Position and Motion of the Human Diaphragm during Anesthesia-paralysis

Sebastian Krayer, M.D.,* Kai Rehder, M.D.,† Jörg Vettermann, M.D.,‡ E. Paul Didier, M.D.,§ Erik L. Ritman, M.D.¶

Regional motion of the human diaphragm was determined by high-speed, three-dimensional x-ray computed tomography. Six healthy volunteers were studied first while awake and breathing spontaneously and again while anesthetized-paralyzed and their lungs ventilated mechanically. Tidal volume (V_T) and respiratory frequency were similar during both conditions. Three subjects were studied while they were supine and three while they were prone. During spontaneous breathing, movement of dependent diaphragm regions was greater than that of nondependent regions in four of six subjects. In five of the six subjects, dorsal diaphragm movement exceeded ventral movement regardless of body position. The volume displaced by the diaphragm (ΔV_{di}) was similar to V_T in supine subjects but tended to be less than V_T in prone subjects. After induction of anesthesia-paralysis, the end-expiratory position of the diaphragm did not change consistently in supine subjects, whereas a consistent cephalad volume shift occurred in prone subjects. During anesthesia-paralysis and mechanical ventilation, ΔV_{di} was reduced to approximately 50% of V_T in both body positions. In the supine position, the pattern of diaphragm motion during mechanical inflation was nearly uniform. By contrast, in the prone position, the motion was nonuniform, with most motion occurring in the dorsal (nondependent) regions. It is concluded that the dominant influence on diaphragm motion may be some anatomical difference between the crural and costal diaphragm regions rather than the abdominal hydrostatic pressure gradient. (Key words: Measurement technique: dynamic spatial reconstructor. Muscle, skeletal: diaphragm. Positions: prone; supine. Ventilation: mechanical; spontaneous.)

FROESE AND BRYAN[1] found that, in awake humans lying either supine or in a lateral decubitus position, during spontaneous breathing, the dependent parts of the diaphragm moved more in a cephalocaudal direction than did the nondependent parts. The authors suggested that the dependent regions of the diaphragm can generate larger pressures than the nondependent regions can, be-

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* Research Associate.

† Professor of Anesthesiology and of Physiology, Mayo Medical School.

‡ Research Fellow.

§ Associate Professor of Anesthesiology, Mayo Medical School.

¶ Professor of Physiology and of Medicine, Mayo Medical School.

cause of a more favorable length-tension relationship and a smaller radius of curvature. They also observed that, during anesthesia-paralysis and mechanical ventilation with normal tidal volume, the pattern of diaphragm displacement was reversed, with more motion occurring in nondependent than in dependent regions, and that a cephalad shift of the end-expiratory position of the diaphragm occurred.

The major mechanism responsible for the shift in end-expiratory position of the diaphragm with anesthesia-paralysis and for the pattern of motion during spontaneous breathing and mechanical ventilation was attributed to the vertical abdominal hydrostatic pressure gradient. If Froese and Bryan's[1] hypothesis is correct, both the position of the diaphragm and its motion should be independent of body position during either mode of ventilation. To test this hypothesis, we examined diaphragm position and motion in six volunteers lying either supine or prone, first while they were breathing spontaneously and then while they were anesthetized-paralyzed and their lungs mechanically inflated. We employed three-dimensional x-ray computerized tomography (Dynamic Spatial Reconstructor) to obtain a detailed description of the shape and motion of the human diaphragm.

Materials and Methods

Six healthy young volunteers (table 1) were accepted for the study after signing an informed consent that explained the nature and risk of the study. Each subject had a complete physical examination, including pulmonary function testing and pregnancy tests in the female volunteers. All subjects were nonsmokers. The study was approved by the Institutional Review Board.

The diaphragm of each subject was scanned with the Dynamic Spatial Reconstructor (DSR),[2,3] first during spontaneous breathing while awake and again during mechanical ventilation after induction of anesthesia-paralysis. Three subjects were studied while they were supine, and the other three subjects were studied while they were prone. The subjects had their arms raised above the head so that the arms would not be in the imaging field of the DSR. The shoulders and hips of the prone subjects were supported to allow the abdomen and rib cage to move freely during respiration. The awake subjects had their nostrils occluded and were instructed to breathe at a slow rate with normal tidal volume through a mouthpiece and

