

TITLE: ISOFLURANE-INDUCED VASODILATION MINIMALLY INCREASES CUTANEOUS HEAT LOSS

Authors: DI Sessler, M.D., J McGuire, A Moayeri, B.A., J Hynson, M.D.

Affiliation: Department of Anesthesia, University of California, San Francisco, California

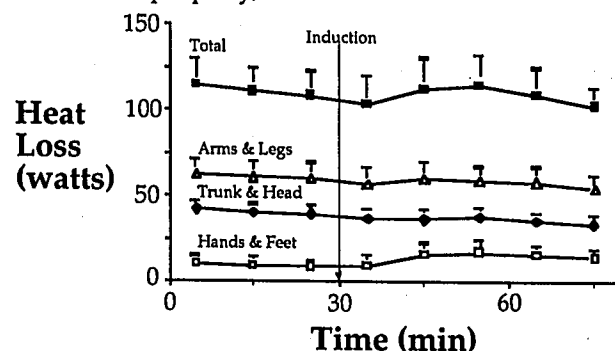
Hypothermia develops rapidly following induction of general anesthesia. We tested the hypothesis that hypothermia during isoflurane anesthesia results from increased heat loss to the environment due to anesthesia-induced vasodilation.

With approval of our IRB, we studied 5 minimally clothed volunteers in a $21.1 \pm 0.7^\circ\text{C}$ environment. After 30 min of control measurements, anesthesia was induced by inhalation of isoflurane and N_2O , and maintained with isoflurane/air. Cutaneous heat loss was measured from 10 area-weighted sites using thermal flux transducers. Peripheral blood flow was evaluated using venous-occlusion volume plethysmography and forearm-fingertip skin-surface temperature gradients.

Prior to induction, forearm-fingertip gradients $> 4^\circ\text{C}$ indicated significant vasoconstriction. Anesthesia increased fingertip blood flow 13-fold and reduced the forearm-fingertip gradients to $< 0^\circ\text{C}$. Total heat loss temporarily increased 7 watts due to a 2-fold increase in

heat loss from the hands and feet (fig). Tympanic membrane temperature remained steady during the control period, but thereafter decreased 1.2°C in 50 min.

Neither increased heat loss nor decreased heat production, alone or in combination, could account for the observed 1.2°C hypothermia. A steady ten watt increase in total heat loss would decrease mean body temperature only 0.1°C in 50 min. Complete cessation of metabolic heat production would reduce mean body temperature only 0.8°C in 50 min. This suggests that the rapid central hypothermia which follows induction of general anesthesia results primarily from redistribution of heat within the body (from the central compartment to the cooler periphery).



Supported by NIH grant #R29 GM39723 and Augustine Medical, Inc.

TITLE: URAPIDIL FOR THE TREATMENT OF STERNOTOMY INDUCED HYPERTENSION

AUTHORS: J. Marty, M.D., Y. Pansard, M.D., J.P. Lançon, M.D.

AFFILIATION: Dept Anesth., Hôp. Bichat, 75018 Paris, Association Claude-Bernard, France

Hypertension remains a common clinical problem during CABG particularly at the time of sternotomy. Urapidil, a new α -1 blocking agent, that has been shown to be effective for treatment of perioperative hypertension, might be an interesting alternative to other agents. This study was designed to evaluate the efficacy and hemodynamic effects of urapidil when used in the treatment of sternotomy induced hypertension.

Following institutional approval and informed consent, 10 patients scheduled for elective CABG were studied. Anesthesia was induced using fentanyl (80 mcg/kg), midazolam (0.1 mg/kg) and pancuronium (0.1 mg/kg). Hypertension was defined as a mean arterial pressure above 100 mm Hg for more than 2 min following sternal retraction. Urapidil was administered IV by 25 mg increments until mean arterial pressure decreased below 100 mm Hg. Hemodynamic data, i.e. cardiac output (CO), heart rate (HR), systemic vascular resistance (SVR) and pulmonary capillary wedge pressure (CWP) were recorded during steady state anesthesia, at the time of hypertension and at the time of maximal therapeutic response.

Results are summarized in the table as

mean \pm SD. Urapidil (50 mg (n = 6), 75 mg (n = 2), 100 mg (n = 2) induced therapeutic responses in all cases by a decrease of SVR. CWP decreased while no significant change in CO were observed. HR increased if compared to control values.

This study indicates that urapidil is an effective treatment of sternotomy induced hypertension. In addition, urapidil reduces both afterload and preload. The combination of vasodilation with moderate effects on HR makes urapidil useful for the management of hypertension in patients with coronary artery disease.

	Anesthesia	Hypertension	Urapidil
MAP (mm Hg)	82 \pm 11	119 \pm 10*	82 \pm 7**
HR (bts/min)	57 \pm 6	61 \pm 11	69 \pm 11*
CO (l/min)	4.1 \pm 0.5	4.6 \pm 1.2	4.2 \pm 0.8
SVR (units)	19 \pm 3	26 \pm 5*	19 \pm 3**
CWP (mm Hg)	7 \pm 3	10 \pm 1*	7 \pm 2**

* p < 0.05 vs anesthesia

** p < 0.05 vs hypertension (ANOVA)

Reference

1. Eur J Anaesth 2:21-27, 1985