Title : ANALYSIS OF VENTILATORY DEPRESSION BY ENFLURANE

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Introduction. The present study was designed to more clearly define the mechanism of ventilatory depression during enflurane anesthesia by measuring tracheal pressure generated during airway occlusion (P^0) at FRC (i.e. the mechanical transform of neuromuscular ventilatory drive[1]), the duration of inspiration, and minute ventilation.

Methods. Ten perfectly healthy, consenting adults ($FEV_{1.0}$ > 85% predicted) scheduled for peripheral operations were included in the study approved by the hospital Ethics Committee. Anesthetic technique: phenothiazine premedication, thiopentone induction, intubation, then enflurane in N_2O: O_2 in a non-rebreathing system. After stabilization for twenty minutes at as many vaporizer settings as possible, we measured: VE, f, $PaCO_2$, P^0 and T_i; and derived: mean inspiratory flow rate (VT/T_i), impedance (P^0 0.5"/VT/T_i) and elastance (P^0 max./VT).

Results. In all subjects ventilation was significantly less at the higher inspired concentrations without change in frequency (Table I). The features of P^0 tracings were: a progressive reduction in amplitude and slope, and a clear cut reduction in the time-tension index. Figure I is a representative tracing (with the expected value at 0.1 sec. in awake subjects rebreathing CO_2 marked by "x"). Mean inspiratory flow rates were invariably less at deeper levels due to the combined effects of reduced VT and shortened T_i. Impedance to flow was unchanged due to the proportionate changes in P^0 and VT/T_i. But, pulmonary elastance (stiffness) increased at deeper levels (Figure 2).

Discussion. Hypoventilation is due to a reduction in the mechanical transform of neuromuscular output coupled with a shorter inspiratory time (i.e. reduced flow rate). Reduced P^0 is due to central depression and the dose-related, curare-like effect of enflurane on muscles, which is not reversed by neostigmine[2]. Differentiation, therefore, is impossible. But the progressive reduction in P^0 slope and the unchanged frequency suggest that the changes in P^0 are largely muscular. The shorter T_i may be a homeostatic reflex. This relative pump failure, together with lung stiffness, will compound the consequences of anesthesia on pulmonary mechanics[3] and central depression.

References.

1. Milic-Emili, J: Recent advances in the evaluation of respiratory drive. Int. Anes. Clin 15(2):39-58, 1977.
2. Lebowitz MH, Blitt CD, Walts LF. Depression of twitch response to stimulation of the ulnar nerve during Ethrane anesthesia in man. Anesthesiology 33:52-57, 1970.
3. Westbrook PR, Stubbs SE, Sessler AD, et al: Effects of anesthesia and muscle paralysis on respiratory mechanics in normal man. J. Appl. Physiol 34:81-86, 1973.

TABLE I

	INSPIRED ENFLURANE CONCENTRATION (%)			
	1.0	1.5	2	3
Min.Ventilat'n ℓ/m²/min.	3.3 .62	3.57 .56	3.17 .36	2.13 • .24
Frequency bpm	20.5 2.0	24.0 3.2	22.9 1.6	20.9 1.6
$PaCO_2$ torr	36.0 2.0	40.6 4.0	47.2 5.0	51.2 • 3.8
Inspir. Time T_i sec.	.98 .12	.88 .13	.79 .10	.91 .13
VT/T_i ℓ/sec.	.36 .05	.37 .04	.36 .07	.24 • .04
Impedance cm H_2O/ℓ/sec	17.0 4.0	22.0 7.0	24.0 5.0	27.2 4.6

• Significantly different from value at 1% or 1½%.