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Editorial Views

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Anesthesia and the Lung

IT IS NOW well established that certain changes in pulmonary function are a normal consequence of anesthesia in healthy patients. To a large extent, these changes are independent of the anesthetic technique and the drugs that are used, but do tend to be greater in older patients. The more important pulmonary consequences of anesthesia may or may not be related to the prevailing type of ventilation. During anesthesia with spontaneous breathing, the principal changes are those of a decrease in the thoracic component of breathing and a decreased sensitivity of breathing to increased P_{CO_2} or decreased P_{O_2}. The principal pulmonary changes during anesthesia with either spontaneous or artificial breathing include an increase in the alveolar component of the respiratory dead space; an increase in the alveolar–arterial P_{O_2} difference, quantified as increased pulmonary venous admixture (Riley-Rahn model); and a decrease in functional residual capacity (FRC).

Some of these changes are secondary to others. It appears, for example, that the decreased sensitivity to increased P_{CO_2} is related primarily to a loss of the thoracic respiratory excursion,[1,2] and the increased alveolar–arterial P_{O_2} difference has been shown to correlate with the decrease in FRC.[3] However, in most cases the cause of the primary phenomenon remains obscure.

The increase in the alveolar component of the respiratory dead space[4] and the pulmonary venous admixture[5,6] have hitherto been expressed in terms of the Riley-Rahn three-compartment model, as if functioning alveoli could be divided into three groups—unventilated, unperfused, and ideal. It was never suggested that this was other than a convenient mathematical model that yielded indices of pulmonary function. These indices did, in fact, fulfill the useful purpose of providing a basis for the calculation of ventilatory requirement and also the inspired oxygen concentration necessary to avoid hypoxemia. The indices, however, told us nothing of the nature of the physiologic defect that developed during anesthesia. Thus, it was always clear that the measured alveolar dead space included a contribution from alveoli that were relatively overventilated (*i.e.*, with a pulmonary ventilation–blood flow [$\dot{V}/\dot{Q}$] ratio that was high but not infinity). Similarly measured venous admixture contained a contribution from alveoli that were relatively underventilated (*i.e.*, with a $\dot{V}/\dot{Q}$ ratio that was low, but not zero). However, studies of venous admixture at different levels of inspired oxygen concentration, and measurement of $\dot{V}/\dot{Q}$ in horizontal slices of the lung, were not sufficiently discriminating to give a clear picture of the precise quantitative changes in the distribution of $\dot{V}/\dot{Q}$ ratios resulting from anesthesia.

The development of the multiple-inert-gas technique[7] has, in effect, expanded the three-compartment model to one of some 50 compartments of differing $\dot{V}/\dot{Q}$ ratios as well as zero and infinity. This permits much greater precision in the definition of the disorder of distribution, although it still does not reveal the nature of the physiologic defect. Furthermore, it should be noted that the plots of distribution of $\dot{V}/\dot{Q}$ ratios are not unique solutions to the primary data of retention and excretion of the six inert gases. Nevertheless, none of these criticisms detracts from the important new information yielded by the technique.

Its application to the anesthetized patient required major technical difficulties to be overcome,[8] and in this issue Dueck and his co-workers[9] describe the distribution of $\dot{V}/\dot{Q}$ ratios in ten subjects before and during anesthesia, which included use of neuromuscular blocking drugs. At the outset it should be noted

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that the subjects were elderly smokers who had abnormal pulmonary function. Dueck's data cannot therefore be extrapolated to young healthy subjects; this population will require separate study. However, Dueck has clearly shown major changes that develop in the regions of zero and low $\dot{V}/\dot{Q}$ after induction of anesthesia. His patients may be divided into three groups: those in whom shunts developed (zero $\dot{V}/\dot{Q}$), those in whom areas of low (but not zero) $\dot{V}/\dot{Q}$ developed, and those in whom both of these abnormalities developed. However, development of a precise mathematical relationship between the alveolar–arterial P_{O_2} difference and these changes was not a practical possibility. The changes were greater in those with the worst pre-existing pulmonary function. There was no evidence that duration of anesthesia or substitution of nitrogen for nitrous oxide had any major effect on distribution.

All three patterns of change of distribution might be attributed to decreased FRC which, for technical reasons, was not measured in Dueck's study. A decrease in FRC below closing capacity would inevitably result in diminished or absent ventilation of some groups of alveoli. Although there is good evidence that FRC is decreased during anesthesia, there are recent data showing that closing capacity is also decreased.[10] Complete elucidation of the significance of the change in FRC will therefore require simultaneous measurements of FRC, closing capacity, and gas exchange in patients of different age groups before and after the induction of anesthesia.

At the other end of the $\dot{V}/\dot{Q}$ scale the position is less clear. Dueck reports values for dead space defined as the ventilation of alveoli with $\dot{V}/\dot{Q}$ ratios > 100, together with anatomic and apparatus dead space (the latter being unchanged). There was no evidence of any major change in dead space, and it may be inferred that, since the anatomic dead space was decreased by tracheal intubation, the alveolar component must have increased. However, Dueck does not report the existence of a "shelf" of ventilation to units of high $\dot{V}/\dot{Q}$ analogous to the "shelf" of perfusion to units of low $\dot{V}/\dot{Q}$, as seen particularly in Patient 8 in the present study. The cause of the inferred increase in alveolar dead space remains a mystery.

The paper from Dueck's group should stimulate much further work in the field of gas exchange during anesthesia. It is obviously important that healthy patients of different ages be studied. The changes in distribution should also be related to FRC, closing capacity, and the alveolar–arterial P_{O_2} difference measured at the same time. Finally, we can still only speculate on the nature of the primary physiologic change or changes that result in the altered distribution that has been reported.

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