aminophylline does increase phrenic nerve activity, its influence on the central drive to other inspiratory muscles is unknown. Intuitively, I would predict that if aminophylline increases central inspiratory drive, it would do so to all inspiratory muscles, not just the diaphragm. If this is correct, one would expect to see proportionate increases in Pdi and Edi with no change in the ratio ΔPga/ΔPdi.

If, however, the results observed by Dureuil *et al.*[1] are explained by an increase in diaphragmatic contractility, one would expect to see an increase in Pdi for any given Edi. Whether this was due to changes in length, velocity of contraction, or inotrophy would be left for future investigations to determine. Nevertheless, the assessment of Pdi/Edi could go a long way to understanding the action of various interventions on inspiratory muscle function. Space does not permit a detailed exposition of the technical problems of measuring Pdi and Edi, particularly when the ratio between these two parameters is measured with long intervals in between. Although I do not disagree with the conclusions of Dureuil *et al.*,[1] the assessment of the contribution of the diaphragm to the respiratory pressure swings and of diaphragmatic contractility are fraught with technical problems. It is not a job for amateurs. Nevertheless, the job requires doing. The influence of methyl xanthines on inspiratory muscle function is important. In certain situations it might be risky. The possibility exists that abolishing a reflex is disadvantageous to the patient. Furthermore, if methyl xanthines increase the contractile force of the diaphragm when the diaphragm is working against a fatiguing load, it may be like flogging a dead horse. If so, diaphragmatic fatigue and respiratory failure may be hastened rather than retarded or prevented. In spite of these caveats, the recent report of Murciano *et al.*[2] suggest that the long-term use of aminophylline indeed may be beneficial.

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New Dimensions of the Respiratory System

A DECREASE in functional residual capacity (FRC) after the induction of general anesthesia is of interest to both the clinician and the investigator. This reduction may provide an explanation for the impairment of oxygenation during anesthesia on the basis of miliary atelectasis, airway closure, altered inspired intrapulmonary gas distribution, or a combination of these factors. Equally important, the finding of this reduction has provided an impetus to evaluate the heretofore little recognized effect of general anesthesia on the chest wall, which includes all structures that move with respiration, that is, thoracic wall, abdominal wall, and diaphragm.

Bergman was the first to report a decrease in FRC after the induction of general anesthesia,[1] but his report virtually was ignored. Two years later, Déry *et al.*[2] attributed the reduction in FRC to the development of absorption atelectasis, because they failed to demonstrate a reduction in FRC in patients breathing gas mixtures that contained less than 100% oxygen. Although many subsequent studies observed similar reductions in FRC, these studies could not confirm the dependence on the composition of the inspired gas mixture. Nevertheless, the Déry *et al.* article formed the basis for further studies. Currently, it is generally accepted that the FRC is reduced by approximately 18%, or 0.5 l. The decrease occurs soon after the induction of anesthesia, is not progressive with time, is not affected by muscle paralysis, and occurs with many anesthetic agents.

The mechanisms causing the reduction in FRC remain unclear. Possibilities include atelectasis, increase in thoracic blood volume, cephalad displacement of the diaphragm, direct effect on the lung, increased elastic recoil of the lung, decreased elastic recoil of the chest wall, or—particularly in children—loss of glottic narrowing (glottic throttling). Any of these factors could, either singly or in combination, reduce the FRC.

Absorption atelectasis probably can be excluded as a major contributing mechanism. Also, the trapping of

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gas distal to closed airways does not seem to be a major mechanism for the reduction in FRC, because comparative measurements of FRC by nitrogen clearance (which measures only gas in contact with the environment through open airways) or by body plethysmography (which measures all thoracic gas, including gas trapped behind closed airways) showed similar results.[3] Similarly, a direct pharmacologic effect of anesthetic agents on the lung is unlikely to be a contributing factor, because FRC is not reduced in sitting subjects after induction of anesthesia[4] when the anesthetic regimen was the same as that used in recumbent subjects.

Changes in elastic recoil of the chest wall may be a major factor.[5] At FRC, the inward recoil of the lung is balanced by the outward recoil of the chest wall. Any imbalance between these recoils will alter FRC. After induction of anesthesia, the outward recoil of the chest wall may be reduced by changes in the muscle tone of the respiratory muscles. Particularly, changes in diaphragmatic tone may contribute because the diaphragm is the major muscle responsible for the maintenance of FRC in recumbent subjects.[6] The loss of tonic electromyographic activity of the diaphragm[7] and its cephalad displacement[8] after induction of anesthesia are consistent with this view. But how anesthesia affects the muscle tone is unclear. The system that controls muscle tone is complex, and our knowledge about the effect of anesthesia on the mechanisms that control muscle tone is incomplete. This area needs much more investigation.

