The Effect of Inhalation Anaesthetics on Pulmonary Ventilation and Perfusion

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During the last 2 decades, a number of investigators have demonstrated that general anaesthesia tends to impair pulmonary gas exchange [15, 16, 17]. Unless this tendency is counteracted by enhancing the oxygen concentration of the anaesthetic gas mixture, arterial hypoxaemia may develop. Although this condition is well documented, the pathophysiological mechanisms involved have been unsettled until the last few years.

General anaesthesia has been shown to give rise to changes in pulmonary ventilation mechanics. Laws [13] found a fall in functional residual capacity upon induction of anaesthesia. At the beginning of the last decade, investigators [7, 10, 11, 12] demonstrated that the reduction in functional residual capacity in certain subjects contributes to the closure of peripheral dependent airways. On the basis of direct roentgenographic observations, Froese and Bryan [9] suggested that changes in ventilation mechanisms of this type are mainly due to a cephalad displacement of the diaphragm during anaesthesia. They further found that most of the lung movements during inflation occur in the non-dependent regions. The high blood solubility of anaesthetic gases in comparison to air facilitates absorption of gas trapped behind closed airways. These changes promote hypoventilation and/or atelectasis, especially in the dependent regions where initial volumes are small. If it should happen that regional perfusion is unchanged or even increased in such hypoventilated or atelectatic lung areas, arterial hypoxaemia will result.

In the awake state vasoconstriction within poorly ventilated regions of the lungs acts to divert local blood flow to better ventilated areas [22]. A possible explanation of the development of arterial hypoxaemia during anaesthesia therefore could be that this beneficial mechanism for redistribution of blood flow is hampered by anaesthetic agents. Already in 1972 and 1973, Sykes and co-workers [18, 19], using different models of isolated dog lungs, published results in support of such an idea.

At the Institute of Physiology, University of Oslo, we started to work on this hypothesis in 1974. Our experimental model was a preparation of isolated rat lungs perfused with blood at constant flow and constant outflow pressure. Pulmonary vasoconstrictor responses were then reflected as increments in inflow pressure — so-called pressor responses. Such responses were elicited by ventilating the lungs for standardized periods with a hypoxic — alternating with a normoxic gas mixture. When hypoxic pressor responses of a slightly increasing or equal magnitude were obtained, the anaesthetic agent to be tested was added either to the ventilation gas or to the blood reservoir. Figure 1 shows eight succeeding pressor responses to ventilation hypoxia. When diethyl ether was added to the ventilation gas at 103 and 135 min respectively from start of perfusion, the responses were almost completely abolished.
In all the experiments with ether, the striking observation was a stepwise reduction of the pressor response with increasing concentration of the anaesthetic (Fig. 2). Such dose-response curves between reduction of response and blood concentration of inhalation anaesthetic were additionally obtained with halothane, methoxyflurane, and enflurane [2, 4]. Confirmatory evidence of a reducing effect of diethyl ether has also been reported by Sykes and co-workers [20], on the basis of observations on dogs exposed to unilateral hypoxia. In contrast, when the effects of halothane [21] and methoxyflurane [14] were evaluated by means of the same model, no change in response could be found.