TABLE 1. Physical Characteristics of Six Volunteers

Subject	Sex and age (yr)	Height (cm)	Weight (kg)	FEV₁/FVC %
Supine				
1	M, 27	170	64	79
2	M, 33	172	78	83
3	M, 34	173	67	77
Prone				
4	F, 31	173	68	80
5	F, 26	167	53	82
6	M, 34	183	73	78

a Hans Rudolph valve connected to a pneumotachograph (Fleisch no. 1) coupled to a differential pressure transducer (Validyne MP45). Tidal volumes were determined by integration of inspiratory gas flow. Tidal volumes while awake averaged 786 ± 134 (SD) ml (11.8 ± 2.3 ml/kg BW), respiratory frequencies 7.7 ± 2.4 min^{-1}; the inspiratory/expiratory ratio was 0.86 ± 0.13. All subjects were asked to take two big breaths 2 min prior to the DSR-scanning.

ANESTHESIA

Before induction of anesthesia, all subjects received 0.4 mg of atropine intravenously. General anesthesia was then induced, while the subjects were supine, with thiopental sodium (2.6–5.7 mg/kg), midazolam (5 mg), and fentanyl (1.4–5.5 μg/kg). Succinylcholine chloride (1.3–1.6 mg/kg) was given and both hypopharynx and trachea were topically anesthetized (lidocaine 2%) to facilitate intubation with a cuffed oral endotracheal tube (I.D. 8.0–8.5 mm). To reduce muscular fasciculation, 2 mg of metocurine were administered intravenously prior to the injection of succinylcholine chloride. The three subjects to be studied prone were repositioned immediately after induction of anesthesia. Anesthesia was supplemented with thiopental sodium (0.6–1.2 mg/kg) and muscle paralysis was induced with succinylcholine chloride (1.2–1.9 mg/kg) and confirmed by ulnar nerve stimulation (train-of-four) prior to all scans. The subjects' lungs were mechanically ventilated (Siemens 900B) with 30% oxygen in nitrogen with tidal volumes (759 ± 95 ml; 11.5 ± 2.0 ml/kg BW) and respiratory frequencies similar to those observed during spontaneous breathing in the awake state. End-inspiratory pressure averaged 12.0 ± 1.8 cm H_2O and end-expiratory pressure was zero. Two hyperinflations to 30 cm H_2O airway pressure were performed 3 min before scanning. The electrocardiogram (lead II), blood pressure, and oxygen saturation (pulse oximetry; Nellcor) were monitored throughout the study. The radial artery was cannulated after induction of anesthesia to determine blood gas tensions and blood pressure.

DYNAMIC SPATIAL RECONSTRUCTOR

The DSR generates simultaneous multiple x-ray projection images that are used to reconstruct a three-dimensional array of gray-scale values in computer memory, producing a volume image. The technique has been described in detail elsewhere.[3] We used the extended transaxial field-of-view scanning mode,[4] which allows the scanning of a cylindrical volume with an axial height of 21.5 cm and transaxial diameter of 39 cm. Previously, we showed that volumes determined by this technique can be determined with an accuracy of 2%.[5]

DSR scans of the lower thorax, including the diaphragm, were triggered manually near end-expiration and near end-inspiration; each scan had a duration of 360 ms. Images at the same point in the breathing cycle were recorded during three consecutive breaths of spontaneous breathing (without breath-holding) in the awake state and again during two consecutive breaths of mechanical ventilation in the anesthetized-paralyzed state. The projection images of the diaphragm at corresponding lung volumes were combined to reconstruct the final volume images of the diaphragm at end-expiration and end-inspiration for both modes of ventilation. The diaphragm could not be scanned during the entire breath because of the limitation in radiation exposure. For the same reason, each subject could be studied in only one body position. The radiation exposure of the skin averaged 893 ± 199 mR (M $\pm$ SD) over the xiphoid, 15 ± 3 mR over the thyroid gland, and 7 ± 3 mR over the gonads. The volume images consisted of cubic volume elements (voxels) with side lengths of 2.6 mm (0.018 ml volume). The scalar value of each voxel represented the average x-ray attenuation at this point in space.

DATA ANALYSIS

Between 4,000 and 5,000 diaphragm surface coordinates were determined using computerized edge detection (the difference in density between the abdominal contents and the lung) and computer-operator interaction. Figure 1 shows examples of computer-generated diaphragm surfaces of a subject lying supine and another lying prone.

Diaphragm surfaces were compared by computing the cephalocaudal distances of horizontally oriented surface coordinates. Thus, the net diaphragm motion was described in cephalocaudal (horizontal) direction relative to a fixed structure such as the spinal column. Therefore, the data do not provide information about the motion of material points of the diaphragm, i.e., cannot be translated into changes of muscle length.

