothane anesthesia (fig. 6). The one subject whose sternal angle moved cephalad (subject 6) was the same subject in whom the nondependent region of the diaphragm also moved cephalad. In all six subjects, the curvature of the thoracic spine, defined by the anterior border of the vertebral bodies, increased with the induction of anesthesia (fig. 7).

Neither $V_{liq}$ nor $V_{ms}$ at end-expiration changed significantly with the induction of anesthesia, indicating that changes in intrathoracic liquid did not consistently contribute to changes in the FRC caused by halothane (table 4).

## Discussion

We report the first direct measurements of the volumes displaced by the ribcage and diaphragm in human subjects during spontaneous breathing while awake or anesthetized. These measurements reveal that (1) a conventional measure of chest wall motion utilizing changes in surface dimensions (*i.e.*, RIP) overestimates the ribcage contribution and underestimates the diaphragm contribution to tidal volume; (2) there are significant increases in intrathoracic blood volume during quiet inspiration; (3) halothane anesthesia does not significantly change the ribcage contribution to inspiratory changes in thoracic volume, despite abolition of inspiratory intercostal activity; and 4) the diaphragm does not consistently contribute to reductions in the FRC caused by halothane anesthesia.

### *EMG Activity*

Although the diaphragm is the primary muscle of respiration in humans, actions of other muscles are required for normal ribcage expansion during quiet breathing. These include the phasic inspiratory activation of the parasternal intercostal[17,25–27] and the scalene muscles,[28,29] phasic expiratory activation of the internal intercostal muscles in the lower lateral interspaces,[17] and under some conditions, the transversus thoracis muscle.[30] Although abdominal muscles exhibit tonic activity in upright positions, they are usually not active in the supine position during quiet breathing.[16,31]

Our findings are consistent with these previous observations. The phasic expiratory activity observed in the diaphragmatic electrode of four awake subjects

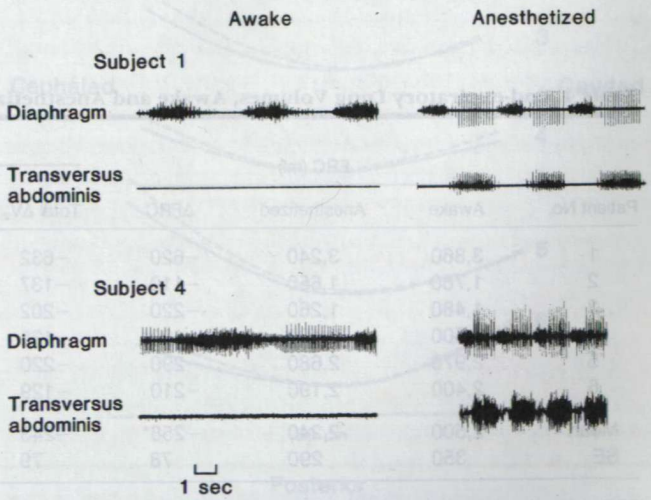



**Fig. 3. Representative electromyogram tracings from two subjects while awake and during halothane anesthesia, showing the phasic expiratory activity detected by the transversus abdominis and diaphragm electrodes.**

