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TITLE: CLONIDINE DOES NOT PREVENT MYOCARDIAL ISCHEMIA DURING NONCARDIAC SURGERY

AUTHORS: M. LIPSZYC M.D., E. ENGELMAN M.D.

AFFILIATION: Anes. Dept., Erasme Hospital, Free University of Brussels, Belgium

INTRODUCTION: The authors investigated in a double-blind placebo controlled study the hypothesis that the oral preoperative administration of 4 $\mu g.kg^{-1}$ clonidine, to patients suffering from coronary artery disease and undergoing noncardiac surgery, reduces the number of patients suffering from myocardial ischemia, or the number of ischemic episodes, during surgery or the early postoperative period.

MATERIAL AND METHODS: After institutional approval, 2 groups of 20 informed consenting patients, undergoing carotid surgery, under a N2O - enflurane anesthesia, were studied. Patients received 4 $\mu g.kg^{-1}$ of the double-blinded tablets 90 min before anesthesia. All patients underwent the day before surgery a 2-lead continuous ambulatory ECG monitoring. Enflurane was administered to maintain systolic blood pressure inside the usual preoperative value ± 20% limits. The inspired and end-tidal concentrations of enflurane were measured using an infrared absorption technique and stored using a computerized system. Myocardial ischemia was defined as a depression of at least 1 mm or an elevation of 2 mm, measured 60 msec after the J point, lasting at least 1 min, of the ST segment of one of the 3 monitored ECG leads (D1, D2, V5), analysed automatically by a Marquette series 7010 monitor. These 3 ECG leads, the heart rate, and the blood pressure were stored every 5 sec during and after surgery using a computerized system.
Heart rate, systolic and diastolic blood pressure were analysed to detect an association of each myocardial ischemic episode and eventual changes in these parameters, by comparison of the data recorded during the episode to the data recorded during normal ST segment intervals during or after anesthesia (Student t-test for unpaired data).

RESULTS: Five patients in the placebo group and 8 patients in the clonidine group presented ischemic episodes (p=0.501, Fisher exact probability test). When the presence or absence of preoperative myocardial ischemia is taken into account as cofactor, there was also no statistical difference between the groups (Mantel-Haenszel test, Z-value=1.43, p=0.1528). All episodes were ST segment depression. All these 13 patients had myocardial ischemia in the operating room; in each group, 3 of these patients had also ischemia in the post-anesthesia care unit.
The number of episodes in patients who had myocardial ischemia was identical in both groups (Mann-Whitney U test):

	Placebo group	Clonidine group	Difference
M ± SD	3.60 ± 2.70	3.62 ± 4.14	p=0.496

When compared to the periods of normal ST segment, the vast majority of ischemic episodes in the placebo group was associated with an increase in heart rate (88.9%) and/or an increase in blood pressure (72.2%). In the clonidine patients only 58.6% of the ischemic episodes were associated with an increase in heart rate, but 51.7% were associated with a decrease in blood pressure. These distributions were statistically different between the groups for heart rate (p=0.029, Chi-square test) and blood pressure (p=0.037, Chi-square test).

CONCLUSIONS: The authors conclude that the preoperative administration of 4 $\mu g.kg^{-1}$ clonidine could not decrease the frequency of myocardial ischemia by reason of the occurence of many episodes associated with a decrease in blood pressure, despite a decrease of 24% to 39.5% in the end-tidal enflurane concentration administered to the clonidine patients.

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TITLE: PERIOPERATIVE MYOCARDIAL ISCHEMIA IN CAROTID ENDARTERECTOMY.

AUTHORS: G. Landesberg MD, L.A. Eidelman MD, Y. Erel MD, H. Anner MD, J. Assaf MD, P. Halimi MD, M.H. Luria MD, M.S. Gotsman MD, Y. Berlatzky MD, S. Cotev MD.
AFFILIATION: Dept's of Anesthesia, Nuclear Medicine, Cardiology and Vascular Surgery, Hebrew University - Hadassah Medical School, Jerusalem, Israel 91120.

The incidence of perioperative myocardial ischemia (PMI - defined as downsloping or horizontal ST segment depression of >1mm, lasting >1 min) and its correlation to hemodynamic changes and cardiac outcome was investigated in 18 consecutive patients [mean age 65.8±9.7(SD), F/M=6/12] undergoing carotid endarterectomy (CEA) under cervical block anesthesia (bupivacaine 0.5%). Twelve pts had preop evidence of IHD either by history, pathological thallium scan or coronary angiography. Following informed consent, all pts had 3 channel (modified aVF, V_4, V_5) ECG Holter monitoring, starting 24h preop and continued for 46±4.0h. All had continuous intra-arterial BP monitoring during and after (24h) operation, and received i.v. nitroglycerin 0.5-5mcg/kg/min - titrated according to BP. Postoperative cardiac course was evaluated by symptoms, daily ECG and cardiac enzymes until discharge from hospital.

Results: One patient was excluded from ST analysis due to complete LBBB. There were a total of 44 PMI episodes, all of which occurred in 12 pts. Twelve preop PMI's occurred in 7 pts, and 32 either at carotid declamping or postop (9 pts). PMI was not detected during surgery before carotid declamping when BP was relatively high. BP usually decreased immediately with carotid declamping (from mean BP 118±14 to 102±22 torr, p=0.005), or upon arrival to PACU (a further decrease to 86±13 torr p=0.003). No significant changes in HR occurred at these periods (79±19, 73±20 & 81±15, respectively). A group of 5 pts exhibited prolonged (>1 hour) ST depressions. In 3/5 pts ST depression started with carotid declamping, and a marked fall in systolic BP (48-50%). This decrease in SBP was the largest in the entire group. In 2/5 pts ST depression occurred upon arrival to PACU. These 5 pts also displayed 25/32 (78%) of all intra and postop PMI's. Moreover, all postop clinical cardiac events occurred in 3 of these 5 pts: 1 myocardial infarction (following the prolonged and severe ST depression) and 2 transient (<48h) unstable angina. The other 2/5 pts were asymptomatic.

Conclusions: In CEA under regional anesthesia, critical myocardial ischemia tends to occur during declamping and immediately after surgery, correlated with significant decreases in BP. These events may be associated with worse postoperative cardiac outcome.