

Editorial Views

Anesthesia and Atelectasis

DOES INHALATION ANESTHESIA cause atelectasis? Prior to the paper by Colgan and Whang in this issue many people would have agreed that it does. Unfortunately, most of the work on this problem has been carried out in dogs in which controlled (nonanesthetized studies) were impossible. Thus, whatever happened could be interpreted as due to the anesthetic. Drinker and Hardenburgh,¹ 20 years ago, noted atelectasis in dogs following prolonged general anesthesia. Mead and Collier² noted a rapid and large fall in compliance of the lungs of anesthetized dogs with only small changes in lung volume. We³ have noted a rapid increase in venous admixture in anesthetized dogs breathing oxygen once positive pressure breathing has been discontinued.

Colgan and Whang, in this issue of the Journal, found that progressive atelectasis was not a problem during general anesthesia. A close look at their data shows that considerable atelectasis had occurred prior to the initial measurements. Their data indicate that compliance of the lung in supine humans breathing 100 per cent oxygen was 0.109 l./cm. H₂O, FRC was 2.26 l., and PaO₂ was 414 mm. Hg. Ferris, Mead and Frank⁴ found values for supine compliance of 0.25 l./cm. H₂O and average FRC's of 3.5 l. in healthy male subjects. Nunn and Bergmann⁵ found the PaO₂ to be greater than 600 mm. Hg in supine patients before anesthesia. It seems fair to assume, then, that there was a significant fall in compliance, FRC and PaO₂ in the preoperative "controls" of Colgan and Whang.

The values for PaO₂ before anesthesia were similar to those found by Bendixen⁶ and Nunn⁷ after anesthesia. PaO₂ rose from 400 to 553 mm. Hg with inflation in the group studied by Bendixen.

The findings raise the question why the changes should occur in supine patients who have received only 100 mg. pentobarbital and 0.6 mg. atropine for preanesthetic medication. Mead and Collier found that the fall in compliance in spontaneously-breathing anesthetized dogs occurred most rapidly in the first 15 minutes and leveled off at about 60 per cent of control in one hour. The lack of progressive changes in the present study, therefore, probably was a manifestation of the fact that major atelectasis had occurred prior to the anesthesia. That anesthesia did not cause further changes suggests that the alterations occur rapidly, may not be related to anesthesia, and do not become progressive after one hour.

If general anesthesia does not cause atelectasis, what does? As Mead and Collier have shown, the volume history of the lung is critical. Deep periodic inflations reduce atelectasis but in a few minutes significant atelectasis recurs. This has been attributed to changes in surface elastic phenomena, so-called "surface aging," easily observed in a surface balance using saline extracts of whole lung. When the barrier that expands and contracts the surface area of the extract is no longer moved, surface tension of the film tends toward a median value—rising from a minimum or falling from a maximum value. Clements⁸ has shown that

this occurs over several minutes, approximately the time course seen in Mead and Collier's study. To date, anesthetics have not been shown to alter the normal surface phenomenon or the surface activity of extracts of lung biopsies.⁷ It appears that atelectasis is the natural consequence of lack of periodic inflation of the lungs, and that anesthesia plays a role only in reducing the frequency of periodic inflations.

The best measure of atelectasis is probably the fall in lung compliance, rather than FRC. However, the Pa_{O_2} during breathing of pure O_2 is considerably easier to measure. The fall in Pa_{O_2} will usually underestimate the amount of atelectasis because of an increased resistance to blood flow through collapsed lung. We⁸ have found the Pa_{O_2} to be over 400 mm. Hg with one lung collapsed in dogs breathing quietly. If increased respiratory depth occurs (increased transpulmonary pressure) without decreasing the atelectasis, the Pa_{O_2} may fall to very low values, indicating an increased venous admixture. Under the latter circumstances, the venous admixture can overestimate the degree of atelectasis. A simple method to ascertain that there is no atelectasis is to apply positive pressure with 100 per cent oxygen until the Pa_{O_2} is ≥ 600 mm. Hg. Under these circumstances the effect of an anesthetic agent on the rate of as well as degree of occurrence of atelectasis may be determinable.

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