Title: EFFECT OF PHENYLEPHRINE ON MYOCARDIAL PERFORMANCE DURING CAROTID ENDARTERECTOMY

Authors: J.S. Smith, M.D., D.J. Benefiel, M. D., P.N. Beaupre, M.D., Y.J. Sohn, M.D., F.W. Lurz, M.S., B. Byrd, M.D., A. Bouchard, M.D., N.B. Schiller, M.D., M.K. Cahalan, M.D., M.F. Roizen, M.D.

Affiliation: Departments of Anesthesia and Medicine, Division of Cardiology, University of California, San Francisco, California 94143

Introduction. Myocardial and cerebral ischemia are the major causes of morbidity and mortality associated with carotid endarterectomy. This places conflicting demands on the anesthesiologist, since maintenance of cerebral perfusion pressure by elevation of systemic blood pressure (BP) increases myocardial oxygen consumption. Vasopressors are often used to maintain BP; alternatively, a light level of general anesthesia may be employed. We investigated whether these two approaches adequately maintain BP during carotid endarterectomy and the physiologic mechanism through which this is accomplished. Additionally, we compared the incidence of intraoperative myocardial ischemia (detected by electrocardiography and 2-D transesophageal echocardiography) associated with each method.

Methods. With approval from our committee on human experimentation and with informed consent, we randomly assigned 38 patients to receive either halothane or isoflurane, with or without the use of phenylephrine (NEO). The patients received no preanesthetic medication. Radial artery catheters and seven electrocardiographic leads were placed. Anesthesia was induced with the inhalational agent plus 2 - 4 mg/kg of thiopental. Endotracheal intubation followed the administration of succinylcholine. All patients received 50% N_2O in oxygen, by controlled ventilation adjusted to maintain normocapnia. Patients assigned to the NEO group were given approximately 1 MAC of isoflurane or halothane with sufficient NEO to keep BP within 20% of mean ward BP. In the remaining patients vapor concentration was limited to that which maintained BP at the predetermined level. A 9-mm gastroscope tipped with a 3.5-mHz ultrasonic transducer was introduced into the esophagus and positioned behind the heart to obtain a short-axis view of the left ventricle at the level of the papillary muscles. The ECG was recorded prior to induction and both ECGs and echocardiograms were recorded at seven subsequent predetermined intervals. The echocardiograms were analyzed with a commercially available computer program (Diasonics Light Pen). Left ventricular end-systolic meridional wall stress (SWS) and velocity of circumferential fiber shortening (Vcf) were determined by standard formulae. Additionally the echocardiograms were analyzed by 2 independent "blinded" observers for the occurrence of segmental wall motion abnormalities (SWMA). All patients had postoperative ECGs and those with changes, or symptoms suggestive of angina also had CK isoenzymes determined. Groups were compared by Student's t-test with a Bonferroni correction as indicated except when Chi square analysis was appropriate.

Results. Preoperatively, the groups were comparable in heart rate, BP, age, sex, history of angina, myocardial infarction, and use of beta blockers. Prior to cross clamp, blood pressure was within the targeted range in 84% of patients in each group and carotid artery stump pressure, systolic BP and heart rate were not significantly different (Table). Wall stress was significantly higher and Vcf lower in the patients receiving NEO. The patients in the NEO group were given a significantly higher MAC multiple. Nine of 19 patients who received NEO, while only 4 of 19 patients who did not, developed SWMA ($p<0.2$). Three (2 in the NEO group, 1 in the remaining group) of the 13 with new SWMA also had concurrent ST segment changes consistant with ischemia. No difference between groups was attributable to the use of isoflurane or halothane. No patient suffered a myocardial infarction. All patients awoke from anesthesia neurologically intact, however, 1 patient in each group sustained a perioperative CVA.

Discussion. Both techniques maintained adequate cerebral perfusion as indicated by BP, stump pressure and immediate neurologic outcome. Greater concentrations of isoflurane and halothane plus NEO were associated with higher left ventricular SWS and diminished Vcf. We have previously demonstrated in humans that increasing concentrations of inhalational agents depress Vcf and do not change or decrease SWS and BP[1]. In this study we maintained BP with NEO and SWS increased because of left ventricular dilatation and consequent wall thinning. Increased wall stress and possible coronary artery vasoconstriction secondary to stimulation of alpha adrenergic receptors might be predicted to lead to myocardial ischemia. Indeed the incidence of myocardial ischemia as defined by SWMA tended to be more frequent in the group who received NEO. This trend deserves further study. Until this is determined, however, we believe NEO should be used with caution for the routine maintenance of BP in patients with coronary artery disease.

References.

1. Beaupre PN, et al: Contractility depression during anesthesia: Comparison of halothane, enflurane, and isoflurane by transesophegeal echocardiography. Circulation 68:III-332, 1983

Table

Hemodynamic Data at Time of Carotid Cross Clamp		
	WITHOUT NEO	NEO
Stump pressure (mmHg)	52 ± 26	60 ± 24
Vcf (circ/s)	.79 ± .28*	.52 ± .29
SBP (mmHg)	139 ± 28	145 ± 25
SWS (dynes $cm^{-2}x10^3$)	99.8 ± 44.4*	172 ± 56.8
Heart rate (bpm)	72 ± 13	72 ± 12
% MAC	.92 ± .14*	1.32 ± .13
Values are mean ± SD.	* $p < 0.05$.	