Volume displacement by the diaphragm during a breath was calculated from the volume (number of voxels) enclosed by the surface of the diaphragm at end-expiration and end-inspiration (fig. 2A). The volume displacement of the cephalad margin of the area of apposition of the diaphragm caused by rib cage shape and motion represents a triangle of rotation whose volume was estimated separately and added to the diaphragm volume displace-
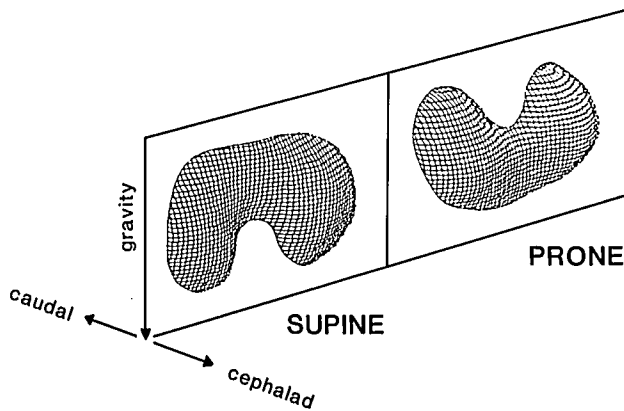
FIG. 1. Wire-mesh graphs of reconstructed diaphragm surfaces at end-expiration of awake subjects lying supine or prone. Surfaces are presented in cephalolateral view. The indentation in the wire-mesh marks the spinal column.

ment. For computational purpose, the surfaces of the diaphragm at end-expiration and at end-inspiration were extrapolated so that they had the same cross-sectional area. The volume displaced by the diaphragm edge was then computed as if it had moved in a cylinder (fig. 2B and C). This volume was divided in half to estimate the volume change in the area of apposition (fig. 2B). This volume averaged $5.1 \pm 2.8\%$ (SD) of the diaphragm volume displacement during tidal breathing.

To display regional diaphragm motion, gray-scale values corresponding to the cephalocaudal motion were assigned to each point in the transverse projections. Thus, the entire diaphragm motion was mapped in a top view (looking down from the head to the diaphragm) of the diaphragm, creating a contour map of regions having equal motion (figs. 3–5). Note that the shape of a top view of the diaphragm does not correspond to its cross-sectional
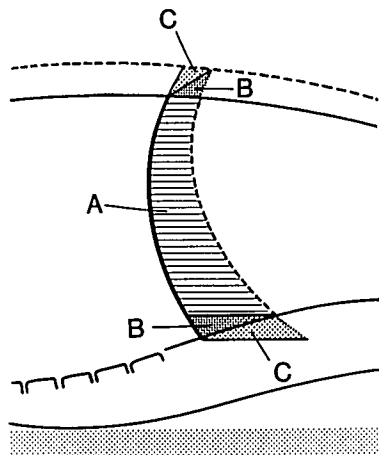
FIG. 3. Horizontal diaphragm displacements of supine subjects are projected on the cephalad view of the diaphragm. In each subject, the gray scale was normalized for the average displacement of the diaphragm. Dark-shaded areas moved more than light-shaded areas. Note the more uniform displacement during anesthesia-paralysis and mechanical ventilation when compared with spontaneous breathing during the awake state.

FIG. 2. Calculation of volume displacement of diaphragm. Contours of thorax, abdomen, and thoracic wall are shown as solid lines at end-expiration and as broken lines at end-inspiration. Volume displacement of diaphragm was obtained from the cephalocaudal difference of surface coordinates (*A*). Diaphragm surfaces were extrapolated to the same cross-sectional area to calculate *B* and *C*. *B* were taken as an approximation of the contribution of the motion of the cephalad margin of the area of apposition to diaphragm displacement.

FIG. 4. Horizontal diaphragm displacements of prone subjects are projected on the cephalad view of the diaphragm. For definition of gray scales, see legend to figure 3. Note the increased vertical gradient of displacement during anesthesia-paralysis and mechanical ventilation when compared with spontaneous breathing during the awake state.

ages were analyzed in duplicate by two computer operators.

Individual data represent the mean of two analyses. Group values are expressed as mean ± SD. Student's paired *t* test was used for statistical comparison when appropriate.

Results

The duration of the study averaged 74 ± 26 min, with a mean anesthesia time of 39 ± 18 min. No untoward reactions occurred. Mechanical ventilation during anesthesia-paralysis resulted in a mean arterial carbon dioxide tension of 31 ± 2 mmHg, a mean arterial oxygen tension of 174 ± 15 mmHg, and a mean pH of 7.49 ± 0.04.

