Title: EFFECTS OF ENFLURANE ON RETINAL BLOOD FLOW IN CATS

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General anesthesia is frequently chosen for retinal surgery, but little data is available on its effects on the ocular circulation. We have previously reported dose-dependent increases in preretinal oxygen tension, which could be caused by either increased retinal blood flow (RetBF) or decreased retinal metabolism, during enflurane anesthesia.[1] In this study we tested the hypothesis that retinal blood flow is increased during general anesthesia with enflurane.

Eleven adult cats weighing 2.5-3.5 kg were studied after approval of our Animal Care Committee. The animals were anesthetized with enflurane and air-oxygen. The lungs were mechanically ventilated to maintain arterial PCO2 at 25-30 mm Hg and PO2 above 100 mm Hg. A left atrial (LA) catheter was placed via a thoracotomy and a femoral artery was cannulated for withdrawal of arterial blood samples. We continuously recorded mean arterial pressure (MAP), left atrial, intraocular (IOP) and airway opening pressures. IOP was measured as previously described.[1] End-tidal enflurane and CO2 were monitored using mass spectrometry. Blood flows were measured by injection of radioactively-labelled microspheres of Sn 113, Ce 141 and Nb 95 into the LA using the reference blood sample method.[2] Measurements were made in eight cats at 0.5, 1.0, and 1.5 MAC enflurane administered in a random sequence. Three other cats served as controls in which blood flow was measured repeatedly at a constant anesthetic concentration At the conclusion of the experiment, after the animals were killed, the eyes were enucleated and then dissected carefully to remove the retina. Results were analyzed using repeated measures ANOVA with $p < .05$ considered statistically significant.

Ocular perfusion pressure (OPP = MAP - IOP) decreased significantly during enflurane anesthesia. There were, however, significant dose-dependent increases in RetBF despite the decline in perfusion pressure (Table).

	Enflurane Concentration (MAC)		
	0.5	1.0	1.5
OPP (mmHg)	104 ± 2	74 ± 5*	57 ± 4§
RetBF (ml/100gm/min)	104 ± 21	208 ± 25*	237 ± 26§

* $p < .05$ vs. values at 1.5 and 0.5 MAC
§ $p < .05$ vs. values at 1.0 and 0.5 MAC
Values are mean ± sem.

These results indicate that increasing anesthetic depth causes vasodilatation in the retina. RetBF is critical for the blood supply to the inner retina; therefore, in conditions under which RetBF may become compromised, such as in diabetic retinopathy, enflurane may be beneficial by increasing RetBF.

References: 1) ARVO Abstracts,179, 1988.
2) Prog Cardiovasc Dis 20:55-78, 1977.

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A346

Title: DESFLURANE MAC IS DECREASED BUT RECOVERY TIME IS UNALTERED FOLLOWING PREMEDICATION WITH MIDAZOLAM (0.05MG/KG)

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Desflurane, a new volatile anesthetic with a very low blood:gas partition coefficient (1), is undergoing clinical trials. We conducted a controlled, randomized study to assess the possible MAC reduction of premedication with midazolam prior to anesthesia with desflurane in 26 ASA Class I or II female patients (age, 30-65 yrs) undergoing abdominal surgery (1.5-3 hrs duration). The protocol was approved by the local Institutional Review Board and after written informed consent the patients were randomly assigned to one of three treatment groups: 1) desflurane/O_2 anesthesia (n=9), 2) desflurane/O_2 anesthesia after premed with iv midazolam, 0.025 mg/kg, (n=8), or 3) desflurane/O_2 anesthesia after premed with iv midazolam, 0.05 mg/kg, (n=9).

Anesthesia was induced in all groups with thiamylal, 4-5 mg/kg, and succinylcholine, 1.5 mg/kg, was used to facilitate tracheal intubation after the vocal cords were sprayed with 4 ml of 4% lidocaine. MAC was determined by response to skin incision at least 20 minutes after thiamylal administration. The initial anesthetic concentrations tested were Group 1: 1 MAC (6%), Group 2: 0.8 MAC (4.8%), Group 3: 0.6 MAC (3.6%). Subsequent anesthetic doses tested (increases or decreases of 0.1 MAC [0.6%]) were determined by the Dixon Up and Down method (2) depending upon the previous patient's response to skin incision. The end-tidal concentration to be tested was stable for 10 minutes before skin incision. Anesthesia was then maintained with oxygen and desflurane with vecuronium for muscle relaxation as necessary.

At the completion of surgery, the time intervals (min) between discontinuation of the desflurane and spontaneous eye opening, response to verbal command, and orientation were recorded. Statistical analyses were performed using analysis of variance with $P<0.05$ considered statistically significant.

MAC values for the three groups are shown in the Table. The value for the control group, 6.0, was identical to the previously reported value for patients in this age group (3). The value for Group 3 was significantly less than those for Groups 1 and 2 ($P=0.0001$). The recovery times were not different between the three groups. We conclude that premedication with midazolam, 0.05 mg/kg, significantly reduces the MAC of desflurane. However, midazolam premedication did not impede recovery from anesthesia in these patients.

	Group 1	Group 2	Group 3
n	9	8	9
Midazolam dose	none	0.025 mg/kg	0.05 mg/kg
Desflurane MAC (Mean± S.D.)	6.0±0.3	5.9±0.3	4.7±0.3*
	*$P<0.05$		
Recovery variables (min) (Mean ± S.D.):			
Opens eyes	6.1±2.0	6.0±2.2	4.6±1.8
Responsive	7.3±2.3	6.8±2.1	5.8±2.2
Oriented	13.3±5.4	16.4±13.8	12.9±6.4

1. Anesth Analg 66:971-973, 1987.
2. McArthur JW, Colton T; Statistics in Endocrinology. Cambridge MA; MIT Press, 1970, pp 251-267.
3. Anesthesiology 71:A269, 1989.