

Title: PROPOFOL EFFECTS ON EEG AND RELATIONSHIP WITH PLASMA CONCENTRATION DURING NEUROSURGERY

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The goal of this study was to assess the relationship between propofol plasma concentration (PPC), EEG activity and cardiovascular (CV) effects¹ during neurosurgery.

The protocol was approved by the ethics committee and informed written consent was obtained from 7 nonpremedicated patients (48.5±6.6 year, weighing 64.9±1.9 kg) with normal preoperative EEG, undergoing brain biopsies. EEG was recorded continuously from 4 electrodes (2 frontals, 2 occipitals). Anesthesia was induced with propofol 2 mg/kg i.v.; dextromoramide 0.05 mg/kg i.v.; vecuronium 0.01 mg/kg i.v. After intubation, anesthesia was maintained with propofol 6 mg/kg/h. The rate of infusion was increased by increments of 2 mg/kg/h until burst suppression was obtained (EEG stage I, maximum dose 14 mg/kg/h). According to the EEG changes, blood samples for PPC were collected during stages I to III. In addition, ECG (lead II), blood pressure and ETCO₂ were monitored prior to and

during surgery.

Duration of anesthesia was 113±11.5 min, propofol dose was 8.93±1.03 mg/kg/h. Results are presented in Tables 1 and 2. Burst suppression required PPC of 5.02 mcg/ml.

Our data indicate that there is a positive relationship between PPC and EEG activity and that CV changes are minimal during propofol anesthesia. These results suggest that propofol may be a useful anesthetic during neurosurgery.

References

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Table 1. EEG Description of the Different Stages

Stage	Description
I	Burst suppression: 10-12 Hz or 1-4 Hz, low amplitude and electrical silence during 4-6 sec.
II	Periodic activity: 10-12 Hz, low amplitude and 1-4 Hz low amplitude.
III	1-4 Hz moderate amplitude achieved with 12-15 Hz low amplitude.

Table 2. Data Function of EEG Stage

EEG	PPC ug/ml	HR bts/min	MAP mmHg	RATE mg/kg/min
AWAKE	0	82±6	113±6	0
Stage I	5.09±0.64	69±3	92±3*	10.7±1.1
Stage II	3.86±0.77	68±4	93±5*	2.7±1.2
Stage III	1.60±3.0	76±4	109±5	0

Mean ± SEM; ANOVA one way; * P < 0.05 vs control

TITLE: CEREBRAL BLOOD FLOW REACTIVITY TO CO₂ DURING HALOTHANE OR ISOFLURANE ANESTHESIA FOR CAROTID ENDARTERECTOMY

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Decreasing PaCO₂ during temporary vascular occlusion may have a beneficial influence on redistribution of CBF to ischemic brain.¹ This study compared the effects of isoflurane (ISO) and halothane (HAL) on cerebral blood flow (CBF) reactivity to changes in PaCO₂ during carotid endarterectomy.

METHODS. After obtaining institutional approval and informed consent, 14 patients, mean (± SE) age 68 ± 2 received either ISO (n = 12), 0.75% or O₂/N₂O, 1:1 or HAL (n = 7), 0.5% in O₂/N₂O, 1:1. Measurements were made during the period of temporary bypass shunting. Global CBF was measured using i.v. 133-Xe.² After a baseline CBF measurement, the PaCO₂ was elevated 8-10 mmHg by addition of CO₂ to the inspired gas mixture and CBF was again measured. Data are expressed as mean ± SEM.

RESULTS. By ANOVA, there was a significant effect of PaCO₂ to increase CBF but no difference between anesthetics, as shown in Fig. 1. There were no significant differences in mean blood pressure, temperature or heart rate between groups during the 2 measurements. No cerebral ischemia was detected by EEG. There was no difference (P = 0.913) in mean CBF reactivity to

changes in PaCO₂ (ml/100g/min/mmHg) between ISO (1.7 ± 0.5) and HAL (1.8 ± 0.4). These values correspond to a relative 4 and 5 %-change in CBF per mmHg, respectively. The extrapolated CBF (ml/100g/min) at PaCO₂ = 40 mmHg tended (P = 0.126) to be higher for HAL (41 ± 4) than ISO (32 ± 3).

DISCUSSION. ISO increases the slope of feline CBF responsiveness to changes in PaCO₂ in the hypocapnic range relative to HAL.³ In elderly patients undergoing carotid endarterectomy with relatively low maintenance concentrations of HAL or ISO in N₂O there was no significant difference in their effects on CO₂ reactivity in mildly hypo- to normocapnic ranges.

REFERENCES

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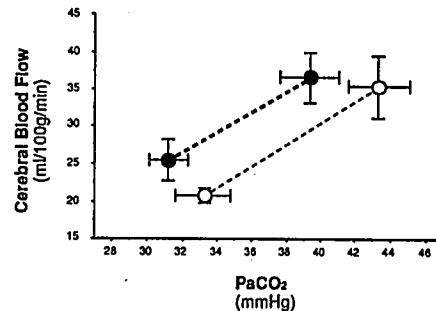


Table 1. Mean CBF as a function of PaCO₂ for ISO (open circles) and HAL (closed circles).