DIAPHRAGM VOLUME DISPLACEMENT IN AWAKE SUBJECTS DURING SPONTANEOUS BREATHING

In the awake state, mean diaphragm volume displacement (ΔV_{di}) was similar to tidal volume (V_T) in supine subjects (94–109% of V_T), but tended to be less than V_T in the prone subjects (63–97% of V_T) (table 2).

Diaphragm motion during spontaneous breathing was nonuniform in both the supine and prone positions (figs. 3 and 4, upper panels). Compared with the left hemidiaphragm, the right hemidiaphragm displaced more volume (9 ± 8% of V_T) ($P < 0.05$) and its dome was more cephalad. The mean difference between the most cephalad portion of the left and right domes was 1.9 ± 0.7 cm at end-expiration and 1.3 ± 0.7 cm at end-inspiration, indicating a larger volume displacement of the right hemidiaphragm during spontaneous breathing.

FIG. 5. Volume displacement by diaphragm after induction of anesthesia-paralysis is projected on the cephalad view of the diaphragm for supine subjects and prone subjects. The volume shift in caudal direction (negative values) is indicated by white areas. Note the consistent cephalad shift in dorsal regions.

area because of the irregular cephalocaudal height of the diaphragm edge. To examine ventrodorsal differences in diaphragm position and average cephalocaudal displacement, the cephalocaudal coordinates at any given ventrodorsal height of the diaphragm were averaged and plotted against ventrodorsal distance (figs. 6, 7). All volume im-
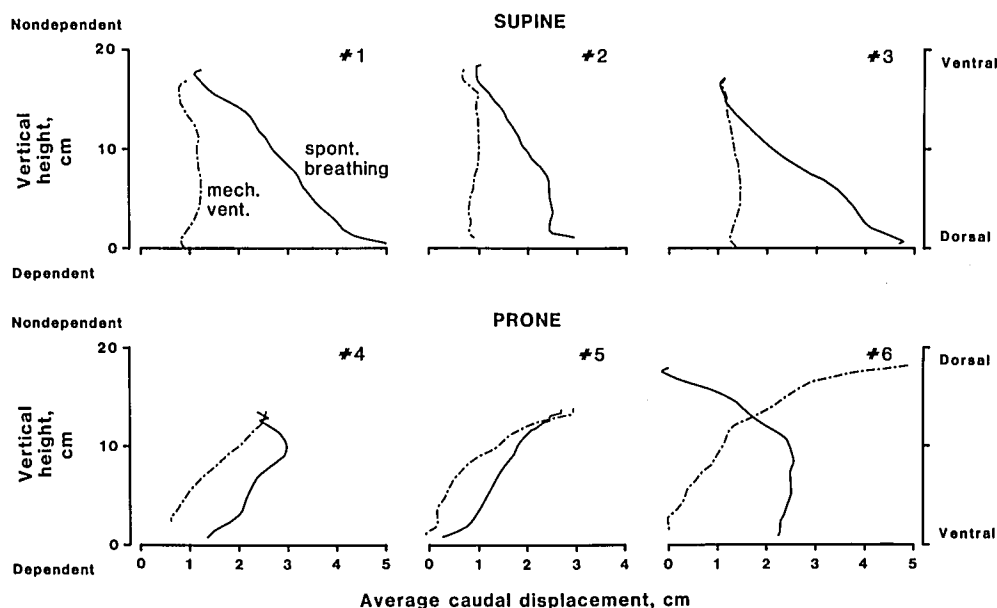

FIG. 6. Average motion of diaphragm for supine and prone subjects during spontaneous breathing while awake (solid lines) and during mechanical ventilation while anesthetized-paralyzed (broken lines). In dependent regions, motion of the diaphragm was less during mechanical ventilation than during spontaneous breathing.

In all three supine subjects and in two of the three prone subjects, movement of the dorsal regions of the diaphragm was greater than that of the ventral regions during spontaneous breathing (fig. 6).

CHANGE IN END-EXPIRATORY POSITION OF DIAPHRAGM AFTER INDUCTION OF ANESTHESIA-PARALYSIS

After induction of anesthesia-paralysis, the dorsal regions of the diaphragm were displaced in a cephalad direction (fig. 5) in all six subjects. However, the overall volume shift of the diaphragm after induction of anesthesia-paralysis was inconsistent in the supine subjects; a cephalad volume shift occurred in two subjects (patients 1 and 3) and a caudal shift occurred in one subject (patient 2) (fig. 5). By contrast, a consistent cephalad volume shift of the diaphragm was observed (615 ± 466 ml) in all three prone subjects (fig. 5).

