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Title : ACCENTUATION OF SHUNT WITH NITROUS OXIDE DURING INHALATION ANESTHESIA IN SHEEP

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The use of nitrous oxide during anesthesia may cause or enhance intrapulmonary shunt development. This can occur through physical mechanisms such as chest wall relaxation with lung volume reduction and airway closure, regional re-distribution of lung volume and ventilation, or even through absorption atelectasis. Alternatively, nitrous oxide may act in a pharmacologic manner to reduce pulmonary vasoconstriction in hypoxic lung regions. We therefore wished to test the hypothesis that nitrous oxide indeed does enhance shunt development. We chose an animal model which enabled us to study the same subject repeatedly for awake control and anesthetized conditions, i.e. to control for within-subject as well as between-subject variation.

Methods: Eleven sheep age 1.5 to 2.0 years, 35 to 45 kg weight range, were surgically prepared with chronic tracheostomy and carotid artery exteriorization. Awake control studies of distribution of ventilation with respect to perfusion (V_A/Q, tracer inert gas elimination), arterial and mixed venous blood gas tension, and lung volume (helium dilution FRC) were obtained during 30% oxygen in air breathing in the lateral position. Anesthesia was induced and maintained with halothane 0.96 to 1.68% end tidal and either balance nitrous oxide (10 sheep) or nitrogen (9 sheep) concentration. Both combinations were studied in 7 sheep. From 1 to 5 anesthetic administrations were studied in each sheep (always on a separate day), with a mean ± SD 2.5 ± 1.3 studies per sheep for N_2O and 2.7 ± 1.7 times per sheep for nitrogen (halothane) studies. Mechanical ventilation was provided during anesthesia, with tidal volume 10 $ml \cdot kg^{-1}$ and minute volume equal to awake. Oxygen consumption rate was estimated from oxygen content (via PO_2, pH, p50, Hb, temp) difference in mixed venous and arterial blood and from cardiac output (inert gas data, Fick method). Arterial halothane concentration was measured by gas chromatography.

Results: Awake control mean ± SD shunt values were 2.1 ± 1.9% and 2.5 ± 3.4% shunt ($p>0.1$) for nitrogen- and N2O-halothane studies, respectively. Shunt increased to 4.3 ± 3.2% during nitrogen-halothane and 6.7 ± 4.1% during N_2O-halothane anesthesia ($p<0.05$, see Table 1). Arterial PO_2 values did not reflect these differences. This may well be explained by the finding that the use of N_2O was associated with significantly higher mixed venous PO_2 values (mean awake values were 52.8 ± 4.7 and 52.3 ± 5.3 torr for the two groups). In addition there was a slightly higher mean cardiac output and lower estimated oxygen consumption rate compared to nitrogen-halothane conditions. Arterial halothane partial pressure ranged from 4.0 to 12.3 (mean 8.8 ± 1.6) torr during nitrogen, and from 4.3 to 10.9 (mean 7.5 ± 1.4) torr during N_2O studies. There was no significant correlation of shunt with arterial halothane partial pressure ($r = -0.13$, $r = 0.04$) or with cardiac output ($r = 0.18$, $r = -0.25$) during anesthesia in either the nitrogen or N_2O group, respectively.

Table 1.

	Shunt Increase % Q_T	Arterial PO_2 torr F_IO_2=0.3	Venous PO_2 torr	Cardiac Output $L \cdot min^{-1}$	Oxygen Consumption Rate $ml \cdot min^{-1}$
N_2-Halothane (n=24)	2.1±3.2	126.4 ±15.3	54.5 ±7.1	4.8±1.3	121.6 ±38.6
N_2O-Halothane (n=24)	4.7±3.2	130.5 ±16.1	63.5 ±7.5	5.6±1.6	112.4 ±30.1
ANOV	p<0.02	NS	p<0.001	NS	NS

Legend: Mean ± SD values for the increase in intrapulmonary shunt (% of cardiac output), the arterial and mixed venous (pulmonary artery) oxygen tension, cardiac output, and the oxygen consumption rate during nitrogen-halothane and nitrous oxide-halothane anesthesia studies are presented. Statistical comparison of the two groups was provided with analysis of variance, ANOV significance level $p<0.05$.

Discussion: The findings in this investigation demonstrated a 2-fold difference in the amount of shunt developed with the use of nitrous oxide instead of nitrogen during halothane anesthesia. This difference did not appear to be due to differences in anesthetic depth or cardiac output, since there was no significant correlation of shunt with these variables. The use of tracer inert gas elimination analysis enabled us to demonstrate this difference in spite of confounding variables such as changes in mixed venous PO_2, cardiac output and oxygen consumption rate. These uncontrolled variables made arterial oxygen tension an unreliable index of pulmonary gas exchange impairment.