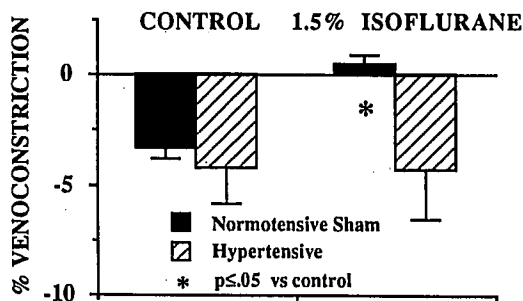


A571

Title: THE DIFFERENTIAL EFFECT OF INHALED ISOFLURANE ON REFLEX MESENTERIC VENOCONSTRICTION IN THE NORMOTENSIVE AND CHRONICALLY HYPERTENSIVE RABBIT
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Hemodynamic fluctuation frequently characterizes the anesthetic course of chronically hypertensive patients.¹ We have recently observed a greater reflex increase in sympathetic efferent nerve activity during isoflurane anesthesia in chronically hypertensive rabbits as compared with normotensive controls under similar conditions. Venous as well as arterial tone is under the control of such reflex neural activity and contributes significantly to hemodynamic control.² In previous studies in rabbits, inhaled anesthetics inhibited reflex mesenteric venoconstriction in response to both carotid sinus reflex and chemoreflex activation.³ The objective of the present study was to compare the effects of isoflurane on carotid sinus-mediated reflex mesenteric venoconstriction and related cardiovascular responses in normotensive and chronically hypertensive rabbits.

Under general isoflurane anesthesia, rabbits (1.5-2.0 kg) were made chronically hypertensive (MAP=106±5 mmHg) by surgical placement of inert latex wraps around both kidneys. Sham control rabbits (MAP=76±8 mmHg) were treated in identical fashion except for placement of the kidney wraps. Following 3 weeks of recovery, all animals were surgically prepared with a tracheostomy for ventilatory control and femoral venous and arterial cannulae for infusion and for blood pressure and heart rate measurement respectively. A 13 cm loop of terminal ileum was externalized through a midline laparotomy and superfused with physiologic salt solution (37°C). The mesentery was transilluminated and the diameters of small mesenteric veins (500-800 μ) were measured continuously by video microscopy. Reflex venoconstriction in response to 40 sec intervals of bilateral carotid occlusion (BCO) was significantly reduced by 1.5% inhaled isoflurane in the sham animals but not in the hypertensive animals (see figure). In addition, the reflex arterial pressure increase in response to BCO in the hypertensive group was less attenuated by isoflurane than in the sham group.



These data are consistent with an enhanced reflex sympathetic nerve activity in hypertensive rabbits, and suggest that increased venous as well as arterial tone may contribute to hemodynamic instability in the chronically hypertensive patient under anesthesia. (Supported by VA Merit Review 7793-02P and NIH training grant GM 08377)

References:

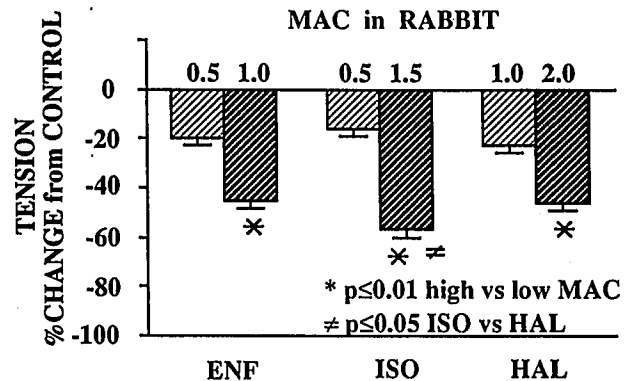
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- 2) *News in physiological science* 5:250-254, 1990.
- 3) *FASEB J* 5(5):A1405, 1991.

A572

TITLE: VOLATILE ANESTHETICS ATTENUATE ENDOGENOUS NOREPINEPHRINE EFFECT IN ISOLATED RABBIT MESENTERIC VEINS
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Volatile anesthetics may induce peripheral vasodilatation not only by altering the central and autonomic nervous system function, but also by exerting direct vasodilator effect on vascular smooth muscle.¹ Recent *in vitro* studies^{2,3} have demonstrated that halothane and enflurane can modulate the response of mesenteric veins to exogenous and endogenous norepinephrine (NE). The purpose of the present study was to compare the direct effect of enflurane (ENF), isoflurane (ISO) and halothane (HAL) on endogenous NE release and the contractile responses of isolated rabbit mesenteric veins.

Small (500 - 800 μm in diameter) mesenteric veins were isolated from the ileal region of 22 thiamylal - anesthetized New Zealand white rabbits (1.5±0.2 kg). The veins were cut into ring segments (n=93) 3 mm in length. Mesenteric vein rings (MVR) were suspended on tungsten triangles in the temperature controlled baths (37°C) containing modified Krebs' solution gassed with 95%O₂-5%CO₂ and were connected to force transducers for isometric tension measurement. To produce electric field stimulation platinum electrodes were placed in the baths at right angles to the vessel rings. After prestretching to optimal resting tension, MVR were allowed to equilibrate for 120 min. Contractile responses of MVR produced by electric field stimulation (10V, 40Hz, 2 ms square pulses of 2 min duration) were measured in control conditions and in the presence of either ENF (0.5 & 1.0 MAC), ISO (0.5 & 1.5 MAC) or HAL (1.0 & 2.0 MAC). The release of endogenous NE by electric field stimulation was verified by inhibiting the contractile responses with phentolamine (10⁻⁶M). Data were analyzed using ANOVA (p ≤ 0.05).



The results of this study demonstrate that ENF, ISO and HAL significantly, and in a dose-dependent manner, attenuate contractile responses of MVR to endogenous NE. The data also indicate that ISO is a more powerful vasodilator of small mesenteric capacitance veins than HAL.

References

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