TITLE: HYPOTENSIVE DRUGS AND OUTCOME IN CEREBRAL ANEURYSM SURGERY

AUTHORS: E.A.M. Frost, M.D., K. Tabaddor, M.D., C. U. Arancibia, M.D.

AFFILIATION: Departments of Anesthesiology and Neurosurgery
Albert Einstein College of Medicine
Bronx, New York 10461

Introduction. Cerebral vasospasm following intracranial aneurysm clipping remains an unresolved complication [1]. To facilitate microdissection, several techniques of controlled hypotension have been advocated [2]. Although previous studies have indicated reversal of experimental cerebral vasospasm in dogs by intravenous nitroprusside therapy [3], clinical trials have not confirmed preventive vasospastic effects of any anesthetic techniques [1].

This study was undertaken to determine any correlation between the use of different hypotensive agents and 1) outcome, 2) angiographic evidence of postoperative vasospasm, and 3) occurrence of side effects.

Method. The hospital course of 126 patients operated for intracranial aneurysm at our institution between the years 1974 - 77 were reviewed. Surgery was performed in each case by one of three attending neurosurgeons. The operating microscope was used in all procedures. Patients operated on less than 10 days since subarachnoid bleed were excluded.

Data on preoperative physical status was recorded. Type, amount and duration of use of hypotensive agents were tabulated. Pulmonary shunts were calculated prior to, during and after periods of controlled hypotension. Normothermia was maintained in all patients throughout the operative course.

Results. Correlation between outcome and hypotensive technique is shown in Table 1.

TABLE 1 - 126 patients 1974 - 77

Outcome	None	Trimethaphan	Halothane	SNP
Intact	7 (41%)	7 (41%)	10 (32%)	50 (82%)
New Def.	6 (35%)	7 (41%)	14 (45%)	10 (15%)
Death	4 (24%)	3 (18%)	7 (23%)	1 (2%)
Totals	17	17	31	61

27 patients in whom preoperative angiograms showed no vasospasm but who developed new postoperative deficits were investigated by angiography. The correlation of angiographic findings with the hypotensive agent used intraoperatively is shown in Table 2.

TABLE 2

Hypotensive Technique	Spasm	No Spasm	Total
SNP	1 (9%)	10 (91%)	11
Halothane	7 (43%)	9 (57%)	16
Totals	8	19	27

Side effects of trimethaphan included tachycardia, failed response and tachyphylaxis in 75%. None of these complications were encountered during nitroprusside (SNP) administration. Average total dose of SNP was 10 mg and duration of infusion was one hour. No significant metabolic acidosis was encountered. Prehypotensive shunt after induction into anesthesia average 9.5%. During halothane hypotension this increased to 16.5%, with trimethaphan, 13.8%, and with SNP 14.8%.

Discussion. Postoperative neurological deficit is often associated with angiographic evidence of vasospasm. Absence of spasm indicated preoperatively, suggests that intraoperative manipulation of the aneurysm and feeder vessels may initiate events leading to vasospasm. The nitrosyl group of SNP exerts a direct vasodilator effect on cerebral vasculature, independent of autonomic control. Our study suggests that this direct vessel action may have the ability to block the sequence of spasmogenic events.

All inhalational anesthetic agents have been shown to reverse hypoxic pulmonary vasoconstriction [4]. A similar effect has been shown with SNP in dogs [5]. This resultant increase in right to left shunting is probably not significant in otherwise healthy patients as baseline levels are quickly regained at the end of the hypotensive period. Nevertheless, the use of hypotensive agents should be minimized in patients with preexisting pulmonary diseases. Arterial blood gas monitoring is mandatory.

Conclusions. Our study suggests that the use of SNP is associated with improved surgical outcome and decreased incidence of postoperative cerebral vasospasm.

References.
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