

Title: ENFLURANE INHIBITS REFLEX VENOCONSTRICTION OF MESENTERIC CAPACITANCE VESSELS

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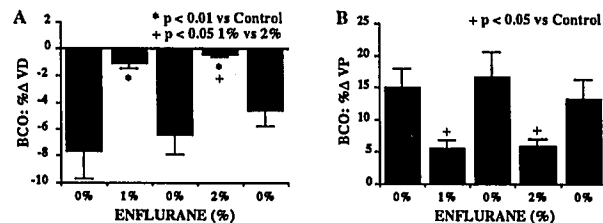
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Recent studies indicate that inhalational anesthetics, halothane and isoflurane, attenuate autonomic reflex response of splanchnic capacitance vessels which play an important role in reflex control of general circulation.^{1,2} The purpose of the present study was to examine the effect of enflurane (ENF) on the response of small mesenteric veins to sympathetic reflex stimulation.

Fifteen New Zealand White male rabbits (1.2 kg) were anesthetized with thiamylal (20 mg/kg i.v.) and maintained with alpha-chloralose (25mg/ml/hr). Surgical preparation included tracheotomy, femoral vein and artery catheterization, and a midline laparotomy. In six rabbits, carotid arteries and aortic depressor nerves were isolated for bilateral carotid occlusion (BCO:30 sec), and for aortic depressor nerve stimulation (ANS:0.05mA, 20 Hz/10sec). In nine rabbits, silver electrodes were implanted on celiac ganglion for electrical stimulation (CGS:3-5 mA, 5,10 & 20 Hz/60sec). For *in situ* preparation, a segment of terminal ileum was exteriorized and placed in a chamber superfused continuously with physiological salt solution (PSS), pH 7.4, at 37°C. Mesenteric vein diameter (VD) was measured using on-line video micrometer system. Intravenous pressure (VP) of the same vessel was recorded simultaneously by means of a microelectrode and the servo-null system.³ Mass spectrometer was used to monitor the level of ENF in expired gases. Blood and PSS concentrations of ENF were measured by gas chromatography. Changes in VD,

VP, heart rate and aortic pressure, in response to BCO, ANS and CGS, were recorded before and after 1% or 2% ENF (blood levels of 0.68 mM and 1.58 mM, respectively). In eight rabbits, the response to CGS was examined after superfusion of mesentery with PSS containing 2.18 mM ENF (5% vaporizer setting). Statistical significance of the data was determined by ANOVA.

ENF (1% and 2% inhaled) significantly inhibited reflex response to BCO, i.e. venoconstriction and simultaneous increase in VP as shown in the figure A and B, respectively. A decrease in venodilation occurring in response to ANS was also observed. There was no marked effect of ENF on active venoconstriction in response to graded CGS during systemic 2% ENF administration or during superfusion with PSS equilibrated with 5% ENF.



In situ, ENF depresses sympathetic reflex mediated venoconstriction of mesenteric capacitance vessels, possibly by influencing autonomic responsiveness at the central and/or preganglionic level.

References: (1) *Anesthesiology* 71:A148, 1989. (2) *Anesth Analg* 70:S260, 1990. (3) *Am J Physiol* 256(25):H1066, 1989.

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Title: EFFECTS OF HEMODILUTION ON RETINAL BLOOD FLOW IN CATS

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Retinal vascular insufficiency with damage to the inner retina resulting in visual loss may occur in the perioperative period. Hemodilution has been proposed as a means of increasing retinal blood flow (RetBF) and thus may be useful under these circumstances.¹ Preretinal oxygen tension, which reflects retinal oxygen availability, has been shown to increase after hemodilution in experimental animals.² We performed this study to test the hypothesis that increases in O₂ availability after hemodilution are caused by an increase in RetBF.

Ten adult cats weighing 2.5-3.5 kg were studied after approval of our Animal Care Committee. The animals were anesthetized with halothane 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 both femoral arteries were cannulated for withdrawal of arterial blood samples and pressure monitoring. The following were recorded continuously: Mean arterial pressure (MAP), left atrial, and intraocular (IOP) pressures. IOP was measured as we have previously described.³ 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.⁴ Measurements were made before and after

isovolemic hemodilution to Hct 21-23% with 6% hydroxyethylstarch. Three animals who were not hemodiluted served as controls. After the animals were killed, the eyes were dissected and the retina was carefully separated. Results were analyzed using t testing with p < .05 considered statistically significant.

Retinal perfusion pressure (RPP = MAP - IOP, mmHg) and IOP were not significantly altered by hemodilution. RetBF was increased nearly twofold by hemodilution from 101 ± 12 to 191 ± 21 ml/100gm/min (mean ± sem, *p < .05).

Baseline				Hemodiluted			
Hct	IOP	RPP	RetBF	Hct	IOP	RPP	RetBF
38±2	22±2	104±6	101±12	22±1	18±1	103±5	191±21*

In control, non-hemodiluted animals, there was no change in retinal blood flow over a comparable time course.

These results document that isovolemic hemodilution produces increases in retinal blood flow. Hemodilution may play a beneficial role in the treatment of ischemic diseases of the retina.

References:

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2. ARVO Abstracts, 138, 1989
3. ARVO Abstracts, 3282, 1990
4. Prog Cardiovasc Dis 20:55, 1977

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