

**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  $\pm$  droperidol in the 3 groups. Sytemic (Swan Ganz and radial artery catheters) and renal (ERPF and GFR as assessed by Hippuran-1131 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	C	69.0 $\pm$ 5.1	60.4 $\pm$ 3.7	61.1 $\pm$ 4.3
	E	80.0 $\pm$ 5.4	66.3 $\pm$ 4.0	75.1 $\pm$ 3.5
	N	* 85.9 $\pm$ 2.4	75.5 $\pm$ 6.2	* 83.5 $\pm$ 7.0
BP	C	85.4 $\pm$ 4.1	84.4 $\pm$ 3.4	84.9 $\pm$ 5.0
	E	82.3 $\pm$ 1.7	84.1 $\pm$ 2.8	87.5 $\pm$ 3.4
	N	89.9 $\pm$ 2.4	86.4 $\pm$ 3.7	83.9 $\pm$ 3.1
PCWP	C	11.6 $\pm$ 1.6	10.1 $\pm$ 1.3	9.4 $\pm$ 1.5
	E	14.0 $\pm$ 1.0	12.0 $\pm$ 1.1	13.0 $\pm$ 1.5
	N	13.3 $\pm$ 1.4	13.2 $\pm$ 1.6	12.2 $\pm$ 2.0
CO	C	7.6 $\pm$ 0.7	5.1 $\pm$ 0.4	5.6 $\pm$ 0.4
	E	6.5 $\pm$ 0.9	5.2 $\pm$ 0.4	6.0 $\pm$ 0.4
	N	8.2 $\pm$ 0.6	4.5 $\pm$ 0.3	6.8 $\pm$ 0.5
ERPF	C	227.3 $\pm$ 62.4	185.4 $\pm$ 44.7	317.5 $\pm$ 61.0
	E	433.0 $\pm$ 193	268.5 $\pm$ 61.8	352.5 $\pm$ 51.8
	N	336.2 $\pm$ 90.4	352.4 $\pm$ 65.2	260.4 $\pm$ 43.4
GFR	C	71.4 $\pm$ 20.3	40.8 $\pm$ 8.5	59.6 $\pm$ 9.0
	E	74.4 $\pm$ 15.3	49.1 $\pm$ 10.7	64.0 $\pm$ 7.4
	N	63.2 $\pm$ 11.0	* 78.9 $\pm$ 14.3	71.6 $\pm$ 10

m $\pm$ sem, \* p<0.05 between groups  
 Thus, renin-angiotensin seems to be not involved in renal function 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  $\beta$ -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<sub>5</sub> (Monitor) and Holter leads II and CB<sub>5</sub> 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	E100	E200
Baseline	77	77
Post-induction	83	75
Post-intubation maximum	131	86 **
0.5 min post-intubation	108	81 **
1.5 min post-intubation	101	78 **
2.5 min post-intubation	96	77 **
3.5 min post-intubation	93	79 *
4.5 min post-intubation	88	78
5.5 min post-intubation	81	72

  

MEAN ARTERIAL PRESSURE	E100	E200
Baseline	100	99
Post-induction	108	86 **
Post-intubation maximum	132	120 **
0.5 min post-intubation	144	107 **
1.5 min post-intubation	149	109 **
2.5 min post-intubation	135	109 *
3.5 min post-intubation	117	110
4.5 min post-intubation	106	96
5.5 min post-intubation	96	91

\* P  $\leq$  .05 (compared to placebo)  
 \*\* P  $\leq$  .01 (compared to placebo)