Title: HEMODYNAMIC EFFECTS OF ONE LUNG ANESTHESIA DURING THORACIC SURGERY

Authors: D.M. Thys, M.D., E. Cohen, M.D., J.B. Eisenkraft, M.D., J.A. Kaplan, M.D.

Affiliation: Department of Anesthesiology, The Mount Sinai School of Medicine, One Gustave L. Levy Place, New York, New York 10029

Introduction: While the effects of one-lung anesthesia on respiratory function have been extensively reported, little information is available on the cardiovascular consequences of the lateral position and of one-lung ventilation.[1] The purpose of this study was to investigate the hemodynamic changes occurring during thoracic anesthesia with N_2O and isoflurane.

Method: After institutional approval and informed consent had been obtained, 8 adult patients (ASA 1-2) scheduled for elective left thoracotomy were admitted to the study. They were premedicated with morphine 0.1 mg/kg, lorazepam 0.05 mg/kg, and atropine 0.006 mg/kg. Prior to induction of anesthesia the left radial artery was cannulated and a pulmonary artery catheter was passed through the right internal jugular vein. Anesthesia was induced with thiamylal sodium 3.5 mg/kg, relaxation produced by pancuronium 0.15 mg/kg, and anesthesia was maintained with isoflurane 1.0-1.5% (inspired concentration) in N_2O/O_2 (50/50%) delivered via a Robertshaw double lumen tube. Hemodynamic measurements were obtained in the supine position, in the closed-chest lateral position, and following the opening of the chest and collapse of the non-dependent lung. In order to achieve a steady-state, a 15 minute stabilization period followed each maneuver. All values are expressed as means plus or minus standard deviations and paired Student's t-test was used for analysis of statistical significance.

Results: The values for each of the reported variables are shown in the Table. The change in position from supine to right lateral decubitus resulted in a significant reduction in central venous pressure (CVP) and pulmonary capillary wedge pressure (PCWP). With collapse of the upper lung, there was a significant increase in cardiac index (CI) and heart rate (HR), and significant decrease in mean arterial pressure (MAP) and systemic vascular resistance (SVR), as compared to the lateral closed chest situation. One lung ventilation caused a highly significant reduction in PaO_2.

Discussion: This study demonstrates that during one lung anesthesia with isoflurane and N_2O/O_2 (50/50%) the cardiac index increases significantly, as a result of a marked increase in heart rate and a modest rise in stroke index (SI). Aalto-Setala et al[2] also reported an increase in heart rate following collapse of the upper lung during halothane anesthesia. However, in their study the rate increase was accompanied by a modest drop in stroke volume, resulting in a non-significant rise in cardiac output. We hypothesize that the profound reduction in PaO_2 resulting from collapse of the non-dependent lung causes a compensatory increase in cardiac output to maintain oxygen delivery and that this response is not blocked by N_2O-isoflurane anesthesia. Attenuation of this response in the study quoted[2] may be due to the myocardial depressant effect of halothane used as the anesthetic in that study.

References:

1) Kaplan JA. Thoracic Anesthesia, New York Churchill Livingstone, p 230-238, 1983.

2) Aalto-Setala M, Heinonen J, Salorinne Y. Cardiorespiratory function during thoracic anesthesia. Acta Anaesth. Scand. 19:287-295, 1975.

Table

	SUPINE	LATERAL CLOSED CHEST	ONE-LUNG LATERAL OPEN CHEST
Heart Rate Beats/min	70 ±15	64 ± 8	79** ±18
MAP mmHg	80 ±13	80 ±14	71* ± 9
Mean Pulm. Art. Pres. mmHg	21 ± 7	19 ± 5	20 ± 3
CVP mmHg	11 ± 4	7* ± 2	7 ± 3
PCWP mmHg	14 ± 5	10* ± 3	11 ± 3
CI $L/min/m^2$	2.04 ±0.44	2.07 ± 0.34	2.73* ±0.62
SI $ml/beat/m^2$	30 ±10	32 ± 5	36 ±11
SVR dynes $sec\ cm^{-5}$	1579 ±427	1678 ±571	1119* ±335
PVR dynes $sec\ cm^{-5}$	179 ± 78	193 ±90	154 ± 59
PaO_2 mmHg	241 ± 74	175 ±63	79** ± 20

*p<0.05 **p<0.01