

The Assessment of Diaphragmatic Contractility

THE ARTICLE by Dureuil *et al.*¹ in this issue of ANESTHESIOLOGY suggests that diaphragmatic dysfunction following upper abdominal surgery partially may be reversed by methyl xanthines. The evidence, as reviewed by the authors, suggests that diaphragmatic contraction may be impaired reflexly by upper abdominal manipulations. The influence of methyl xanthines could be to reverse the reflex, to increase the central drive producing diaphragmatic contraction, and to improve diaphragmatic contractility.

The authors used the index of $\Delta P_{ga}/\Delta P_{di}$ as an index of the diaphragm's contribution to the respiratory pressure swings, where ΔP_{ga} is the amplitude of the gastric pressure swings and ΔP_{di} is the amplitude of ΔP_{ga} minus the amplitude of pleural pressure swings, ΔP_{pl} . The authors correctly conclude that the use of this index does not distinguish between the three possibilities outlined above, namely between the reversal of a reflex, increased central diaphragmatic drive, and improved diaphragmatic contractility. The purpose of this editorial is to suggest ways by which diaphragmatic contractility can be assessed and thus distinguish between the various possibilities.

Even today there is no agreement on the best way to assess myocardial contractility so that it is highly unlikely that, in these early days, a method to assess diaphragmatic contractility is available that will satisfy everyone. However, I believe that the state-of-the-art is sufficiently advanced that one can attempt such an assessment. Before suggesting how to do so, a critical assessment of the $\Delta P_{ga}/\Delta P_{di}$ index is in order.

I do not wish to appear too critical, as the clinical assessment of diaphragmatic function is still in its infancy. Nevertheless, the measurement of P_{di} as the difference in the amplitude of P_{ga} minus the amplitude of P_{pl} is open to serious error if the peaks do not occur simultaneously. In a patient with airway obstruction, for example, the swing in P_{pl} will be closely in phase with flow. The swing in P_{ga} , however, will be closely in phase with volume. The amplitudes of P_{pl} and P_{ga} will be out of phase with each other, and the difference between the two will give an incorrect measure of the amplitude of P_{di} . Measurements of P_{di} must be made on simultaneous differences between P_{pl} and P_{ga} .

If simultaneous measurements are made, then the

ratio $\Delta P_{ga}/\Delta P_{di}$ can be used to make a qualitative assessment of the diaphragm to the respiratory pressure swings, but additional problems exist. Even mild abdominal muscle contraction can increase the magnitude of ΔP_{ga} . Assessment of the diaphragmatic contribution to the respiratory pressure swings must take into account, not only the amplitude of P_{ga} , but also its absolute values, which will be altered if the abdominal muscles contract. Furthermore, values of P_{ga} during expiration probably should be disregarded and $\Delta P_{ga}/\Delta P_{di}$ measured only during inspiration. This measurement, however, is not a measurement of diaphragmatic contractility, if one defines contractility as the transdiaphragmatic pressure produced in response to a given degree of excitation. The latter can be measured as the relationship between the integrated electrical activity of the diaphragm, Edi as assessed by rectifying and integrating the diaphragmatic EMG (best measured by an esophageal electrode), and the resulting P_{di} . This gives the ratio P_{di}/Edi . An increase in this ratio signifies improved contractility, while a decrease indicates impaired contractility. Note that this index of diaphragmatic contractility contains the force-length and force-velocity characteristics of the diaphragm. According to this definition, contractility can change if there are changes in diaphragmatic length and/or the velocity of diaphragmatic contraction or in the inotropic state of the muscle (defined as P_{di}/Edi when length and velocity remain constant or otherwise have been taken into account). Once a change in contractility has been observed, then the investigator is faced with the problem of deciding whether the change results from change in length, velocity or inotropy—a task that may not be easy.

Nevertheless, such a measurement of contractility would be useful in distinguishing between the three mechanisms proposed to explain the action of aminophylline.

If aminophylline had acted to abolish a reflex, both P_{di} and Edi would have increased, but the relationship between the two would be along the same relationship pertaining before aminophylline administration. A similar finding would be obtained if aminophylline acted to increase the central drive to the diaphragm, increasing phrenic nerve activity and thereby both Edi and P_{di} . However, an increase in central drive would only increase $\Delta P_{ga}/\Delta P_{di}$ if the action were selectively on the drive of the diaphragm without a proportionate increase in the drive to the other inspiratory muscles. Although

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 $\Delta P_{ga}/\Delta P_{di}$.

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 inotropy 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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Anesthesiology
62:230-233, 1985

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