DIAPHRAGM VOLUME DISPLACEMENT IN ANESTHETIZED-PARALYZED SUBJECTS DURING MECHANICAL VENTILATION

In both body positions, displacement of dependent regions of the diaphragm was consistently less during anesthesia-paralysis and mechanical ventilation than during spontaneous breathing (fig. 6). The displacement of nondependent regions was also less or similar during mechanical ventilation than during spontaneous breathing in five of the six subjects.

During mechanical ventilation, mean ΔV_{di} was reduced from $101 \pm 7\%$ to $53 \pm 5\%$ of V_T ($P < 0.02$) in supine subjects and from $82 \pm 16\%$ to $51 \pm 10\%$ of V_T ($P < 0.02$) in prone subjects (table 2). Mechanical ventilation also altered the pattern of motion of the diaphragm (figs. 3, 4). In supine subjects, the motion of the diaphragm during anesthesia-paralysis and mechanical ventilation was nearly uniform (piston-like) (fig. 3, lower panel). By contrast, the motion of the diaphragm was nonuniform during mechanical ventilation in the anesthetized-paralyzed prone subjects (fig. 4, lower panel).

At end-expiration during anesthesia-paralysis and mechanical ventilation, the right diaphragm dome was more cephalad than the left dome (1.7 ± 0.8 cm). The difference in position between the left and right hemidiaphragms was unchanged with inspiration; the right hemidiaphragm remained 1.6 ± 0.6 cm more cephalad.

Discussion

DIAPHRAGM CONTRIBUTION TO TIDAL VOLUME

Some studies[6-10] have estimated that the rib cage contributes between 15% and 40% to the tidal volume during
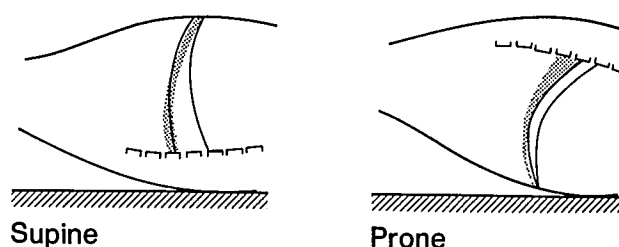

Supine   **Prone**

FIG. 7. Diagrammatic diaphragm position and displacement for supine or prone subjects. Solid lines represent end-expiratory and end-inspiratory diaphragm positions during spontaneous breathing while awake. Stippled areas represent diaphragm excursions during mechanical ventilation while anesthetized-paralyzed.

spontaneous breathing in awake supine humans; cortical influences may have contributed to these large differences. In those studies, sensors were used to detect the motion of the body surface. These measurements may have overestimated the contribution of the rib cage to tidal volume.[11] Recently, Whitelaw,[12] employing x-ray computerized tomography, estimated the diaphragm contribution to be 68% of the tidal volume in one awake supine subject. In that study, the diaphragm was scanned during breath-holding with the glottis open. This maneuver may have altered both the chest wall shape and the intrathoracic blood volume and thereby altered the position of the diaphragm. To circumvent these problems, we used three-dimensional reconstruction of the diaphragm to determine its contribution to the tidal volume during a normal breathing cycle, i.e., without breath-holding. In awake supine subjects with their arms raised above their heads, we found that the diaphragm volume displacement alone was as large as the tidal volume. This surprising finding suggests that the overall increase in thoracic cavity volume during spontaneous breathing was larger than the tidal volume, because the rib cage presumably also contributed to an increase in thoracic volume. The most likely explanation for this finding is an increase in thoracic blood volume with spontaneous inspiration. The contribution of the diaphragm to the tidal volume was consistently reduced ($P < 0.01$) during anesthesia-paralysis and mechanical ventilation. This observation agrees with measurements using body surface sensors.[6-10]

TABLE 2. Contribution of Diaphragm to Tidal Volume (Mean ± SD)

	Supine (n = 3)		Prone (n = 3)	
	Awake	Anesthesia-paralysis	Awake	Anesthesia-paralysis
V_T (ml)	865 ± 147	795 ± 128	707 ± 67	723 ± 47
ΔV_{di} (ml)	874 ± 159	419 ± 72	585 ± 158	371 ± 88
% of V_T	101 ± 7	$53 \pm 5^*$	$82 \pm 16\dagger$	$51 \pm 10^*$

Statistical significant difference: *awake vs. anesthesia-paralysis, $P < 0.02$; †supine vs. prone, NS.

