

Title : THE PHYSIOLOGY OF NARCOTIC-INDUCED RIGIDITY

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Introduction. Muscle rigidity is a familiar though poorly understood side effect of high-dose narcotic anesthesia. While compromised ventilation due to rigidity has been described, the hemodynamic, metabolic, electromyographic (EMG) and electroencephalographic (EEG) features have not been well documented. We investigated these characteristics of rigidity in patient volunteers during the induction of anesthesia with alfentanil (ALF).

Methods. With UCSD and VA human research committees' approval and patient written informed consent, 20 patients of ASA class 1-3, were randomly assigned sodium thiopental (STP) 4 mg/kg (10 patients) or alfentanil (ALF) 175 mcg/kg (10 patients) for induction. Arterial (MAP) and central venous (CVP) pressure and continuous pulse contour cardiac output were measured. Bilateral frontal EEG with on-line aperiodic analysis (Neurometrics), as well as 8 surface EMGs to assess rigidity of the neck, thorax and extremities were recorded. Oxygenation was assessed by transcutaneous O₂ and pulse oximetry. Arterial blood gases were sampled pre- and postinduction. Preinduction, an infusion of Ringers lactate 7 ml/kg was followed by five minutes of oxygenation at 8L/min. ALF or STP was administered over one minute. With ALF, rigidity was assessed in an apneic state for up to 5 minutes with the adequacy of oxygenation continuously assessed. Patients were then paralyzed, ventilated with 100% O₂ and maintained with inhalational anesthesia for surgery. Control patients received STP followed at one minute by relaxants to maintain apneic periods comparable to the alfentanil group. Statistical significance was assessed via analysis of variance and t tests.

Results. All patients receiving ALF manifested rigidity in every EMG, while there was no EMG activity with STP. With the onset of rigidity there was a significant fall in MAP ($-10 \pm$ mmHg, $P < 0.05$) which returned to baseline in 30 seconds. Cardiac index (CI) was unchanged during rigidity. Heart rate (HR) increased significantly after 1 1/2 minutes (8.0 ± 3 beats/m, $P < 0.05$). CVP rose immediately with rigidity, peaking after 3 minutes (5.3 ± 1.3 mmHg, $P < 0.02$). Patients who were rigid became significantly more acidotic due to greater increases in base deficit (BD) while the rise in PaCO₂ was not statistically different between groups (Table 1). PaO₂ decreased at a significantly greater rate in the patients who were rigid. There was no EEG evidence of seizure activity. The EEG demonstrated a greater depth of anesthesia in patients receiving ALF when compared with STP.

Discussion. Our results show the following: 1) Rigidity is present in all

muscle groups and its onset corresponds with unconsciousness. 2) ALF-induced rigidity is associated with an immediate rise in CVP. EMG evidence suggests that elevation of the intrathoracic pressure by the thoraco-abdominal musculature is a mechanism 3). CI was unchanged. Following the initial fall, MAP was unchanged during rigidity. Changes in pH and BD during ALF-induced rigidity are significantly greater than those occurring with in non-rigid apneic patients receiving STP. 4) Mitchell et al¹ described reflex increases in HR and MAP which accompany increased muscle tone and maintain O₂ delivery. Metabolic changes during rigidity may result from the absence of these cardiovascular reflexes. 5) No seizure activity was detected in the EEG during rigidity. An animal model of narcotic induced rigidity has been described² and used to define opiate receptors in the basal ganglia mediating rigidity^{3,4}.

References

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	ALFENTANIL	THIOPENTAL
Duration of Apnea (min)	4.3 \pm 0.2 +	3.8 \pm 0.2
PaO ₂ mmHg Pre Induction	401.0 \pm 19.0 +	425.0 \pm 19.0
Δ PaO ₂ / Δ t mmHg/min	51.0 \pm 12.0 *	12.0 \pm 6.0
Δ pH/ Δ t u/min	- 0.035 \pm -0.002 **	- 0.026 \pm 0.002
Δ CO ₂ / Δ t mmHg/min	4.49 \pm 0.36 +	4.41 \pm 0.40
Δ BE/ Δ t u/min	- 0.72 \pm 0.17 **	- 0.14 \pm 0.11

Table 3 - Comparative metabolism during apnea. (mean \pm SE)

+ NS
* P < 0.02
** P < 0.01