**Table 3. Volume Displacements**

| Patient No. | f ($min^{-1}$) | $V_T$ (ml) | $\Delta V_{th}$ (ml) | $\Delta V_{liq}$ (ml) | $\Delta V_{ms}$ (ml) | DSR: Volume Displaced by Rib Cage ml | DSR: Volume Displaced by Rib Cage % $\Delta V_{th}$ | DSR: Volume Displaced by Diaphragm ml | DSR: Volume Displaced by Diaphragm % $\Delta V_{th}$ | RIP: Volume Displaced by Rib Cage ml | RIP: Volume Displaced by Rib Cage % $V_T$ | RIP: Volume Displaced by Diaphragm—Abdomen ml | RIP: Volume Displaced by Diaphragm—Abdomen % $V_T$ |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Awake | | | | | | | | | | | | | |
| 1 | 11.4 | 670 | 746 | 76 | 58 | 218 | 29 | 528 | 71 | 169 | 23 | 554 | 77 |
| 2 | 11.5 | 482 | 601 | 119 | 138 | 148 | 25 | 453 | 75 | 188 | 41 | 272 | 59 |
| 3 | 13.0 | 507 | 694 | 187 | 166 | 130 | 19 | 564 | 81 | 208 | 46 | 247 | 54 |
| 4 | 8.1 | 801 | 878 | 77 | 105 | 149 | 17 | 729 | 83 | 236 | 33 | 475 | 67 |
| 5 | 7.2 | 673 | 838 | 165 | 118 | 174 | 21 | 663 | 79 | 170 | 25 | 507 | 75 |
| 6 | 14.6 | 482 | 707 | 225 | 185 | 292 | 41 | 415 | 59 | 304 | 59 | 214 | 41 |
| Mean | 11.0 | 603 | 744 | 141 | 128 | 185 | 25 | 559 | 75 | 213 | 38† | 378† | 62† |
| SE | 1.2 | 54 | 41 | 25 | 19 | 25 | 4 | 49 | 4 | 21 | 5 | 61 | 5 |
| Anesthetized | | | | | | | | | | | | | |
| 1 | 18.1 | 403 | 432 | 29 | 81 | 48 | 11 | 384 | 89 | 110 | 30 | 262 | 70 |
| 2 | 29.4 | 258 | 385 | 127 | 103 | 84 | 22 | 301 | 78 | 75 | 27 | 204 | 73 |
| 3 | 33.1 | 223 | 308 | 85 | 76 | 41 | 13 | 267 | 87 | 91 | 36 | 163 | 64 |
| 4 | 36.6 | 230 | 251 | 21 | 51 | 51 | 20 | 200 | 80 | 75 | 31 | 166 | 69 |
| 5 | 30.6 | 289 | 291 | 2 | 31 | 48 | 16 | 243 | 84 | 93 | 32 | 202 | 68 |
| 6 | 24.0 | 266 | 394 | 128 | 38 | 109 | 28 | 285 | 72 | 64 | 23 | 216 | 77 |
| Mean | 28.6* | 278* | 344* | 65* | 63* | 63* | 18 | 280* | 82 | 85* | 30† | 202*† | 70† |
| SE | 3.0 | 27 | 29 | 23 | 11 | 11 | 2 | 25 | 2 | 7 | 2 | 15 | 2 |

f = breathing frequency; $V_T$ = tidal volume, measured by integrating gas flows; $\Delta V_{th}$ = change in thoracic volume during inspiration, measured from DSR images; $\Delta V_{liq}$ = change in thoracic liquid volume during inspiration; $\Delta V_{ms}$ = change in volume of heart and mediastinal structures during inspiration; DSR = dynamic spatial reconstructor; RIP = respiratory impedance plethysmography.

* Significant difference from awake ($P < 0.05$, paired $t$ test).

† Significant difference between DSR and RIP estimates ($P < 0.05$, paired $t$ test).

**Table 4. End-expiratory Lung Volumes, Awake and Anesthetized**

| Patient No. | FRC (ml) Awake | FRC (ml) Anesthetized | FRC (ml) ΔFRC | $\Delta V_{th}$ (ml) Total $\Delta V_{th}$ | $\Delta V_{th}$ (ml) Volume Displaced by Rib Cage | $\Delta V_{th}$ (ml) Volume Displaced by Diaphragm | $\Delta V_{liq}$ (ml) | $\Delta V_{ms}$ (ml) |
|---|---|---|---|---|---|---|---|---|
| 1 | 3,860 | 3,240 | −620 | −632 | −270 | −362 | −12 | −83 |
| 2 | 1,760 | 1,650 | −110 | −137 | −211 | 74 | −27 | −120 |
| 3 | 1,480 | 1,260 | −220 | −202 | −244 | 42 | 18 | −11 |
| 4 | 2,500 | 2,400 | −100 | −135 | −171 | 36 | −35 | −102 |
| 5 | 2,970 | 2,680 | −290 | −220 | −289 | 69 | 70 | −21 |
| 6 | 2,400 | 2,190 | −210 | −129 | −102 | −28 | 81 | 28 |
| Mean | 2,500 | 2,240 | −258* | −243* | −214* | −28 | 16 | −52 |
| SE | 350 | 290 | 78 | 79 | 28 | 68 | 20 | 24 |