Independent of body position and mode of ventilation, the right diaphragm dome was always more cephalad than the left diaphragm dome. The volume displacement of the right hemidiaphragm was also larger than that of the left hemidiaphragm during spontaneous breathing in the awake state, but not during mechanical ventilation. This difference in volume displacement between spontaneous breathing and mechanical ventilation is consistent with the hypothesis that the shortening may be different for the left and right hemidiaphragms.[12] This difference in shortening may be caused by differences in the mechanical properties of the two hemidiaphragms, in the phrenic innervation, in the preload and afterload of the muscle fibers of the two hemidiaphragms, or in some combination of these.

PATTERN OF DIAPHRAGM DISPLACEMENT DURING SPONTANEOUS BREATHING

In the three supine subjects and in one prone subject, the dependent regions of the diaphragm moved more than the nondependent regions during spontaneous breathing while awake. This observation is in agreement with that of Froese and Bryan[1] in supine subjects. Those authors suggested that the abdominal hydrostatic pressure gradient caused by gravity determines the pattern of diaphragm motion. Froese and Bryan[1] argued that the dependent parts of the diaphragm developed a greater pressure than did the nondependent parts. They based their argument on LaPlace's law $[\Delta P = T \times (1/r_1 + 1/r_2)]$, in which ΔP is the pressure developed by the diaphragm, T is the tension of the diaphragm, and r_1 and r_2 are the principal radii of curvature of the diaphragm. Dependent parts of the diaphragm have smaller principal radii of curvature because the larger abdominal hydrostatic pressure in this region preferentially stretches the muscle fibers. Therefore, if tension (T) were uniform, a greater pressure (ΔP) could be developed. Currently, however, it is recognized that the canine diaphragm is not uniform but consists of two muscles (crural and costal)[13] that are arranged partially in series and partially in parallel.[14] Tension between these two muscles may not be completely transmitted; that is, the tension may not be uniform. In the presence of nonuniform diaphragm tension, the pressure developed by the diaphragm does not depend only on the principal radii of curvature. Froese and Bryan[1] also suggested that the dependent diaphragm regions, which are more stretched, are able to generate a greater force. Stretching of the diaphragm may produce a more favorable length-tension relationship because the diaphragm operates normally at a shorter length than optimal.[15] Stretching of the muscle fiber, however, depends not only on the transdiaphragmatic pressure, but also on the compliance of the fibers. Whether regional differences

in compliances exist in the human diaphragm is unknown, but the crural part of the canine diaphragm is more compliant than the costal part.[16]

In five of the six subjects in the present study, dorsal regions moved more than ventral regions during spontaneous breathing, regardless of the body position. This preferential motion of the dorsal regions could be caused either by a difference in neural activation of the diaphragm between crural (dorsal) and costal (ventral) regions or by a different force resulting from a common neural activity. Therefore, the determinants of the pattern of active diaphragm displacement seem to be more complex than proposed by Froese and Bryan.[1] But, it should be noted that our subjects had their arms raised above their heads, which may have resulted in traction on the chest wall that may affect both the motion and position of the diaphragm.

CHANGE IN END-EXPIRATORY POSITION OF THE DIAPHRAGM WITH INDUCTION OF ANESTHESIA-PARALYSIS

Induction of anesthesia-paralysis altered the end-expiratory position of the diaphragm. In supine subjects, the volume displacement associated with the change in the end-expiratory position of the diaphragm was small and the direction was not consistent. This finding confirms our previous observation that a cephalad shift of the diaphragm contributes little to the reduction in resting lung volume (FRC) after induction of anesthesia-paralysis in supine normal humans.[2] The finding also agrees in part with that of Froese and Bryan,[1] who noted little or no cephalad displacement of the diaphragm in one of two subjects. By contrast, in prone subjects, a cephalad volume shift of the end-expiratory position of the diaphragm occurred consistently with induction of anesthesia-paralysis. Thus, the change in the end-expiratory position does not seem to be a major contributor to the reduction of FRC in supine subjects, but may be a major contributor to the reduction of FRC in prone subjects.[17] The underlying mechanisms for the change in the end-expiratory diaphragm position after induction of anesthesia-paralysis in the prone position are unknown.

Regardless of body position, dorsal diaphragm regions were consistently shifted cephalad, a finding suggesting that these regions may have an active tone when the subjects were awake.

