PULMONARY RESISTANCE DURING HALOTHANE ANESTHESIA IS NOT DETERMINED ONLY BY AIRWAY CALIBER

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Introduction. Most studies of the effect of anesthetics on airway smooth muscle have used pulmonary resistance as an index of airway caliber. However, pulmonary resistance (Rl) is the sum of airway resistance (Raw), which changes with airway caliber, and a tissue resistance (Rtl), which depends on the pressure-volume hysteresis of the lung tissue. Therefore, reductions in pulmonary resistance during administration of volatile anesthetics may be due to a reduction in airway resistance, tissue resistance, or both. To separate the effects of halothane on airway caliber from its possible effects on tissue pressure-volume hysteresis in the unstimulated lung and during bronchoconstriction, we measured both Raw and Rtl before (unstimulated) and during vagus nerve stimulation (bronchoconstriction) in 10 vagotomized dogs before and during the administration of halothane.

Methods. Ten mongrel dogs were anesthetized with chloralose (60 mg/kg) and urethane (600 mg/kg i.v.), paralyzed with vecuronium (0.5 mg/kg i.v.), and the trachea intubated. The lungs were mechanically ventilated with an Inspired oxygen concentration of 30%, a tidal volume of 15 ml/kg, an end-expiratory pressure of 3 cm H2O, and a breathing frequency of 15/min. Sternotomy was performed and the rib cage widely retracted to expose the lungs. Tracheal pressure was measured at the distal end of the endotracheal tube, and gas flow was measured with a pneumotachograph. Partitioning of pulmonary resistance into airway and tissue components requires measurement of alveolar pressure. Small capsules (1,2) were glued to the lung surface and holes punched in the underlying pleura so that pressure in the capsules equaled the alveolar pressure beneath the capsule. Stimulating electrodes were applied to the distal ends of both cut cervical vagus nerves, and the dog was placed in a body plethysmograph that measured changes in lung volume. Measurements were made before and during 30 seconds of vagus nerve stimulation both before halothane administration and with stable end-tidal concentrations of halothane corresponding to 0.1, 0.2, 0.4, 1.0, and 2.0 MAC, applied in random order. Resistances were calculated by multiple linear regression, assuming that the lung mechanics followed a linear equation of motion; this method has been validated in our laboratory (1). Single paired comparisons were made by paired t test, and multiple comparisons were made by repeated-measures analysis of variance.

Results. Before vagus nerve stimulation, Rtl comprised 77% of Rl. Stimulation consistently increased Rtl, Raw, and Rtl. The increase in Rtl (1.09±0.49 cm H2O L-1 s) was not significantly different from the increase in Raw (1.21±1.47 cm H2O). Halothane had no effect at any MAC value on the prestimulation value of any resistance (P > 0.05, ANOVA). Halothane attenuated the response of both Rtl and Raw to vagus nerve stimulation in a dose-dependent fashion. When the response to vagus nerve stimulation was expressed as a percent of the control response (before halothane administration), the attenuation of the Raw response was slightly greater than the attenuation of the Rtl response, while attenuation of the Rtl response was somewhat less than that of the Rtl response at each halothane dose (Figure). However, the attenuation of the Rtl and Raw responses was not significantly different at any halothane dose (P > 0.07 at each dose).

Discussion. Tissue resistance, an index of the pressure-volume hysteresis of the lung tissue, was a significant component of pulmonary resistance in the unstimulated airway and during vagus nerve stimulation (2). Halothane attenuated increases in Rtl during stimulation by reducing the responses of both Rtl and Raw. During stimulation, nearly half of the decreases in Rtl responses at all MAC levels were caused by reductions in Rtl. Thus, a significant part of changes in Rtl caused by halothane during vagus nerve stimulation was not caused by changes in airway caliber, and absolute changes in Rtl overestimated the effect of halothane on airway caliber. However, the reduction in the Raw response, expressed as a percent of the control response, was nearly identical to the reduction in the Rtl response caused by halothane, so that qualitative estimates of the effect of halothane on airway caliber could still be made from the dose-response relationship for Rtl (Figure). We conclude that changes in pulmonary resistance during anesthesia are not only caused by changes in airway caliber, as previously assumed, but also reflect an effect of halothane on the pressure-volume hysteresis of the lung tissue. Supported in part by NIH grant HL-21584.

References.

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