FRC = functional residual capacity, measured by $N_2$ dilution; $\Delta V_{th}$ = change in thoracic volume, measured using DSR images; $\Delta V_{liq}$ = change in thoracic liquid volume; $\Delta V_{ms}$ = change in the volume of heart and mediastinal structures.

Values for changes are calculated as the differences between anesthetized and awake values.

* Significant difference from 0 (paired $t$ tests).

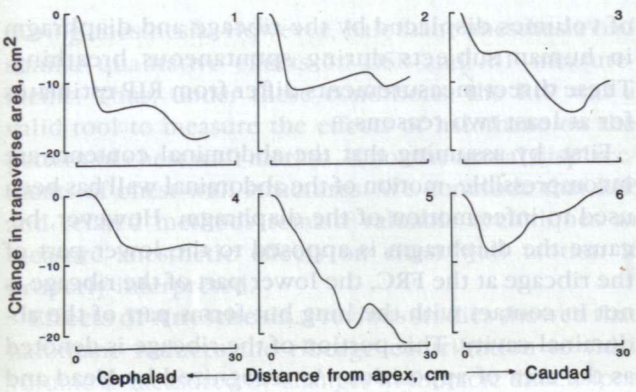



**Fig. 4. Change in end-expiratory transverse cross-sectional area of the thoracoabdominal cavity with the induction of halothane anesthesia in six subjects. Note the considerable intersubject variability in the pattern of change.**

represents either phasic expiratory diaphragmatic activity, which seems unlikely, or activity in adjacent muscles. Possibilities include the adjacent internal intercostal muscle, or the more superficial serratus anterior and external abdominal oblique muscles. Because phasic respiratory activity has not been documented in the latter two muscles in previous studies,[16,17] it seems likely that the source of phasic expiratory activity in the diaphragmatic electrode is the internal intercostal muscle, which exhibits phasic expiratory activity in supine subjects breathing quietly.[17] Alternatively, the diaphragmatic electrode could have been located in the internal intercostal muscle and detected phasic inspiratory activity from the adjacent diaphragm. It is unlikely that the external intercostal muscles contributed to inspiratory EMG activity, as these muscles are not active at this location in humans during quiet breathing.[27]

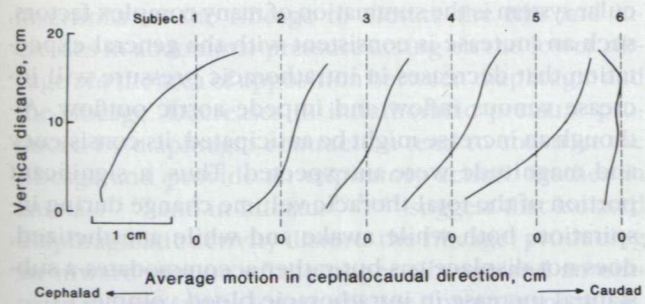
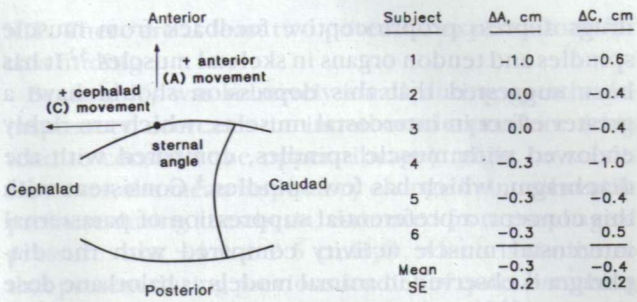