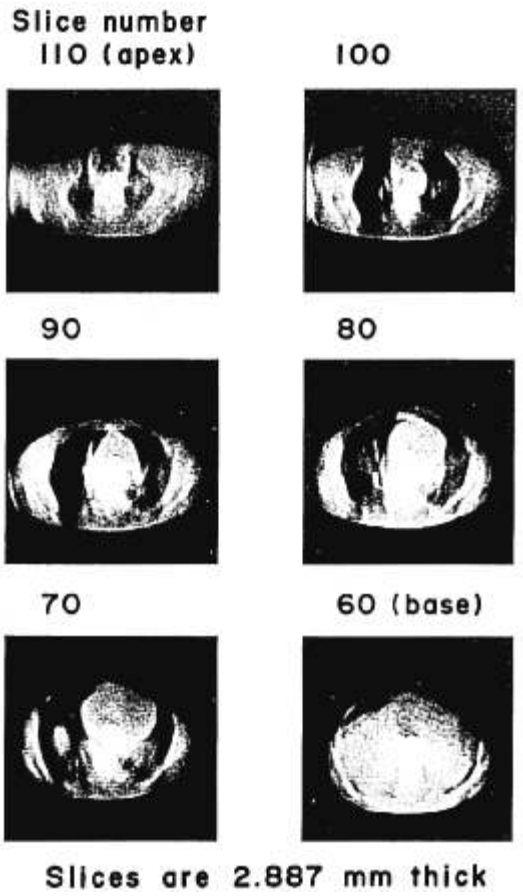

FIG. 1. Six transverse sections of the thorax of an anesthetized–paralyzed volunteer lying supine. Each slice is 2.887 mm thick. Left top slice (slice no. 110) is a section through the apex, and the right bottom slice (slice no. 60) is a section through the base of the thorax. From a total of 128 slices, the entire thorax was reconstructed in three dimensions.

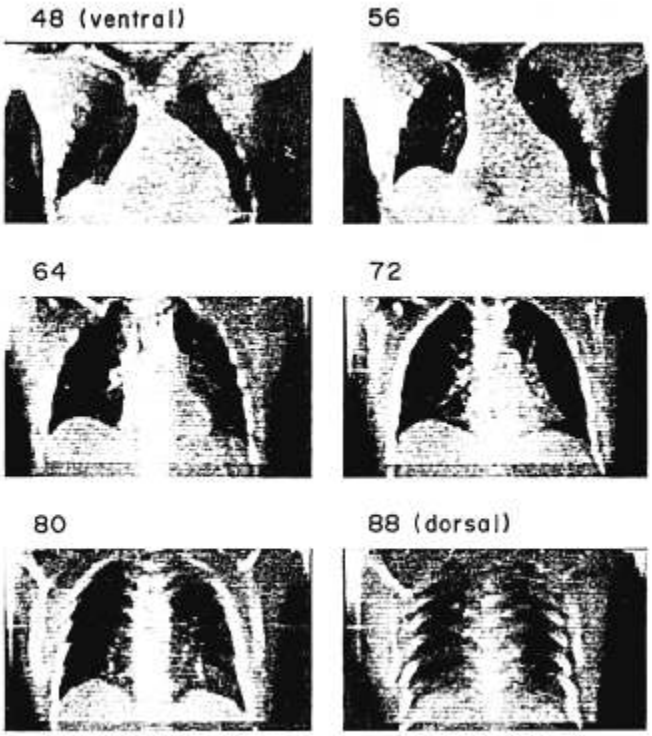

FIG. 2. Six coronal sections of a three-dimensionally reconstructed thorax. The left top slice (slice no. 48) is a section through the ventral part of the thorax, and the right bottom slice (slice no. 88) is a section through the dorsal part of the thorax (behind the heart). From all coronal slices, one can estimate the thoracic (gas plus tissue) volume.

In this issue of ANESTHESIOLOGY, Hedenstierna *et al.*[9] used state-of-the-art methods to measure the changes in thoracoabdominal dimensions and central blood volume associated with the induction of anesthesia. These investigators determined thoracic and abdominal dimensions by computerized tomography at three abdominal and three thoracic levels before and after the induction of anesthesia. The total volume of thorax (gas and tissue) and abdomen were computed from the areas and heights of the six transverse sections by making assumptions about the shape of the thorax and abdomen between the imaged slices. The central blood volume was determined from dye–dilution curves. From these data an interesting balance of gas and blood shifts