Hedenstierna *et al.*[18] stated that "the diaphragm moved cranially, the major part of the dome by an average of 1.9 cm," and concluded that "the diaphragm moved cranially by a distance corresponding to a volume of about 500 ml." We confirmed that some parts of the diaphragm are always displaced in a cephalad direction after induction of anesthesia-paralysis (black areas in figure 5). However,

other parts may be displaced caudally at the same time (white areas in figure 5). The overall cephalad displacement of the diaphragm is therefore much less than estimated by Hedenstierna *et al.* from computerized tomographs. Thus, we question the validity of the balance of volume shifts occurring after induction of anesthesia-paralysis proposed by Hedenstierna *et al.*[18]

PATTERN OF DIAPHRAGM DISPLACEMENT DURING MECHANICAL VENTILATION

Regardless of body position, dependent diaphragm regions moved less during anesthesia-paralysis than during spontaneous breathing (fig. 6). This finding is in agreement with the observations of Froese and Bryan.[1] In our study, the contribution of the nondependent part of the diaphragm to the tidal volume, however, was not larger than that during spontaneous breathing in five of six subjects. This finding is not in agreement with the observations and hypothesis of Froese and Bryan.[1] It is unlikely that this different finding is due to the choice of anesthetic, since Froese and Bryan found no difference between two subjects anesthetized with halothane and N_2O and one subject anesthetized with a barbiturate. Another possibility includes the influence of the size of tidal volume. In Froese and Bryan's study,[1] predominant motion of nondependent diaphragm regions during mechanical ventilation was observed when the tidal volume was in the range of 5–6 ml/kg BW and a piston-like motion when the tidal volume was larger than 14 mg/kg. In our study, the subject with the smallest tidal volume (8.3 ml/kg) and lying supine showed already a piston-like diaphragm motion. Froese and Bryan found that the nondependent region of the human diaphragm moved more during mechanical ventilation with normal tidal volume than during spontaneous breathing and explained this finding by suggesting that the uniform increase in thoracic pressure during mechanical ventilation displaces the diaphragm most where the opposing abdominal pressure is least; that is, in nondependent regions. However, a uniform increase in thoracic pressure should uniformly increase the pressure difference across a paralyzed diaphragm if the vertical abdominal pressure gradient is not changed. Thus, mechanical ventilation should result in a uniform (piston-like) motion, as was observed in the supine subjects. However, since the diaphragm does not move as a piston in prone subjects during anesthesia-paralysis, the pattern of motion of a paralyzed diaphragm apparently does not depend only on the distribution of trans-diaphragmatic pressure. Other mechanisms must also contribute to the pattern of motion of the paralyzed diaphragm, as suggested by the difference in pattern of motion during spontaneous breathing between the supine and prone positions. The distribution of passive tension

in the diaphragm, the principal radii of curvature of the diaphragm, and the thoracoabdominal coupling of the diaphragm, including thoracoabdominal anatomy, may all contribute to the pattern of motion of the paralyzed diaphragm during mechanical ventilation.

Considering figure 6, it is apparent that muscle paralysis does not impact on dependent diaphragm motion nearly as much in the prone as in the supine position, largely because the dependent diaphragm moved only little during spontaneous breathing in the prone position. This observation suggests that the costal diaphragm regions do not load-compensate as well as crural regions during active contraction, *i.e.*, there may be a neuromuscular difference between these regions.

DIAPHRAGM DISPLACEMENT AND DISTRIBUTION OF VENTILATION

Although we did not measure intrapulmonary inspired gas distribution, it is of interest to relate it to the observed pattern of diaphragm movement. *In vivo*, the shapes of the diaphragm and lungs must conform to each other. The motion of the diaphragm may therefore affect the pattern of lung expansion and hence the distribution of inspired gas. During spontaneous breathing in supine or lateral decubitus positions, dependent diaphragm regions move more than nondependent regions, and the former regions have a larger ventilation per unit lung volume than the latter.[19–21] After induction of anesthesia-paralysis and mechanical ventilation of supine subjects, changes in the pattern of motion of the diaphragm correspond to changes in distribution of inspired gas.[19] Both the displacement of the nondependent diaphragm and the ventilation of nondependent lung regions increase relative to that of dependent regions.

By contrast, in the prone position, diaphragm displacement does not match the distribution of inspired gas. During spontaneous breathing and during anesthesia-paralysis with mechanical ventilation, the ventilation per unit lung volume of dependent regions is large,[17] but the nondependent diaphragm regions are preferentially displaced (fig. 6).