**Fig. 5. Change in the average position of the diaphragm with the induction of halothane anesthesia in six subjects. Note that the posterior regions consistently moved cephalad and the anterior regions moved caudad in five subjects.**




| Subject | ΔA, cm | ΔC, cm |
|---|---|---|
| 1 | -1.0 | -0.6 |
| 2 | 0.0 | -0.4 |
| 3 | 0.0 | -0.4 |
| 4 | -0.3 | -1.0 |
| 5 | -0.3 | -0.4 |
| 6 | -0.3 | 0.5 |
| Mean | -0.3 | -0.4 |
| SE | 0.2 | 0.2 |

**Fig. 6. Changes in the end-expiratory position of the sternum with the induction of anesthesia, as determined from dynamic spatial reconstructor images. The sternal angle moved caudad with the induction of anesthesia in every subject except subject 6.**

Consistent with previous observations in three subjects,[3] halothane abolished activity in the parasternal intercostal muscle. This effect occurred at low halothane doses in some subjects, suggesting that it is not related simply to a loss of consciousness. Anesthetic

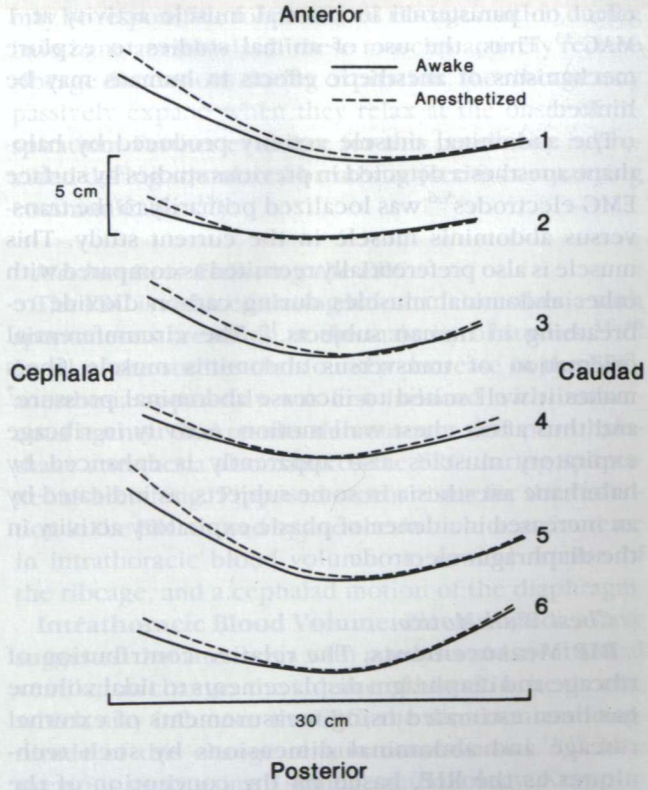



**Fig. 7. Curvature of the anterior border of the vertebral bodies of the thoracic spine, measured in a midsagittal section, while awake and while anesthetized. Anesthesia consistently increased spinal curvature.**

drugs depress proprioceptive feedback from muscle spindles and tendon organs in skeletal muscles.[32] It has been suggested that this depression should have a greater effect in intercostal muscles, which are richly endowed with muscle spindles, compared with the diaphragm, which has few spindles.[3] Consistent with this concept, a preferential suppression of parasternal intercostal muscle activity compared with the diaphragm is observed in animal models as halothane dose increases.[33,34] However, this explanation alone cannot suffice, because the abdominal muscles, which are recruited by halothane anesthesia, also have many spindles. In vagotomized dogs, expiratory bulbospinal neurons, which provide input to expiratory motoneurons, are more resistant to isoflurane-induced depression compared with the phrenic nerve, suggesting a differential effect of anesthetics on respiratory premotor neurons.[35] Unfortunately, interpretation of this and other studies is complicated by profound species differences between anesthetic effects in dogs and humans. In dogs, halothane abolishes phasic expiratory activity in abdominal and ribcage muscles but has little effect on parasternal intercostal muscle activity at 1 MAC.[33] Thus, the use of animal studies to explore mechanisms of anesthetic effects in humans may be limited.

