Rhinology, 31, 81-83, 1993

A preliminary comparison of the effects of halothane and isoflurane on nasal mucosal blood flow*

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SUMMARY

The differing effects of halothane and isoflurane on nasal mucosal blood-flow was investigated by means of laser-doppler flowmetry in a total of fourteen patients who received one of these inhalational agents during anaesthesia. A trend towards lower nasal flux was seen in the halothane group but, due to insufficient numbers, significance could not be demonstrated. These changes in flux appeared not to be related to the falls in perfusion pressure which were seen in both groups of patients and were thought to be due to locally vasoactive effects.

Key words: nasal mucosa, blood flow, anaesthesia

INTRODUCTION

During nasal surgery, local vasoconstrictive agents and hypotensive anaesthesia may both be used to decrease operative blood loss and thereby improve the operating field. The effects of inhalational anaesthetics on nasal mucosal blood flow have not been previously documented, although their effects on flow in many other organs have been extensively studied (Eger, 1981). Both Halothane and Isoflurane may be used to produce hypotensive anaesthesia, albeit by different actions. This study was designed to detect any significant differences in nasal mucosal blood flow not due to the hypotension produced but due to the specific action of each agent respectively, and preliminary results are published here.

METHODS

After approval from the Local Ethics Committee, 16 ASA-Class 1 and 2 patients, undergoing elective hypotensive anaesthesia for routine middle ear surgery, were selected (Table 1). Patients with a history of hypertension, active nasal pathology of any form and patients using oral contraceptives or any form of nasal medications were excluded. Written informed consent was obtained prior to commencement.

After pre-operative medication with 2 mg Lorazepam, the patients had anaesthesia induced with 2.5 μg/kg Fentanyl followed by 5 mg/kg Thiopentone; 0.1 mg/kg Vecuronium was given to facilitate tracheal intubation. Ventilation was maintained with 70% nitrous oxide and 30% oxygen using a Siemens "Servo-C" ventilator. The end-tidal carbon dioxide was monitored using an Engstrom Elisa infrared carbon-dioxide analyser, and carbon dioxide added to the inspired mixture as required to maintain the end-tidal value between 5.0 and 5.5 kPa.

Table 1. Patients' data. Mean (SD) patients' age, weight and sex ratio according to group

	group		
	Halothane (n=7)	Isoflurane (n=7)	significance
age (years)	35 (10.0)	33 (9.1)	NS
weight (kg)	67 (16.6)	63 (9.6)	NS
male/female ratio		5:2	5:2

NS: not significant ($p < 0.05$).

A radial arterial cannule and a right basilic vein central-venous catheter were then inserted. Arterial and venous central pressures were then measured by Hewlett-Packard transducers and the traces displayed to verify the positions of the cannules.

A laser Doppler flowmeter (Moor Instruments MBF3) was calibrated and placed under direct vision through a speculum to rest just in contact with the mucosa covering the anterior end of the most prominent inferior turbinate. The probe was adjusted to give a reading under baseline conditions at approximately the middle of the instrument's range (flux readings in arbitrary units of 0 to 1,000). The

* Accepted May 11, 1992

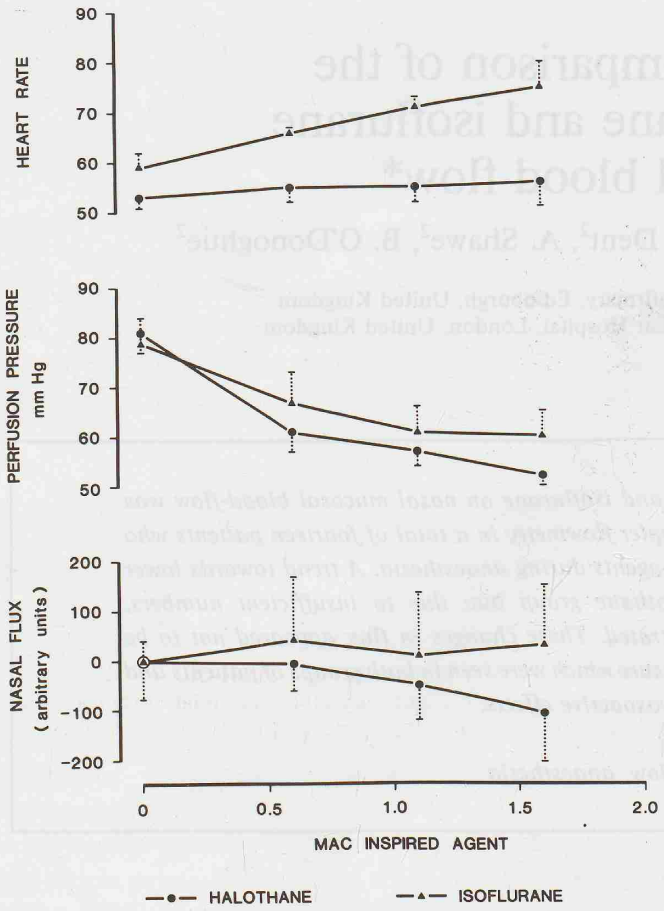

Figure 1. Nasal flux (change from control in arbitrary units) and values of key haemodynamic variables in relation to end-tidal anaesthetic agent concentration (mean±SEM).

probe was then fixed in this position, still supported by the speculum which was then also fixed.

