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TITLE: CLONIDINE DOES NOT INFLUENCE ANESTHETIC DRUG REQUIREMENTS AND HEMODYNAMIC RESPONSE DURING AORTIC SURGERY UNDER BALANCED ANESTHESIA

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The efficiency of clonidine (CLON) in improving the hemodynamic stability, in decreasing drug requirements and in blunting the humoral response during aortic surgery remains controversial (1,2).

Methods: With Ethical Committee approval and informed consent, we studied 20 ASA II-III patients scheduled for elective aortic abdominal grafting surgery under general anesthesia (thiopental / isoflurane / nitrous oxide / oxygen / fentanyl). In this double-blind placebo-controlled study, they received 90 minutes (min) before induction oral midazolam 7.5-15 mg together with oral CLON 5.6 ± 0.6 µg/kg (n=10) or placebo (PLAC) (n=10). A same dose of CLON or PLAC was administered 4 h later during surgery. Arterial blood samples and hemodynamic data were collected 15 min before aortic cross clamping (BXC), 30 min after cross clamping (AXC) and 30 min after bilateral aortic cross clamp release (AXCR). These were assayed for epinephrine (E), norepinephrine (NE), plasma renin activity (PRA) and atrial natriuretic factor (ANF). Hemodynamic profiles were also obtained prior to induction as well as 1,3,5, and 10 min after endotracheal intubation, upon skin incision and closure, 15 min before BXC, 2,10, 20 and 30 min after AXC and AXCR respectively. Values are expressed as mean ± SE. Comparisons within the same group were assessed by ANOVA for repeated measures followed by Duncan's multiple range test. Student's unpaired t-test was used for comparisons between groups. Significance was defined as p<0.05.

Results: Demographic and surgical data were equal in both groups. In the CLON group, mean doses of fentanyl (1.95 ± 0.29 versus 2.6 ± 0.27 µg/kg/h) and of isoflurane (0.14 ± 0.01 versus 0.19 ± 0.02 ml/kg/h) were lower than in the PLAC group. These differences were statistically not significant. HR, MAP, PAP, CVP, PCWP, CI, PVR and SVR were not different between groups throughout the whole study period. NE levels were lower after CLON (p<0.05 during clamping). Both NE and E were decreased during clamping and tended to recover thereafter. Although initial PRA tended to be lower under CLON, no difference was found after clamping. Plasma ANF tended to decrease after clamping, particularly in the absence of CLON.

Conclusion: In infrarenal aortic surgery performed under balanced anesthesia, preoperative administration of CLON decreased plasma catecholamine levels, but failed to offer real hemodynamic benefits.

References: 1. Anesthesiology 71: 178-187, 1989.
2. Acta Anaesthesiol Scand 34: 132-7, 1990.

Hormonal effects of aortic cross clamping and CLON treatment are summarized in table below.

		E pg/ml	NE pg/ml	PRA fmol/ml	ANF ng/ml/h
BXC	plac	202±55	560±64	2.5±0.5	48±7
	clon	272±129	470±68	1.8±0.6	48±4
AXC	plac	79±24	8409±34	2.6±0.5	41±5
	clon	84±23	8306±32*	3.3±2	46±5
AXCR	plac	157±59	448±39	2.3±0.3	36±3
	clon	318±117	373±61	3.7±2.5	41±7

* p<0.05 from plac values after cross clamping (AXC)
& p<0.05 from BXC value within the same group

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Title: ANESTHETIC INDUCTION OF HIGH-RISK PATIENTS: EFFECTS OF DESFLURANE ON HEMODYNAMICS AND ECHOCARDIOGRAPHIC MEASURES OF ISCHEMIA

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Introduction: Induction of anesthesia carries a substantial risk potentially associated with marked changes in hemodynamics. However, the potential to produce ischemia in high-risk patients during induction has not been rigorously studied. To determine the effects of two new anesthetic agents (desflurane [D], sufentanil [S]), we compared the hemodynamic (HEMO) and transthoracic echocardiographic (TTE) effects of D and S during anesthetic induction for elective coronary artery surgery.

Methods: With institutional approval, we studied 150 patients with an ejection fraction ≥0.30. Premedication included the patient's cardiac medications, morphine and midazolam. Anesthesia was induced with thiopental IV 2-4 mg/kg, vecuronium 0.1 mg/kg IV, and either D 1-2 MAC end-tidal (ET) or S 5-10 µg/kg IV via controlled ventilation. D was maintained at 1 MAC ET for ≥ 5 min prior to intubation. The D and S were titrated to maintain heart rate (HR) < 120% and systolic arterial pressure (SBP) ± 20% of ward values. If D or S were insufficient, vasoactive drugs were given. HEMO and TTE measurements were made at three intervals: baseline (B), end induction (IND), and 1 min after intubation (IT). HEMO measurements included systemic and pulmonary arterial pressures; HR; cardiac index (CI); ET anesthetic concentration. TTE (short-axis-left ventricle) was assessed in 80 consecutive patients (45 interpretable). The wall motion of each of the four segments was graded: 0-normal to 4-dyskinesis.

TTE ischemia was defined by wall motion worsening ≥2 grades, lasting ≥1 min. TTE ejection fraction area (EFA) was determined quantitatively at the above intervals. Statistical analysis included t-test and chi-square.

Results: Baseline HEMO measurements were similar between D and S groups. However, significant increases (percent change from baseline) were found at IND and IT in the D group.

Table 1		Baseline	IND (% of B)	IT (% of B)
SBP (mmHg)	DES	125±24	8%±20 *	8%±20 *
	SUF	126±20	-8%±10	-10%±20
HR (bpm)	DES	63±11	20%±20 *	20%±20 *
	SUF	62±11	-3%±10	-6%±10
PAS (mmHg)	DES	28±6	30%±30 *	40%±30 *
	SUF	31±8	7%±20	7%±20
PCWP (mmHg)	DES	13±4	30%±30 *	30%±30 *
	SUF	13±4	-9%±40	-10%±40
CI (l/min/m²)	DES	3.0±1.0	-9%±20	-6%±20
	SUF	3.0±0.5	-9%±10	-8%±20
EFA (%)	DES	52%±9	-20%±20**	-20%±20***
	SUF	56%±12	0.3%±10	0.3%±10

(Mean ±SD); *p < 0.0001, **p<0.0006, ***p< 0.001

The incidence of TTE ischemia tended to occur more frequently in the D group 4/26 (15%) as compared to the S group 0/19 (0%) (P = 0.20). The baseline mean segmental wall motion score (SWMS) was: D 0.1±0.2 vs. S 0.3±0.5 (P= 0.02); with a mean increase in SWMS at IND: D 0.3±1.0 vs. S 0.03±0.1 (P=0.04), and increase at IT: D 0.4±1.0 vs. S 0.04±0.01 (P=0.025).

Discussion: Our findings suggest that anesthetic induction with D: 1) increases PAS and PCWP with a decrease in SVI (CI unchanged), 2) decreases EFA, and 3) increases SWMS. Thus, D, used as the primary anesthetic agent for induction in patients with coronary artery disease, may be associated with left ventricular dysfunction and/or ischemia.