During anesthesia-paralysis with mechanical ventilation, the ventrodorsal distribution of inspired gas is similar in prone and supine positions,[17] although diaphragm motion differs considerably between these body positions (fig. 7). The discrepancy between intrapulmonary inspired gas distribution and diaphragm motion demonstrates that regional ventilation is not determined by the motion of the diaphragm alone. Intrapulmonary inspired gas distribution is modulated by slippage of lung lobes, by mechanical interdependence of the lung parenchyma, or by both means.

In summary, during spontaneous breathing, dorsal regions of the diaphragm moved more than ventral regions in five of six subjects, regardless of body position. There was no consistent volume shift by the diaphragm after induction of anesthesia-paralysis in supine subjects. By contrast, a consistent volume shift occurred in prone subjects. In both the supine and prone positions, the pattern of motion of the paralyzed diaphragm differed during mechanical ventilation. In the supine position, the diaphragm moved uniformly (piston-like), while, in the prone position, the motion was nonuniform, with the dependent (ventral) parts moving least. We conclude from these findings that the anatomical features of the diaphragm may be a more important determinant of the pattern of diaphragm motion than the abdominal hydrostatic pressure gradient.

References

1. Froese AB, Bryan AC: Effects of anesthesia and paralysis on diaphragmatic mechanics in man. ANESTHESIOLOGY 41:242–255, 1974

2. Krayer S, Rehder K, Beck KC, Cameron PD, Didier EP, Hoffman EA: Quantification of thoracic volumes by three-dimensional imaging. J Appl Physiol 62:591–598, 1987

3. Ritman EL, Robb RA, Harris LD: Imaging Physiological Functions: Experience with the Dynamic Spatial Reconstructor. New York, Praeger, 1985

4. Hoffman EA, Sinak LJ, Robb RA, Ritman EL: Noninvasive quantitative imaging of shape and volume of lungs. J Appl Physiol 54:1414–1421, 1983

5. Krayer S, Decramer M, Vettermann J, Ritman EL, Rehder K: Volume quantification of chest wall motion in dogs. J Appl Physiol 65:2213–2220, 1988

6. Grimby G, Hedenstierna G, Löfström B: Chest wall mechanics during artificial ventilation. J Appl Physiol 38:576–580, 1975

7. 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

8. Vellody VPS, Nassery M, Balasaraswathi K, Goldberg NB, Sharp JT: Compliances of human rib cage and diaphragm-abdomen pathways in relaxed versus paralyzed states. Am Rev Respir Dis 118:479–491, 1978

9. Jones JG, Faithfull D, Jordan C, Minty B: Rib cage movement during halothane anaesthesia in man. Br J Anaesth 51:399–407, 1979

10. Hedenstierna G, Löfström B, Lundh R: Thoracic gas volume and chest-abdomen dimensions during anesthesia and muscle paralysis. ANESTHESIOLOGY 55:499–506, 1981

11. Mead J, Loring SH: Analysis of volume displacement and length changes of the diaphragm during breathing. J Appl Physiol 53:750–755, 1982

12. Whitelaw WA: Shape and size of the human diaphragm in vivo. J Appl Physiol 62:180–186, 1987

13. De Troyer A, Sampson M, Sigrist S, Macklem PT: The diaphragm: Two muscles. Science 213:237–238, 1981

14. Decramer M, De Troyer A, Kelly S, Macklem PT: Mechanical arrangement of costal and crural diaphragms in dogs. J Appl Physiol 56:1484–1490, 1984

15. Braun NMT, Arora NS, Rochester DF: Force-length relationship of the normal human diaphragm. J Appl Physiol 53:405–412, 1982

16. Road J, Newman S, Derenne JP, Grassino A: In vivo length-force relationship of canine diaphragm. J Appl Physiol 60:63–70, 1986

17. Rehder K, Knopp TJ, Sessler AD: Regional intrapulmonary gas distribution in awake and anesthetized-paralyzed prone man. J Appl Physiol 45:528–535, 1978

18. Hedenstierna G, Strandberg Å, 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

19. Rehder K, Sessler AD, Rodarte JR: Regional intrapulmonary gas distribution in awake and anesthetized-paralyzed man. J Appl Physiol 42:391–402, 1977

20. Kaneko K, Milic-Emili J, Dolovich MB, Dawson A, Bates DV: Regional distribution of ventilation and perfusion as a function of body position. J Appl Physiol 21:767–777, 1966

21. Milic-Emili J, Henderson JAM, Dolovich MB, Trop D, Kaneko K: Regional distribution of inspired gas in the lung. J Appl Physiol 21:749–759, 1966