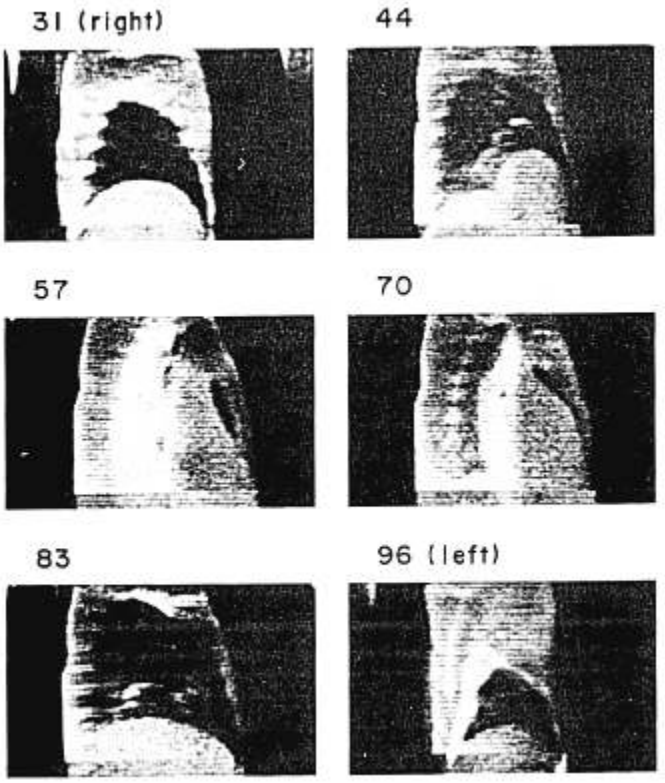

FIG. 3. Six sagittal planes of a three-dimensionally reconstructed thorax. The left top slice (slice no. 31) is a section through the right side of the thorax, and the right bottom slice (slice no. 96) is a section through the left side of the thorax. By comparing sagittal sections between the awake and anesthetized–paralyzed states, one can evaluate unequivocally the effect of anesthesia–paralysis on the regional shape change of the diaphragm.

occurring after induction of anesthesia is provided. According to these calculations, total thoracic volume is reduced (750 ml) by the combined effect of the cephalad shift of the diaphragm (500 ml) and change in thoracic geometry (250 ml). The reduced total thoracic volume is associated with a reduction in gas volume (450 ml) and central blood volume (300 ml). In spite of a small reduction in abdominal volume due to change in geometry (100 ml), the overall abdominal volume is increased (400 ml) by the cephalad shift of the diaphragm (500 ml). The increase in abdominal volume is associated with a shift of blood from the thorax (300 ml) and from the periphery (100 ml) into the abdomen. The data balance but the study is descriptive and, as the authors recognize, the underlying mechanisms for the reduction in FRC remain unclear. What causes the shift of blood volume? Why was the relatively large reduction of thoracic volume not detected by previous investigators using magnetometry?

Many questions regarding the methods also exist. Hedenstierna *et al.* used computerized tomography to determine the effect of general anesthesia on thoracoabdominal dimensions. This method provided more data than were previously available by either magnetometry or mercury-in-rubber strain-gauge techniques. But the data for a complete description of the system still are incomplete; only three thoracic and abdominal transverse sections each were available for computation of thoracic and abdominal volumes. Hence, the geometric data had to be interpolated between the scanned slices. Moreover, the slices were not obtained simultaneously, so that computed answers can be only rough approximations.

Nonetheless, the authors are on the right track because these two limitations can be overcome by high repetition rate cylindric volumetric scanning, a technique applying computed tomography for complete three-dimensional reconstruction of the thorax.[10] This technique allows more detailed description of shape changes of the chest wall, which occur after induction of general anesthesia. Figure 1 shows images of six (selected from 128) 2.887-mm-thick transverse slices of the thorax of an anesthetized–paralyzed subject. The image data from which these slices were computed were obtained simultaneously during a single breath-holding. If the cephalocaudal extent of the thoracic cavity is less than 21.5 cm, this scan requires approximately 0.5 seconds, but if it is larger than 21.5 cm, the subject must be moved in between two 0.5-s scans so that the total scan and move time (*e.g.*, three cardiac cycles) is approximately 3 s. A complete three-dimensional reconstruction of the thorax results so that coronal (fig. 2) and sagittal (fig. 3) sections can be generated readily. By subtracting the lung gas volume, which can be measured independently, one then may estimate the intrathoracic tissue volume. Changes in thoracic tissue volume with induction of anesthesia most likely represent changes in thoracic fluid, presumably blood volume. Using this technique, we found in eight supine volunteers that the average thoracic volume was reduced considerably less (340 ml) than the average gas volume (590 ml), which suggests, in contrast to the data of Hedenstierna *et al.*, an increase in thoracic fluid volume (250 ml) with induction of anesthesia.

In spite of its obvious shortcomings, the article of Hedenstierna *et al.* is an important step toward the understanding of the underlying mechanisms for the impaired pulmonary gas exchange during anesthesia. It will be interesting to follow this fascinating development.

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