TITLE: CONTINUOUS ENDOBRONCHIAL INSUFFLATION DURING MEDIAN STERNOTOMY

AUTHORS: R. Watson, R. Szarko, A. Sequeira*, CF Mackenzie, L. Mark. Dept. of Anesthesiology and *Thoracic Surgery, Univ. of Maryland, Baltimore, MD 21201.

During internal mammary artery harvest (IMAH) for coronary bypass, lung inflation with conventional mechanical ventilation (CMV) impairs surgical access. Continuous endobronchial insufflation of O_2 (EIO) could maintain stationary lungs and adequate ventilation[1] without obscuring the IMA. After consent and Review Board approval the efficacy of EIO was assessed after median sternotomy and opening of the pericardium (MSOP) in 7 males undergoing IMAH. Methods Patients (mean ± SE) 87 ± 18.9 kg, age 64 ± 5.5 yrs were anesthetized using opiates and paralyzed. Mean arterial (MAP) and pulmonary artery (PA) pressures, cardiac output (Qt), mixed venous saturation (SvO_2) and arterial blood gases (ABG) were measured after MSOP on CMV. CMV was disconnected. EIO then was given through two 2 mm internal diameter (ID) catheters confirmed to be positioned 3 cm into each mainstem bronchus by bronchoscopy. Total flow was 45 L/min humidified 100% O_2 (Fisher-Pakel) half going to each lung. Measurements were repeated serially at 5 min intervals for 20-40 min of EIO and compared using Bonferroni t-tests. Results In all patients surgeon judged access to IMA was improved. For the same surgeon time was reduced for IMAH from CMV average 23 min to 17 min with EI. All patients received at least 20 min EIO. $PaCO_2$ (mmHg) at time 0 on CMV of 32 ±1.5 (mean ±SE) rose ($p<0.05$) after 15 and 20 min EIO when $paCO_2$ was 50 ± 4.7 and 51 ± 4.4 respectively. pH decreased at all time points compared to time 0, but Qt, MAP and PA pressure were unchanged. PaO_2 (mmHg) was 373 ± 56.4 after 20 min. Two patients of 58 and 95 kg had $paCO_2$ less than 38 mmHg throughout 20 min EIO. Discussion EIO at 45 L/min produced gas exchange with adequate oxygenation in all patients and an average rise in $paCO_2$ of 5 mmHg between 5 and 20 min (0.3 mmHg/min) and a 19 mmHg rise from $paCO_2$ values on CMV. However there were large individual variations in oxygenation and CO_2 removal which were not in all instances related to previous lung dysfunction or body weight. None of these variations were clinically unacceptable because paO_2 was never below 129 and $paCO_2$ never above 65 mmHg. The mechanism for the variations may be related to atelectasis or catheter position in the bronchi, airway branching and turbulence or differences in collateral airways. Cardiogenic oscillations would not have contributed to these differences because of MSOP[2]. The EIO benefits included diminished surgical time and better access. No adverse effects to the airway or complications were noted during or after surgery. Conclusions EIO with 45 L/min O_2 1) ventilates effectively with stationary lungs after MSOP; 2) produces variations in gas exchange independent of body weight; 3) improves access and decreases time for IMAH. References 1) Babinski et al. Continuous flow ventilation during thoracotomy. Anesthesiol 65:399, 1985. 2) Cybulsky et al. Contribution of cardiac oscillations to gas exchange in constant flow ventilation. J Appl Physiol 63:564, 1987.

A1193

TITLE: EFFECT OF HALOTHANE ON GUINEA PIG AND HUMAN BRONCHI

AUTHORS: L. Zhou, M.D., J. Vann, Ph.D., J.A. Will, Ph.D.,D.V.M.

AFFILIATION: Anes. Dept., University of Wisconsin, Madison, WI 53792

We have studied the effect of halothane on human and guinea pig bronchi. Using both epithelium-intact and epithelium-removed groups, accumulating doses of halothane produced contraction of guinea pig hilar bronchi (Fig.1). There were no significant differences between the intact epithelium group and the epithelium-removed groups. The mechanism of the contractile response of guinea pigs hilar bronchi to halothane may not be epithelium-dependent. In vitro preparations of human bronchi of 2-4mm diameter were used to determine the response of human smooth muscle to halothane. In contrast to guinea pigs, human bronchi relaxed in response to halothane (Fig.2).

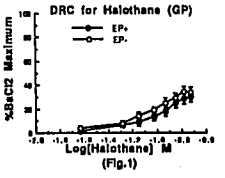
Both guinea pig bronchi (Fig.3) and human bronchi (Fig.4) pretreated with carbachol relaxed in response to halothane.

Indomethacin (5x10-6M) significantly reduced the magnitude of the contractions evoked by halothane in guinea pigs (Fig.5). In the presence of indomethacin, human airways contracted in response to halothane (Fig.6)

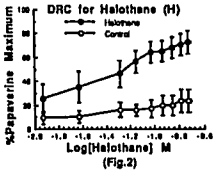
These results suggest that halothane induces the release of cyclooxygenase metabolites which mediate smooth muscle response. The mechanism of airway smooth muscle response to prostaglandins in humans is different than that in guinea pigs. We have previously demonstrated differences in leukotriene receptors between human and guinea pig airways (1). The different effects of cyclooxygenase products of human versus guinea pig smooth muscle to halothane may be due to different receptor systems or different effect of halothane on cyclooxygenase metabolism.

Reference

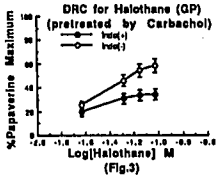
(1). Ann N Y Acad Sci [5NM] 1988; 524: P 181-186



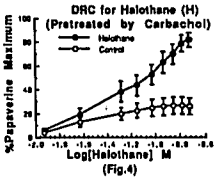
(Fig.1)



(Fig.2)



(Fig.3)



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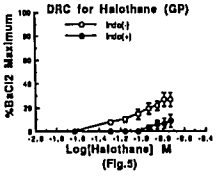
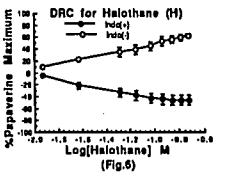


(Fig.5)



(Fig.6)