ANALYSIS OF VENTILATORY DEPRESSION BY ENFLURANE Title

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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^{O}) at FRC (i.e. the mechanical transform of neuro-muscular ventilatory drive), the duration

of inspiration, and minute ventilation.

Methods. Ten perfectly healthy, consenting adults (FEV₁₀>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 No0: 0, in a non-rebreathing system. After stabilization for twenty minutes at as many vaporizer settings as possible, we measured: VE, f, PaCO, PO and T; and derived: mean inspiratory flow rate (VT/Ti), impedance (PO 0.5"/VT/Ti) and elastance (PO max./VT).

Results. In all subjects ventilation

was significantly less at the higher inspired concentrations without change in frequency (Table I). The features of PO tracings were: a progressive reduction in amplitude and slope, and a clear cut reduction in the timetension index. Figure I is a representative tracing (with the expected value at 0.1 sec. in awake subjects rebreathing CO2 marked by "x"). Mean inspiratory flow rates were in-variably less at deeper levels due to the combined effects of reduced VT and shortened T_1 . Impedance to flow was unchanged due to the proportionate changes in P^O and VT/T_1 . 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 Po is due to central depression and the dose-related, curare-like effect of enflurane on muscles, which is not reversed by neostigmine². Differentiation, therefore, is impossible. But the progressive reduction in Po slope and the unchanged frequency suggest that the changes in Po are largely muscular. The shorter T_1 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.

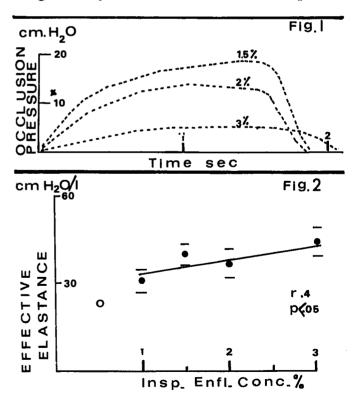
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	TABLE I				
	INSPIRED EMPLURANE CONCENTRATION (%)				
	1.0	1.5	2	3	
Min.Ventilatn L/m²/min. Frequency bpm	3.3 .6? 20.5 2.0	3.57 .56 24.0 3.2	3.17 .36 22.9 1.6	2.13 • .24 20.9 1.6	
PacO ₂ torr Inspir. Time T ₁ sec. VI/T ₁ 1 7sec.	30.0 2.0 .90 .12 .36 .05	40.6 7.0 .00 .13 .37 .04	47.2 5.0 .79 .10 .36	51.2 3.8 .fi .13 .24	•
Impedance cm HoO/4/sec	17.9 4.0	7.0	24.0 5.0	27.2 4.6	

Significantly different from value at 1% or 1½%.



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