The abdominal muscle activity produced by halothane anesthesia detected in previous studies by surface EMG electrodes[5,6] was localized primarily to the transversus abdominis muscle in the current study. This muscle is also preferentially recruited as compared with other abdominal muscles during carbon dioxide rebreathing in human subjects.[16] The circumferential orientation of transversus abdominis muscle fibers makes it well suited to increase abdominal pressure[7] and thus affect chest wall motion. Activity in ribcage expiratory muscles also apparently is enhanced by halothane anesthesia in some subjects, as indicated by an increased incidence of phasic expiratory activity in the diaphragm electrode.

*Chest Wall Motion*

**RIP Measurements.** The relative contribution of ribcage and diaphragm displacements to tidal volume has been estimated using measurements of external ribcage and abdominal dimensions by such techniques as the RIP, based on the conception of the chest wall as a system with two moving parts proposed by Konno and Mead.[9] Our measurements obtained with the DSR are the first direct measurements of volumes displaced by the ribcage and diaphragm in human subjects during spontaneous breathing. These direct measurements differ from RIP estimates for at least two reasons.

First, by assuming that the abdominal contents are incompressible, motion of the abdominal wall has been used to infer motion of the diaphragm. However, because the diaphragm is apposed to the lower part of the ribcage at the FRC, the lower part of the ribcage is not in contact with the lung but forms part of the abdominal cavity. This portion of the ribcage is denoted as the area of apposition. As recognized by Mead and Loring,[10] this means that, as the ribcage expands, motion of this lower ribcage increases the volume of the abdomen, which accommodates descent of the diaphragm. This expansion of the area of apposition does not contribute directly to changes in lung volume. Thus, measurements of ribcage motion should overestimate the actual contribution of the ribcage to lung volume change. Conversely, measurements of abdominal motion should underestimate the actual contribution of the diaphragm. Our finding confirms the prediction of Mead and Loring, demonstrating that RIP measurements of ribcage volume displacements overestimate the actual displacements by approximately 50% during quiet breathing while lying supine.

Second, inspiration increased the amount of intrathoracic liquid, presumably blood, in the thorax. Measurements of external thoracic dimensions cannot quantify this increase. Previous studies have documented an increase in blood flow velocity in the inferior vena cava with inspiration,[36] but we are unaware of other estimates of within-breath changes in intrathoracic blood volume in human subjects. Similar increases in intrathoracic blood volume have been observed during spontaneous breathing in anesthetized dogs.[12,13] Although the net effect of breathing on the cardiovascular system is the summation of many complex factors, such an increase is consistent with the general expectation that decreases in intrathoracic pressure will increase venous inflow and impede aortic outflow. Although an increase might be anticipated, its consistency and magnitude were unexpected. Thus, a significant portion of the total thoracic volume change during inspiration, both while awake and while anesthetized, does not displace gas but rather accommodates a substantial increase in intrathoracic blood volume.

RIP measurements continued to underestimate the actual contribution of the diaphragm and overestimate the actual contribution of the ribcage to tidal volume

during anesthesia. However, halothane anesthesia had similar qualitative effects on DSR and RIP measurements. Thus, under these conditions, the RIP was a valid tool to measure the effects of halothane on the pattern of chest wall motion, if not the actual displacements of chest wall structures. We conclude that RIP and related methods remain valuable techniques to measure anesthetic effects on chest wall motion if properly interpreted.

**Effects of Anesthesia.** Previous studies showed that halothane reduces the ribcage contribution to tidal volume as measured by changes in thoracic dimensions in the majority of subjects.[3,4] However, we found that, despite the loss of parasternal intercostal muscle activity, the outward displacement of the ribcage was relatively well preserved, with only a tendency toward a decrease in its relative contribution to the total change in intrathoracic volume; this was true for both the contribution measured directly with the DSR and the contribution estimated by RIP. The reasons for the differences from these previous studies are unknown. Differences in experimental conditions include (1) thiopental induction of anesthesia,[4] (2) study of naive adolescent subjects with a relatively high contribution of the ribcage to tidal volume while awake,[3] and (3) concomitant use of nitrous oxide.[4] Recent studies of patients anesthetized with isoflurane,[37] ketamine,[18] or methohexital[38] also noted a relative preservation of ribcage expansion during quiet breathing, but these findings may be explained by differences in anesthetic agent.

