Title: EFFECT OF NITROUS OXIDE ON ICP FOLLOWING CRANIAL-DURAL CLOSURE

Authors: J.R. Hemstad, M.D., K.B. Domino, M.D., A.M. Lam, M.D., V. Laohaprasin, M.D., T.A. Slee, M.D., M.S. Grady, M.D., and H.R. Winn, M.D.

Affiliation: Departments of Anesthesiology and Neurosurgery, Harborview Medical Center, University of Washington, Seattle, WA 98195

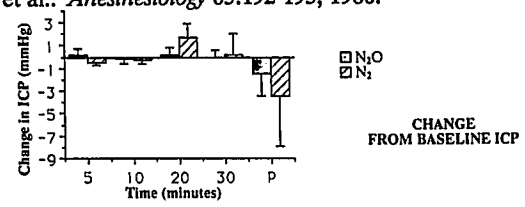
Following cranial-dural closure, nitrous oxide (N_2O) may diffuse into and expand an entrapped volume of intracranial air, thereby raising intracranial pressure (ICP).[1] Continuation of N_2O, however, does not cause further intracranial hypertension when ICP is elevated by an intraventricular infusion of air in rabbits.[2] We performed a randomized prospective study to determine the effect of continuation of N_2O on ICP in patients following dural closure.

Methods: The study was approved by the University of Washington Human Subjects Review Board, and informed consent was obtained from 14 patients undergoing craniotomy for cerebral aneurysm clipping, resection of arteriovenous malformation, or resection of brain tumor. Patients requiring prolonged hyperventilation or in whom N_2O was relatively contraindicated were excluded. All patients received standard anesthesia management consisting of thiopental, narcotics, muscle relaxants, 0-1.5% inspired isoflurane, and 60% N_2O with intraoperative use of hyperventilation, diuretics, and hemodynamic control as required by surgical procedure. $PaCO_2$ was normalized prior to, and $P_{ET}CO_2$ was kept constant after dural closure. The patients were randomized into two groups: N_2O continued following dural closure (n=9), and N_2O discontinued and replaced with nitrogen at the time of dural closure (n=5). ICP, measured by ipsilateral subarachnoid bolt, was recorded at 5 minute intervals after dural closure until completion of skin closure and immediately postoperatively (P). Presence of intracranial air was determined by head CT scan immediately postoperatively. Repeated measures data were analyzed by a mixed design analysis of variance and post hoc Newman-Keuls test.

Results: The ICP did not differ between the groups (N_2O: -0.3 ± 1.9 mm Hg vs. N_2: 3.4 ± 1.3 mm Hg at time 0). ICP was not affected by the continuation of N_2O following dural closure (see figure). Postoperative CT scans demonstrated the presence of intracranial air in all subjects.

Discussion: ICP did not change after dural closure in patients undergoing craniotomies, regardless of whether N_2O was continued or discontinued. These results provoke the question of whether routinely eliminating N_2O prior to dural closure for reasons of avoiding expansion of intracranial air and raising ICP is indicated.

References: [1]Artru: *Anesthesiology* 66:719, 1987. [2]Skahen, et al.: *Anesthesiology* 65:192-195, 1986.



A178

TITLE.: EFFECT OF HALOTHANE IN LOW CONCENTRATIONS ON CEREBRAL BLOOD FLOW AND CEREBRAL METABOLISM IN THE BABOON

AUTHORS: T. Brüssel, M.D., H. VanAken, Ph.D., W. Fitch Ph.D.

AFFILIATION: Anes. Dept., University of Münster, 4400 Münster, FRG, Anes. Dept., Kath. University of Leuven, 3000 Leuven, Belgium, Anes. Dept., University of Glasgow, Glasgow G31 2ER, Scotland

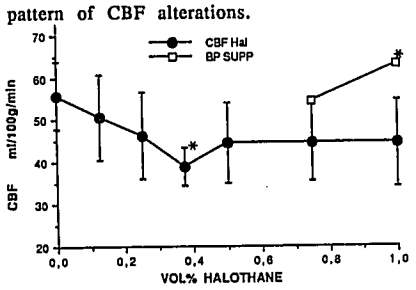
Increasing concentrations of isoflurane (I) are associated with a biphasic change of the cerebral blood flow (CBF) (1). The purpose of this study was to see whether halothane (H) in low concentrations similarly exerts a biphasic effect on the CBF.

Methods: In 8 young adult baboons (9.0-12.5 kg) anesthesia was induced with thiopental 7.5 mg/kg i.v. Muscle relaxation was achieved with 20 mg i.v. succinylcholin (S). After tracheal intuba-tion the animals were ventilated with 67% nitrous oxide in oxy-gen. Anesthesia was maintained by an infusion of phencyclidine, 0.15 mg $kg^{-1}min^{-1}$. To provide muscular paralysis S, 50 mg i.m. was added every 30 min. Arterial carbon dioxide tension was kept between 38-42 mmHg. End-tidal carbon dioxide concentration was measured continuously. Catheters were placed in the aorta for blood pressure monitoring, in the sagittal sinus to collect cerebral venous blood, and subdurally to monitor intracranial pressure. CBF was determined from a scintillation washout curve recorded over the right parietal area after the injection of $^{133}Xenon$. Mean CBF was calculated from this curve by hight-area equation. With each determination of CBF, the oxygen content of arterial and cerebral venous blood was assessed and $CMRO_2$ calculated. After baseline values were obtained H was administered stepwise in increasing concentrations (0.125, 0.25, 0.375, 0.5, 0.75 and 1.0 vol%). 10 min after each desired H concentration was reached CBF and $CMRO_2$ were determined. In addition in 6 animals blood pressure was supported at 0.75 and 1.0 vol% of H to a mean arterial pressure (MAP) of 70 mmHg by angiotensin infusion. Mean values and standard deviation were calculated for each measurement. Statistical analysis was performed by repeated measures ANOVA. $P<0.05$* was considered statistically significant.

Results: H caused a decrease in $CMRO_2$ from 3.4±0.8 at baseline to 2.4±0.4 ml $100g^{-1}min^{-1}$ at 1.0 vol% H ($p<0.05$). This decrease was accompanied by a significant reduction in CBF at 0.375 vol% of H (Fig). However, with 0.5 vol% of H CBF increased. With 0.75 and 1.0 vol% of H CBF remained unchanged compared to 0.5 vol% of H. If MAP was supported at 0.75 and 1.0 vol% H the stable CBF described before could not be observed any more. Instead CBF increased to 12.9% above baseline ($p<0.05$) with 1.0 vol% of H.

Conclusion: The administration of H in low concentrations caused a decrease in CBF concomittant to a decrease in $CMRO_2$, whereas higher concentrations, with sufficient cerebral perfusion pressure, led to an increase in CBF despite further reduction in metabolism. H does not seem to differ from I in the general biphasic pattern of CBF alterations.

Fig.



Ref.: 1. H. Van Aken, Anesth Analg 1986;65:565-74