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TITLE: THE EFFECT OF DESFLURANE AND ISOFLURANE WITH N_2O ON CEREBROSPINAL FLUID PRESSURE IN PATIENTS WITH SUPRATENTORIAL MASS LESIONS

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Introduction: Desflurane is a new volatile anesthetic with a blood:gas partition coefficient of 0.42, that can result in rapid awakening. In humans with supratentorial mass lesions, 1 MAC desflurane (7.0%) has been shown to increase cerebrospinal fluid pressure (CSFP) despite prior establishment of hypocapnia.(1) The purpose of this study was to determine and compare the effects of 0.5 MAC desflurane (3.5%) and isoflurane (0.6%) both in 50% N_2O in O_2 on CSFP in neurosurgical patients with supratentorial mass lesions.

Methods: After Institutional Review Board approval, 14 patients age 21-75 with supratentorial mass lesions were randomized to receive desflurane or isoflurane. Prior to induction a radial artery catheter for blood pressure (BP) measurement and a lumbar subarachnoid needle for monitoring CSFP were placed. Anesthesia was induced with thiopental (4-7 mg/kg) followed by vecuronium (0.2 mg/kg), and ventilation was adjusted to maintain $PaCO_2$ at 24-28 mmHg. Anesthetic maintenance consisted of 3.5% end-tidal desflurane or 0.6% end-tidal isoflurane in 50% N_2O in O_2. Blood pressure was maintained within 20% of the patient's average ward value with esmolol or phenylephrine. Intravenous fluids were limited to less than 500 cc lactated Ringers prior to dural incision. CSFP was recorded with the patient awake (baseline), post-induction, post-intubation, at institution of anesthetic agent, and every 5 min until dural incision. Results were analyzed using Students t-tests for paired and unpaired data, with $p < 0.05$ considered significant.

Results: There was no difference in the mean baseline CSFP between groups. The mean baseline CSFP was 9 $\pm$ 4 mmHg (range 3-14 mmHg) for isoflurane, and 10 $\pm$ 2 mmHg (range 8-13 mmHg) for desflurane. There was no difference in the CSFP between the desflurane or isoflurane groups at any time during the study period. Maximum mean CSFP did not differ from baseline in either group. Maximum mean CSFP following isoflurane was 10 $\pm$ 6 mmHg (range 3-21 mmHg) occurring within 5-40 min. Maximum mean CSFP following desflurane was 11 $\pm$ 4 mmHg (range 4-15 mmHg) occurring within 10-75 min.

Discussion: Desflurane is reported to produce dose-dependent cerebrovasodilation.(2) This can increase CSFP at higher end-tidal concentrations in the presence of reduced intracranial compliance. A previous human study has shown that in patients with supratentorial mass lesions 1 MAC desflurane (7.0%) produced an increase in CSFP despite prior establishment of hypocapnia.(1) The results of this study indicate that in neurosurgical patients with supratentorial mass lesions, the administration of 0.5 MAC desflurane (3.5%) or isoflurane (0.6%) in 50% N_2O in O_2 with prior establishment of hypocapnia does not affect CSFP.

References

1. Muzzi DA, et al. Anesthesiology 73:A1215, 1990
2. Lutz LJ, et al. Anesthesiology 73:125-131, 1990

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TITLE: **NITROUS OXIDE IS A MORE POTENT CEREBROVASODILATOR THAN ISOFLURANE IN HUMANS**

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Use of nitrous oxide(N_2O) in neuroanesthesia is controversial as it has been shown to be a cerebral vasodilator[1,2] and may cause an increase in intracranial pressure in susceptible patients.[3] However, its cerebrovascular effects in comparison to an equipotent dose of isoflurane(**I**) has not been studied in humans.

Methods: With institutional human subjects committee approval and informed consents, 6 healthy male patients age 32± 7 yrs, weight 90±21 Kg were studied. Anesthesia was induced with thiopental 5-6 mg/kg and vecuronium 0.1 mg/kg. In each patient four steady-state anesthetic conditions with 15 minutes of unchanged end-tidal anesthetic concentrations as monitored by mass-spectrometry were studied; (A) 0.5MAC **I** (B) 0.5MAC **I** + 0.6MAC N_2O(C) 1.1MAC **I** + 0.6MAC N_2O (D) 1.1MAC **I**. B and D were considered to be equipotent. Because of concern with awareness, 0.6MAC N_2O alone was not studied. To minimize time-related changes the study sequence was randomized. In each study condition the variables measured and/or calculated included mean intra-arterial blood pressure(MAP), heart rate, cerebral arteriovenous oxygen content difference($AVDO_2$ - difference between arterial and right internal jugular bulb O_2 content), 2-channel EEG with 80% activity edge(Lifescan by Diatek), and mean right middle cerebral artery flow velocity(V_{mca}) using a transcranial Doppler(Medasonics) probe anchored to the right temporal region with a harness throughout the study. Cerebral metabolic equivalent (CME - product of V_{mca} and $AVDO_2$) was also calculated for each state. ANOVA for repeated measures and Fisher's PLSD was used for comparisons between the study conditions. $P<0.05$ was considered significant.

Results and Discussion:

	MAP (mmHg)	$PaCO_2$ (mmHg)	A-VDO_2 (vol%)	EEG (Hz)	Vmca (cm/s)	CME (cm/s.vol%)
A) .5M Iso	86±7*#	39±1	4.9±.8#	12±1*#	54±9#	264±59#
B) .5M Iso + 0.6M N_2O	74±6	40±1	4.8±.5◆	10±1◆	56±8◆	264±43◆
C) 1.1M Iso + 0.6M N_2O	64±4	41±2	3.3±.4	7±1	58±7†	180±29†
D) 1.1M Iso	66±3	40±1	3.7±.6	8±0	44±6	152±27

all values are ± sem , M = MAC
◆- p<0.05 'B'compared to'D' †- p<0.05 'C'compared to'D'
*- p<0.05 'A'compared to 'B' #- p<0.05 'A'compared to 'D'

Compared to 1.1MAC **I**(D), the equipotent mixture of **I** and N_2O(B) was associated with a higher blood flow velocity, but also a higher $AVDO_2$ and CME. EEG activity was consistent with increased cerebral metabolic activity. Adding N_2O to 1.1MAC **I**(C) increased V_{mca} and CME without altering EEG activity. These results suggest that N_2O is a more potent cerebrovasodilator compared to an equipotent dose of **I** in a **I**-N_2O mixture and that there are no advantages in adding N_2O when **I** is used in the practice of neuroanesthesia.

References 1. Stroke 21:1293-1298,1990. 2. Br J Anaesth 63:290-295,1989. 3. Br J Anaesth 45:486-492, 1973.