The mechanism by which inspiratory ribcage expansion is relatively well preserved despite the loss of parasternal intercostal activity is unclear. Isolated contraction of the diaphragm has both inspiratory and expiratory actions on the ribcage.[39] Inspiratory forces result from the direct action of the diaphragm *via* its insertions on the ribcage to elevate the ribs and increases in abdominal pressure acting on the lower ribcage *via* the area of apposition between diaphragm and the ribcage. Decreases in intrathoracic pressure produced by diaphragm contraction tend to constrict the ribcage and provide an expiratory action. Studies in animals[13,19] and in humans[26,39,40] suggest that isolated diaphragmatic activity distorts the ribcage, producing an inward motion of the upper ribcage and an outward motion of the lower ribcage during inspiration. We did not observe this pattern of motion during halothane anesthesia, in either DSR measurements or measurements of upper ribcage dimensions with the RIP. There are at least two potential explanations for this finding.

Because $V_T$ was relatively small during anesthesia, inspiratory decreases in intrathoracic pressure (that tend to constrict the upper ribcage) might be small. However, the mean inspiratory change in intrathoracic pressure during anesthesia, estimated from esophageal pressure, was $-4.9 \pm 2.5$ $cmH_2O$, which is similar to that present during previous studies of quiet breathing while awake.[41] This finding may reflect decreases in lung compliance caused by anesthesia.[42] Although data in humans are lacking, decreases in intrathoracic pressure of this magnitude are sufficient to constrict the upper ribcage in dogs.[13] Thus, it is likely that other muscles acted to expand the ribcage. Possibilities include other portions of the parasternal intercostal muscles that were not monitored, the posterior external intercostal muscles, the levator costae muscles, or the scalene and sternocleidomastoid muscles, all of which can demonstrate phasic inspiratory activity.[17,25–29,31,43] The scalene muscle is particularly important for upper ribcage expansion.[26,39] Expiratory ribcage muscles also may be responsible for preserved ribcage motion during halothane anesthesia. If these muscles actively reduce ribcage dimensions during expiration, the ribcage may passively expand when they relax at the onset of inspiration. Further evidence for this possibility is provided during stimulated breathing (see accompanying communication[15]).

*Mechanisms Reducing the FRC*

The FRC is reduced during halothane anesthesia with spontaneous breathing in the majority of studies.[44–48] In all of the studies that found a decrease in the FRC, anesthesia apparently was first induced with intravenous agents. Our results demonstrate that pure halothane anesthesia also reduces the FRC during spontaneous breathing. Proposed mechanisms for the reduction in the FRC caused by anesthesia include an increase in intrathoracic blood volume, an inward motion of the ribcage, and a cephalad motion of the diaphragm.

**Intrathoracic Blood Volume.** Previous studies have suggested that anesthesia with paralysis and mechanical ventilation changes the volume of blood in the thorax, which may influence the FRC; the direction and magnitude of this change remain controversial.[14,49] In a previous study using a similar methodology, Krayer *et al.* found that the induction of thiopental/fentanyl anesthesia with paralysis and mechanical ventilation produced a significant increase in thoracic liquid vol-

ume.[14] However, we found no consistent change in intrathoracic blood volume with the induction of halothane anesthesia, either when estimated from the difference between changes in the FRC and $V_{th}$ ($V_{liq}$) or measured directly from images of the heart and great vessels ($V_{ms}$). This may reflect differences in intrathoracic pressure produced by spontaneous breathing and mechanical ventilation, or different anesthetic regimens.

