Title: **NICARDIPINE BUT NOT ENALAPRIL PREVENTS RENAL HEMODYNAMIC ALTERATION DURING INFRARENAL AORTIC CROSS-CLAMPING**

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Infrarenal aortic cross-clamping during abdominal aortic surgery induces renal vasoconstriction, decreases Effective Renal Plasma Flow (ERPF) and Glomerular Filtration Rate (GFR) (1). Treatments with preoperative Enalapril (E), a converting enzyme inhibitor, or peroperative Nicardipine (N), a calcium antagonist, have been tested to assess renin-angiotensin system or calcium channel involvement during infrarenal aortic surgery.

After institutional approval and informed consent, 24 patients (40 to 73-yrs-old) were randomly divided into 3 groups: control (n=8), enalapril (n=8), nicardipine (n=8). E group received E (20mg/bid, PO), 2 days before surgery and placebo during surgery. N group received preoperative placebo and N (2mg bolus, then 2 mg/h) at the time of incision. C group received pre- and peroperative placebos. Anesthesia consisted of flunitrazepam, fentanyl, pancuronium and ± droperidol in the 3 groups. Sytemic (Swan Ganz and radial artery cathters) and renal (ERPF and GFR as assessed by Hippuran-I131 and DTPA-Tech99 clearances) hemodynamics were periodically recorded: 1- before aortic clamping (preclamp), 2- during aortic clamping (perclamp), 3- after aortic declamping (postclamp).

		Preclamp	Perclamp	Postclamp
HR b min	C	69.0±5.1	60.4±3.7	61.1±4.3
	E	80.0±5.4	66.3±4.0	75.1±3.5
	N	* 85.9±2.4	75.5±6.2	*83.5±7.0
BP mmHg	C	85.4±4.1	84.4±3.4	84.9±5.0
	E	82.3±1.7	84.1±2.8	87.5±3.4
	N	89.9±2.4	86.4±3.7	83.9±3.1
PCWP mmHg	C	11.6±1.6	10.1±1.3	9.4±1.5
	E	14.0±1.0	12.0±1.1	13.0±1.5
	N	13.3±1.4	13.2±1.6	12.2±2.0
CO l/min/m	C	7.6±0.7	5.1±0.4	5.6±0.4
	E	6.5±0.9	5.2±0.4	6.0±0.4
	N	8.2±0.6	4.5±0.3	6.8±0.5
ERPF ml/min	C	227.3±62.4	185.4±44.7	317.5±61.0
	E	433.0±193	268.5±61.8	352.5±51.8
	N	336.2±90.4	352.4±65.2	260.4±43.4
GFR ml/min	C	71.4±20.3	40.8±8.5	59.6±9.0
	E	74.4±15.3	49.1±10.7	64.0±7.4
	N	63.2±11.0	* 78.9±14.3	71.6±10

m±sem, * p<0.05 between groups

Thus, renin-angiotensin seems to be not involved in renalfunction alteration during infrarenal aortic cross clamping. The beneficial effect of low doses of N might be accounted for by the abolition of renal autoregulation (2), which appears to be impaired during aortic cross-clamping.

References: 1) Anesthesiology 61:394,1984.
2) Circ Res 59:483, 1986.

TITLE: BOLUS DOSES OF ESMOLOL FOR THE PREVENTION OF PERIO-OPERATIVE HYPERTENSION AND TACHYCARDIA IN CARDIOVASCULAR SURGERY PATIENTS.

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Introduction Endotracheal intubation frequently results in marked elevation in heart rate (HR) and/or blood pressure. The objective is to demonstrate the effectiveness of bolus doses of esmolol (E), a short acting β-selective blocking agent, to prevent intubation induced elevations in HR and mean arterial pressure (MAP) in patients undergoing peripheral vascular (PV) procedures who are at risk to develop myocardial ischemia.

Methods With Ethics Committee approval, 27 PV (ASA II, III) patients were studied in a double blind manner and were randomly divided into 3 groups: placebo (P), esmolol 100 mg (E-100) or esmolol 200 mg (E-200) by bolus, 90 seconds prior to induction with thiopental. HR and direct MAP were observed at baseline and post-induction (PI) at 0.5, 1.5, 2.5, 3.5, 4.5 and 5.5 minutes. ECG leads II and V_5 (Monitor) and Holter leads II and CB_5 were used to monitor for myocardial ischemia. Analysis of covariance was utilized to determine significance difference between the 3 groups.

Results Mean HR's PI were lower (p<.01) in each of the E groups compared to P. The PI HR was 44% above baseline in the P group, 9% below baseline in the E-100 group (p<.01 vs P), and 3% below baseline in the E-200 group (p<.01 vs P). Mean HR's were consistently lower in the E groups than in the P group for up to 4.5 minutes PI. No significant differences were detected between the esmolol groups. Ischemia was detected in 6 patients: P=3, E-100=1 and E-200=2.

Conclusion In PV patients, bolus doses of 100 or 200 mg of E, 90 seconds prior to intubation, effectively blunts maximum HR responses to intubation. Mean PI MAP(s) were significantly lower after E compared to P. No difference was detected in the incidence of hypotension. bradycardia, arrhythmias, or ischemia.

HEART RATE	PLAC	E100	E200
Baseline	77	78	77
Post-induction	83	73	75
Post-intubation maximum	111	86 **	88 **
0.5 min post-intubation	108	81 **	80 **
1.5 min post-intubation	101	76 **	82 **
2.5 min post-intubation	96	77 **	80 **
3.5 min post-intubation	93	79 *	76 **
4.5 min post-intubation	88	78	72 *
5.5 min post-intubation	83	78	73
MEAN ARTERIAL PRESSURE			
Baseline	100	99	103
Post-induction	168	86 **	86 **
Post-intubation maximum	152	130 **	125 *
0.5 min post-intubation	144	107 **	112 *
1.5 min post-intubation	149	109 **	117 **
2.5 min post-intubation	135	109 *	117
3.5 min post-intubation	117	110	107
4.5 min post-intubation	106	96	101
5.5 min post-intubation	96	91	96

* P ≤ .05 (compared to placebo)
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