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TRANSCRANIAL DOPPLER SONOGRAPHY MONITORS CHANGES IN CEREBRAL BLOOD FLOW DURING ISOFLURANE ANESTHESIA

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This study investigates the effects of incremental concentrations of isoflurane on cerebral blood flow (CBF) and cerebral oxygen consumption (CMRO2). CBF measured with radioactive microspheres is correlated with cerebral blood flow velocity measured by transcranial Doppler sonography (TCD).

Following approval of the Institutional Animal Care Committee, 10 mongrel dogs were anesthetized with isoflurane and catheters were inserted into both femoral arteries, veins and the lateral ventricle for blood pressure measurement, blood sampling, and ICP determination. Brain temperature, arterial blood gases and pH were maintained constant. Arterial blood pressure was maintained constant over time using infusion of phenylephrine. Radioactive microspheres were injected using a left atrial catheter for measurement of global CBF. A pulsed TCD-probe (2 MHz, Transpect TCD™, Medasonics) was placed on the dura via a temporal bone window to measure mean flow velocity (Vmean, cm/s) in the middle cerebral artery. At completion of surgery isoflurane was discontinued and a 30 min equilibration period was started with infusion of fentanyl (45µg/kg/h) and 50% N2O/O2. Following baseline recordings, the animals were divided into two groups. In group 1 animals (n=7), isoflurane was added to the inspiratory gas mixture and cerebral hemodynamic and metabolic measurements were repeated at end-tidal concentrations of 1 %, 2 %, and 3 % isoflurane, respectively. Animals in group 2 (control, n=3) received the same measurements of CBF, Vmean and CMRO2 at 30 min, 60 min and 90 min but were not given isoflurane.

Data for CBF, Vmean, CMRO2 and ICP of isoflurane anesthetized dogs are given in table 1. Isoflurane significantly increased CBF, Vmean and ICP at concentrations of 2 % and 3 % compared to baseline values. Cerebral hemodynamics and metabolism were unchanged in animals of group 2 (control). Changes in CBF and Vmean were closely correlated ($r = 0.81$, $p<0.01$). The increase in CBF and Vmean was associated with decreases in CMRO2.

The close correlation between increases in CBF and Vmean indicates that TCD continuously measures changes in CBF following administration of incremental concentrations of isoflurane. These changes are not due to differences in brain temperature, arterial blood gases or deterioration of the surgical preparation over time as indicated by constant data in group 2. The significant increase in ICP indicates that isoflurane increases both, CBF, and cerebral blood volume. The reductions of cerebral oxygen consumption parallel to increases in ICP indicates uncoupling of the ratio between cerebral metabolism and CBF during isoflurane anesthesia.

	CBF (ml/100g/min)	Vmean (cm/s)	CMRO2 (mlO2/100g/min)	ICP (mmHg)
baseline	71±4	38±3	7.4±0.6	13±2
1% isoflurane	66±5	34±3	7.6±0.3	17±2
2% isoflurane	92±13*	46±3*	5.5±0.3*	23±2*
3% isoflurane	138±19*	63±4*	1.8±0.2*	28±2*

Table 1: Cerebral hemodynamic and metabolic data (mean±SE) during baseline measurements compared to incremental concentrations of isoflurane (* = $p < 0.05$ compared to baseline).

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CHANGES IN EEG, CEREBRAL BLOOD FLOW, AND CEREBRAL BLOOD FLOW VELOCITY WITH REPEATED PROPOFOL INFUSIONS IN DOGS

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The purpose of the present study was to evaluate cerebral vascular and neuronal functional parameters with repeated infusion of propofol. Propofol was infused to produce burst suppression of EEG.
Methods: Following approval of the Institutional Animal Care Committee, 6 dogs (27-33 kg) were anesthetized with isoflurane and N_2O in oxygen. Arterial, venous and sagittal sinus catheters were inserted for blood pressure (MABP) measurement, blood sampling (blood gases, propofol plasma concentration) and drug administration. Blood flow velocity (Vmean) and pulsatility index (PI) in the middle cerebral artery (MCA) were measured continuously using a pulsed Doppler ultrasound system. The EEG was recorded from parieto-temporal recording sites and stored on magnetic tape for off-line analysis. Rectal temperature, arterial blood gases and pH were maintained constant. After equilibration with 0.7% isoflurane and 50% N_2O in oxygen baseline measurements were obtained for cerebral blood flow (CBF, microsphere technique), Vmean, EEG, MAP and blood gases. Propofol was infused iv at a rate of 0.8 $mg \cdot kg^{-1} \cdot min^{-1}$ until the onset of EEG burst suppression. Propofol infusion was then discontinued. After recovery of EEG to predominant theta-alpha activity (5-10 Hz) propofol infusion was repeated to again produce EEG burst suppression. Measurements were made during each of the treatment conditions.
Results: Changes in EEG (median frequency), Vmean, PI, CBF and cerebral metabolic rate for oxygen ($CMRO_2$) are given in figure 1. Median EEG frequency remained suppressed for a period of 20 to 30 min following each propofol infusion, whereas recovery of Vmean and PI started within 5 min following EEG burst suppression. Overall, the correlation of EEG and Vmean with plasma propofol was $r = 0.46$ and $r = 0.79$ ($p<0.05$), respectively.
Discussion: These data show that propofol decreases CBF, Vmean, and $CMRO_2$ and increases PI concurrently to a similar extent. Vmean recovered earlier than EEG median frequency after propofol infusion. Changes in CBF and Vmean correlated closely. Our data indicate an uncoupling between propofol-induced cerebral vasoconstriction and EEG.

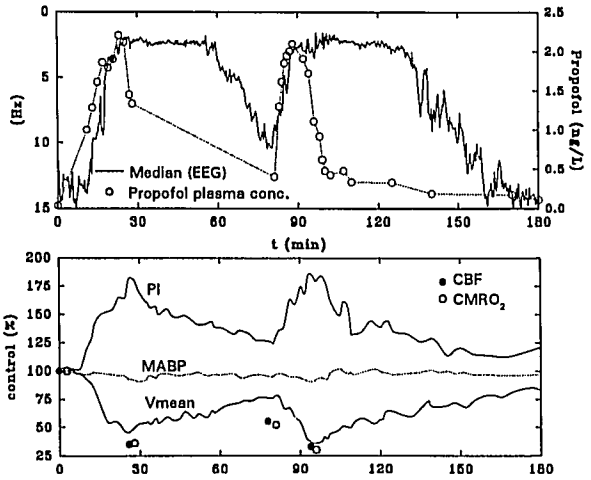

Figure 1: Median EEG frequency, propofol plasma concentrations (up-per graph), MABP, mean blood flow velocity (MCA), pulsatility index (PI), CBF and $CMRO_2$ following repeated propofol infusion.