A variable time-period was allowed for equilibration of all parameters, such that each would show no change after a 3-min continuous period. At this point the readings of mucosal flux, mean arterial blood pressure, central venous pressure, and end-tidal carbon dioxide were taken. Once this had been done, the patients were randomly assigned to either the Halothane or Isoflurane group, with 8 patients in each. The patient then received the inhalational agent at 3 different concentrations, but in a randomized varying order. To ensure equipotency, the concentrations were chosen to be multiples of the minimum alveolar concentration (MAC). The concentrations chosen were 0.6, 1.1, and 1.6 MAC, since these are convenient vapour concentrations of the magnitude used in routine anaesthesia. The MAC values were adjusted for age in accordance with the values measured in previous studies (Gregory et al., 1969; Stevens et al., 1975). The inspired and expired vapour concentrations were monitored with the calibrated Datex "Normac" infrared analyser attached to the sampling side port of the endotracheal tube. When the end-tidal concentration of the Halothane or Isoflurane was at the target value and differed by less than 50% from the inspired concentrations, repeat readings of all parameters were again

Table 2. Nasal flux expressed as change from control values in relation to end-tidal vapour concentration (in multipes of MAC_{O_2}) for Halothane and Isoflurane. Corresponding changes in key haemodynamic variables are also shown. Perfusion pressure is calculated as the mean arterial blood pressure minus mean central venous pressure. Values quoted are the mean (SD).

	group		
	Halothane	Isoflurane	significance
Control			
flux	0 (191)	(97)	NS
perfusion pressure	80 (9.8)	79 (12.8)	NS
pulse rate	52 (5.8)	59 (6.3)	NS
0.6 MAC			
flux	-7 (133)	43 (306)	NS
perfusion pressure	61 (8.8)	67 (14.9)	NS
pulse rate	55 (7.6)	66 (3.0)	*
1.1 MAC			
flux	-50 (171)	10 (312)	NS
perfusion pressure	57 (8.5)	61 (12.5)	NS
pulse rate	55 (7.6)	71 (5.1)	*
1.6 MAC			
flux	-108 (238)	29 (291)	NS
perfusion pressure	52 (4.8)	60 (11.9)	NS
pulse rate	56 (12.0)	75 (11.0)	*

*: significance between groups ($p<0.05$). NS: not significant.

noted. At this point the arterial concentration of anaesthetic vapour is known to lie within 10% of the end-tidal value (Eger and Bahlman, 1971). Between the administration of each of the three vapour concentrations, another period of stabilization was allowed during which the patient received nothing but oxygen and nitrous oxide. The expired anaesthetic concentration was allowed to return to its baseline level to allow for any possible carry over effect.

The grouped data for each concentration of inhaled agents were tested by analysis of variants. The Student's unpaired t-test was then performed on groups that showed significance, a value of $p<0.05$ was taken to indicate significance.

RESULTS

In one patient in each group there were inaccuracies of the flow measurement as a result of movement artifact due to displacement of the laser Doppler flowmeter probe. These two patients were excluded, leaving seven patients in each group.

The mean blood flux in the Isoflurane group showed an increase from the baseline levels which was not related to vapour concentrations (Figure 1). In the Halothane group, the mean blood flux decreased as alveolar concentration increased but there were no significant differences between the groups at any concentration, or between concentrations in either group (Figure 1).

Perfusion pressure was lower in the Halothane groups at all concentrations, but the difference between the two groups did not reach statistical significance at any stage. The fall in perfusion pressures seen in both groups as concentration

increased, was significant when compared to control levels.

Heart rate was significantly higher in the Isoflurane group at all concentrations except control, and this increase at 1.5 MAC from control was significant within the group itself. No significant change was observed between concentrations in the Halothane group (Table 2).

DISCUSSION

The principles and accuracy of laser Doppler flowmetry are discussed in detail elsewhere (Oberg et al., 1980; Johnson et al., 1984; Smits et al., 1986), and is now generally accepted as an extremely accurate measurement of surface blood flow.

Previous studies have shown that Halothane and Isoflurane differ in their effects on the cardiovascular system. Both produce hypotension at increasing concentrations but with Halothane the hypotension is caused by depression of cardiac output, accompanied by a rise in central venous pressure (Gregory et al., 1969). Isoflurane, however, produces hypotension by reducing systemic resistance while cardiac output is preserved (Eger, 1981). With Halothane, total peripheral resistance is unchanged because decreases in vascular resistance in organs such as skin and brain are balanced by increases in others, such as liver and muscle (Gregory et al., 1969).

The perfusion pressure – defined as the difference between mean arterial pressure and the central venous pressure – was not significantly different between the two groups at any concentration but there was a trend towards lower values in the Halothane group (Figure 1). Likewise, with the small numbers of subjects involved, no statistically significant difference in nasal mucosal blood flux could be demonstrated, despite their being apparent increases in patients given Isoflurane and decreases in those given Halothane (Figure 1). However, it is of interest that no correlation could be demonstrated between turbinate flux and the fall in perfusion pressure produced by either agent. The differences in flux produced by the two agents, therefore, is likely to be due to local vasoactive properties and not due to hypotension *per se,* although larger numbers of patients would be required for results to achieve statistical significance.

We have concluded, therefore, from this preliminary report that Halothane has a tendency to produce lower turbinate blood flux than Isoflurane and that this seems to be independent of falls produced in perfusion pressure, as long as the lower autoregulatory limit is not exceeded. More patients will need to be studied to produce results which achieve statistical significance.

ACKNOWLEDGENENTS

The study was supported by a grant from the Royal National Throat, Nose and Ear Hospital Trustees Fund. The authors would like to thank Abbot Pharmaceuticals Ltd. for kindly providing assistance with equipment and Mrs. A. White for typing the manuscript.

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