TITLE: DESFLURANE (I-653) AND ISOFLURANE IN SURGICAL PATIENTS: COMPARATIVE HEMODYNAMICS AND EMERGENCE

AUTHORS: MF Battito, MD, R Langner, RN, EL Bradley, PhD
AFFILIATION: Department of Anesthesiology, University of Alabama Medical Center, Birmingham, Alabama 35233

Hemodynamic effects and emergence times with desflurane anesthesia have been reported in volunteers and small groups of surgical patients (1-4). This study was designed to compare the hemodynamic effects of and emergence from desflurane (n = 21) and isoflurane (n = 12) in surgical patients.

Methods: After IRB approval and informed consent, 33 ASA I or II patients (ages 22-65 y.o.) scheduled for elective orthopedic surgery were studied. Patients were premedicated with I.V. midazolam 0.025-0.05 mg/kg, 5-10 min prior to anesthesia induction. Thiopental was administered (3-5 mg/kg) to induce anesthesia (unconsciousness). Succinylcholine was given to facilitate intubation. After endotracheal intubation, mechanical ventilation was initiated with oxygen and desflurane or isoflurane. Mean arterial pressure (MAP) and heart rate (HR) were measured with a Dinamap 1846SX.

Results: The groups were similar in their doses (MAC values) of inhalation anesthetic, thiopental, midazolam, and fentanyl. They were also similar in age, weight, and induction-to-incision time. Differences in MAP and HR between the two groups did not reach a level of statistical significance (table). "Wake-up" times (average response time between opening eyes and squeezing hands) (3) were significantly shorter with desflurane (8.1 ± 3.1 [S.D.] min) than with isoflurane (25.4 ± 15.4 min), even though anesthesia time was not shorter for the desflurane group (144 ± 67 min *vs.* 129 ± 55 min ($p > 0.05$) for isoflurane). In addition, Aldrete recovery scores on arrival to RR were significantly ($p < 0.05$) higher with desflurane (8.5 ± 0.9) than with isoflurane (6.5 ± 2.3). Aldrete scores were similar after 30 min. The incidences of postoperative nausea, vomiting, and headache for desflurane *vs.* isoflurane were similar.

Conclusions: Thus, patients recovered significantly faster after desflurane than after isoflurane anesthesia. Changes in MAP and HR were similar in desflurane and isoflurane groups.

This study was supported by a grant from Anaquest, Inc.

References:

(1) *Anesth Analg* 70:S426, 1990 (2) *Anesthesiology* 71:A25, 1989
(3) *Anesth Analg* 70:S378, 1990 (4) *Anesthesiology* 71:A269, 1989

Table 1. Hemodynamics of Desflurane *vs.* Isoflurane (mean ± S.D.)

Time	Desflurane (n = 21)		Isoflurane (n = 12)	
	MAP	HR	MAP	HR
Baseline (preinduction)	105 ± 16	75 ± 13	101 ± 10	73 ± 11
2 min preincision	83.5 ± 21*	75 ± 19	87 ± 15*	81 ± 16
Peak after incision	108 ± 27†	92 ± 19*†	102 ± 19†	97 ± 22*†
OR, last value	102 ± 1†	84 ± 19*†	101 ± 17†	81 ± 13
RR, first value	102 ± 12†	89 ± 16*†	104 ± 15†	87 ± 14*

$p < 0.05$* = Δ from baseline
$p < 0.05$† = Δ from preincision

A394

TITLE: ISOFLURANE, HALOTHANE AND AGONIST-INDUCED RESPONSES IN CULTURED VASCULAR SMOOTH MUSCLE CELLS

AUTHORS: R Nelson, JC Sill, M.B.B.S., C Uhl, R Van Dyke, Ph.D., P Ialzzo, Ph.D.
AFFILIATION: Mayo Foundation, Rochester, MN 55905

Isoflurane and halothane attenuate the effects of contractile agonists on coronary arteries. The mechanism is unknown although they may act upon Ca^{2+} mobilization and perhaps inositol phosphate formation (an intracellular second messenger). Here vascular smooth muscle cells and 3 different agonists were used to ask--do isoflurane and halothane: (a) depress cytosolic Ca^{2+} transients measured using the fluorescent indicator Indo-1?; (b) decrease $^{45}Ca^{2+}$ influx?; (c) depress inositol phosphate formation?

Vascular smooth muscle cells (A10, BC3H-1) were cultured and studied with and without isoflurane or halothane (1.5%) using three protocols: (a) Cells were loaded with Indo-1/AM. Cytosolic Ca^{2+} responses to ATP 10^{-4}M and vasopressin 10^{-6}M were measured in individual cells at 400-800 cells.sec^{-1} using a flow cytometer. (b) $^{45}Ca^{2+}$ influx was measured in response to angiotensin II 10^{-6}M. (c) Cells were labeled with ^{3}H-inositol and inositol phosphate levels measured following addition of angiotensin II 10^{-6}M.

Results indicate (a) isoflurane attenuated the increase in cytosolic Ca^{2+} evoked by vasopressin and measured by indo-1. ATP response was unchanged by isoflurane. Halothane attenuated responses to both agonists (Fig.); (b) isoflurane and halothane depressed $^{45}Ca^{2+}$ influx evoked by angiotensin II; (c) neither anesthetic decreased agonist induced inositol phosphate formation.

Halothane may be more consistent than isoflurane in inhibiting increase in cytosolic Ca^{2+}. In this experiment, at 1.5% concentrations, neither aneshetic inhibited inositol phosphate formation. The mechanism of anesthetic action remains elusive.

$^{45}Ca^{2+}$ influx . counts min^{-1} x 10^2

Angio II	Angio II + hal	Angio II + iso
22 ± 2	14 ± 2	16 ± 1