**Ribcage.** Although an initial study suggested otherwise,[50] it is now apparent that internal ribcage cross-sectional area at end-expiration consistently decreases during anesthesia with paralysis and mechanical ventilation.[14,49] The amount of volume displaced by this inward motion ($\approx$0.21) is similar to that found in the current study of spontaneous breathing. In contrast, Jones *et al.*[4] found that halothane anesthesia with spontaneous breathing did not change the end-expiratory ribcage circumference measured at the body surface. However, external sensors may not reflect changes in the internal dimensions of the ribcage.[14]

Drummond[8] attributed the decrease in ribcage dimensions produced by anesthesia to a loss of tonic activity in the parasternal intercostal, scalene, and sternocleidomastoid muscles as measured by surface EMG electrodes.[8] However, another study could find little evidence of such activity with subjects in the supine position.[31] We could not detect tonic activity using fine-wire electrodes placed directly in the parasternal intercostal muscles, which avoids several problems inherent to surface electrodes used in these previous studies.[51] We did not examine the scalene or sternocleidomastoid muscles in these subjects, although previous studies[31] and our own preliminary observations using fine-wire electrodes suggest that tonic activity is not present in these muscles (unpublished observations). We cannot exclude that tonic activity exists in other ribcage muscles not monitored in our studies.

Our results suggest two additional mechanisms that may constrict the ribcage. First, phasic expiratory activity was observed in the diaphragmatic electrode, presumably reflecting internal intercostal muscle activity, which could decrease ribcage dimensions. Other ribcage muscles normally not active during quiet breathing in supine humans, such as the transversus thoracis, also have expiratory actions and could be recruited during halothane anesthesia. In addition, it is possible that expiratory activity in the abdominal muscles could reduce ribcage dimensions,[7] although the net effect of isolated transversus abdominis activity on the human ribcage is not known. However, it is clear from previous studies during anesthesia-paralysis that this phasic expiratory activity is not necessary for a decrease in end-expiratory ribcage dimensions.[14,49] Also, decreases in the FRC produced by anesthesia and paralysis are similar in magnitude to those produced by halothane anesthesia with spontaneous breathing.[52] Thus, the mechanical consequences of expiratory electrical activity produced by halothane are unclear. Second, the curvature of the spinal column consistently increased, which should decrease ribcage dimensions.[53] However, in awake subjects, this inward ribcage motion does not consistently decrease lung volume[53]; effects during anesthesia are unknown. The mechanism producing changes in spinal curvature is unknown. If thoracic back muscles exhibit tonic activity while subjects are awake in the supine position, anesthesia could suppress this activity; however, it is not known whether such activity exists. Expiratory activity in the rectus abdominis muscle also could flex the spine; however, such activity is rarely observed during breathing maneuvers.[16]

**Diaphragm.** Initial studies suggested that a cephalad motion of the diaphragm was responsible for the decrease in the FRC caused by anesthesia with paralysis.[11,49] However, subsequent studies have found that, although anesthesia-paralysis changes the end-expiratory shape of the diaphragm, there is no consistent net shift in its position.[14,29,54] Only one other study has examined diaphragm position during anesthesia with spontaneous breathing. Froese and Bryan[11] examined the diaphragm silhouette in two subjects breathing spontaneously during halothane anesthesia. In one subject, they found a cephalad shift of the end-expiratory diaphragm position, greatest in the dependent regions. In the other subject, the nondependent regions moved caudad and the dependent region moved cephalad. Examples of both behaviors were seen in our study, with the latter pattern being more common (five of six subjects). In all subjects, the most dependent regions of the diaphragm moved cephalad. This pattern is similar to that seen during anesthesia with paralysis.[20] Several factors may be responsible for these changes in diaphragm shape.

The observed change in diaphragmatic shape may be related to changes in the neural activation of the diaphragm. Changes in diaphragm position with anesthesia have been attributed to loss of tonic diaphragmatic activity.[11,55,56] However, the presence of such tone in awake subjects lying supine is controversial.[31] Because

of the presence of expiratory activity in the diaphragmatic electrode, probably originating from the adjacent internal intercostal muscle, our measurements cannot confirm or deny the presence of such activity. In the two subjects with no phasic expiratory activity in the diaphragmatic electrode, tonic activity was not readily apparent in this costal portion of the diaphragm.

Alterations in the shape of the diaphragm also may be related to motions of its insertions on the thoracoabdominal wall. Because the anterior diaphragm inserts near the costal margin, motion of the anterior ribcage may affect the diaphragm. In the five subjects in whom the sternum moved caudad with the induction of anesthesia, the anterior portion of the diaphragm also moved caudad. The converse was true in subject 6, whose sternum moved cephalad with the induction of anesthesia (fig. 6). Thus, changes in the end-expiratory position of the anterior diaphragm appear to be closely related to motion of the ribcage. Because the posterior portion of the diaphragm inserts on vertebral bodies, an increase in spinal curvature may tend to move the posterior portion of the diaphragm cephalad.

Finally, expiratory activity in the abdominal muscles should increase pressure within the abdomen and tend to displace the diaphragm cephalad. The transversus abdominis, which exhibited the most consistent phasic expiratory activity during halothane anesthesia, is particularly efficient at increasing abdominal pressure.[7] However, it is not known whether the change in gastric pressure produced by such activity during quiet breathing is sufficient to affect diaphragm position.

These results demonstrate that the effects of anesthetics on respiratory muscle activity cannot be simply inferred from chest wall motion. Despite the abolition of parasternal intercostal muscle activity, the contribution of the ribcage to tidal volume is relatively well preserved during halothane anesthesia. Thoracic imaging further reveals that an inward displacement of the ribcage accounts for most of the decrease in the FRC caused by halothane with spontaneous breathing, accompanied by changes in diaphragm shape that may be related to motion of its insertions on the thoracoabdominal wall.

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## Appendix

Our measurements were performed with the subject's arms elevated above the head to permit thoracic imaging and with the

**Table A1. Effects of Arm Position and Mouthpiece on Pattern of Quiet Breathing**

| | | Compartmental Displacement | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | | Rib Cage | | Diaphragm–Abdomen | | | | |
| Arm Position | Mouthpiece | ml | % $V_T$ | ml | % $V_T$ | $V_T$ (ml) (RIP) | $V_T$ (ml) (pneumotachograph) | f (min) |
| Up | In | 227 ± 34 | 33 ± 4 | 492 ± 98 | 67 ± 4 | 718 ± 124 | 740 ± 135 | 10.7 ± 2.1 |
| | Out | 196 ± 30 | 33 ± 4 | 418 ± 65 | 67 ± 4 | 613 ± 88 | — | 12.3 ± 2.5 |
| Down | In | 223 ± 49 | 34 ± 7 | 459 ± 100 | 66 ± 7 | 682 ± 126 | 684 ± 118 | 10.7 ± 1.5 |
| | Out | 213 ± 35 | 39 ± 6 | 353 ± 62 | 61 ± 6 | 566 ± 85 | — | 12.2 ± 1.7 |

$V_T$ = tidal volume, measured using respiratory impedance plethysmograph (RIP) or a pneumotachograph; f = breathing frequency. Compartmental displacements were measured using RIP.

Values are mean ± SE.

subject breathing through a mouthpiece while using a nose clip. In animals, elevation of the upper limbs can increase the FRC,[13] and in human subjects, breathing through a mouthpiece can affect the pattern of breathing.[57,58] To assess these possible effects in our subjects, measurements before DSR scanning while awake were obtained during quiet breathing with the arms up in the position used during scanning (humerus approximately vertical) and with the arms down at the subject's side. Measurements were made in both positions with and without the mouthpiece and nose clip. The RIP was calibrated with the arms in both positions. The FRC measured before the study, with the arms at the side (2.50 ± 0.35 l), did not differ significantly from the FRC measured during the study with the arms up (2.54 ± 0.31 l). Also, the volume-motion coefficients of the ribcage and abdomen, which depend in part on chest wall geometry, did not differ with the arms up or down (data not shown). As noted in previous studies, breathing through a mouthpiece tended to increase tidal volume and decrease breathing frequency,[57,58] but these changes were not significant (table A1). We thus consider that the results obtained are representative of quiet breathing.