

Title : ABSENCE OF PROTECTIVE RENAL EFFECTS OF EPIDURAL ANESTHESIA IN PATIENTS UNDERGOING AORTIC ABDOMINAL SURGERY

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Introduction. The depressive effects of infrarenal aortic cross clamping (IRACC) on renal hemodynamics under general anesthesia in man have recently been documented (1). The sustained renal hypoperfusion observed after the release of the aortic clamp could be related to a persistent renin or catecholamine induced renal vasoconstriction. The purpose of this study is to evaluate the possible protective effects of a sympathetic blockade, produced by an epidural anesthesia combined with a light general anesthesia, on renal perfusion and function during and after IRACC.

Method. 8 ASA II-III patients with a mean age of 60 ± 9 yr (x ± SD) consented to participate in the study which obtained institutional approval. After hydration (15 ml/kg of Ringer's lactate), a lumbar L2-L3 epidural catheter was inserted and plain bupivacaine 0.5% injected until adequate sensory anesthesia (T₆ level) was obtained. General anesthesia was then induced with thiopental (3-5 mg/kg), fentanyl (2 µg/kg) and pancuronium (0.1 mg/kg) and maintained with N₂O 70% in O₂, the patients being ventilated at a PaCO₂ of 35-40 mmHg. Additional doses of bupivacaine 0.5% and pancuronium were injected as required clinically. In all patients heart rate (HR), mean arterial pressure (MAP) using a radial catheter, central venous pressure (CVP) and urinary output (UO) were continuously monitored. In five patients cardiac output (CO) and pulmonary capillary wedge pressure (PCWP) were measured using a triple lumen catheter. Glomerular filtration rate and cortical renal plasma flow were measured using standard clearances of ⁵¹Cr EDTA (C_{EDTA}) and ¹²⁵I Hippuran (C_{HIP}). These labeled compounds were injected as a bolus at a dose of 60 µCi and 30 µCi respectively, followed by a continuous infusion of ⁵¹Cr EDTA (72 µCi) and ¹²⁵I HIP (36 µCi) in 500 ml of mannitol 20%, which was administered at a rate of 100 ml/h throughout the study in order to obtain stable blood levels and an adequate UO. After an equilibrium period of 67 ± 21 min, urine was collected: a) before IRACC for 63 ± 21 min, b) during IRACC for 48 ± 15 min, c) after IRACC for 64 ± 8 min. At midway of each period of urine collection, left renal vein blood, obtained by direct puncture and arterial blood were sampled in order to calculate extraction fraction of HIP (E_{HIP}) and renal clearances, and hemodynamic variables were determined. Administration of Ringer's lactate and blood was adjusted to maintain the filling pressures and hematocrit at pre-clamp levels. At the end of the surgery, all patients were immediately extubated and sensory anesthesia level, which was retested, remained always at T₆ or above. E_{HIP} was obtained by dividing arterial HIP - renal venous HIP levels by arterial HIP concentration. Total renal plasma flow was determined by dividing C_{HIP} by E_{HIP}. Renal blood flow (RBF), renal fraction of CO (RF) and systemic and renal vascular resistances (SVR, RVR) were calculated using standard formulas. Filtration fraction

(FF) was derived from C_{EDTA} and C_{HIP}. All renal data are reported for a body surface area of 1.73 m². The data were compared with one-way analysis of variance and test of Scheffé, p < 0.05 being considered significant.

Results. The results are summarized on table 1. and 2. Besides the significant increase in SVR during IRACC the systemic hemodynamics remained stable. Important impairment of renal hemodynamics was however observed during IRACC without recovering in the early post-clamp period. The creatinine clearance (C_{Cr}) measured 3 days after surgery (78 ± 21 ml/min) was significantly decreased (p < 0.05) when compared with the preoperative value (90 ± 19 ml/min).

Discussion. The high values of renal perfusion measured in the pre-clamp period are most probably related to the T₆ level of sympathetic blockade associated with unimpaired systemic hemodynamic variables. However IRACC produced an important decrease in RBF (-55%) and a dramatic increase in RVR (+159%), persisting during the first hour after the release of the aortic clamp. Because of the complete renal sympathetic blockade these changes cannot be attributed to increased sympathetic activity.

Conclusion. Since with renal sympathetic blockade, renal perfusion and function during and after IRACC were not different from data previously reported under general anesthesia alone (1), and since significant decrease in postoperative C_{Cr} was observed, we conclude that epidural anesthesia administered during aortic abdominal surgery does not prevent the impairment in renal perfusion and function induced by IRACC.

TABLE 1 : Systemic hemodynamic variables (x ± SD)

PERIOD	HR n=8	MAP (mmHg) n=8	CVP cmH ₂ O n=8	PCWP (mmHg) n=5	CO (l/min) n=5	SVR (units) n=5	RF n=5
PRE	75	85	7	9	6.4	982	0.31
CLAMP	± 14	± 15	± 3	± 3	± 1.4	± 140	± 0.06
PER	69	93	7	8	4.5	1503**	0.22
CLAMP	± 8	± 15	± 3	± 4	± 0.9	± 242	± 0.08
POST	66	92	8	9	5.4	1254	0.19
CLAMP	± 8	± 9	± 3	± 2	± 0.9	± 216	± 0.07

Statistical difference from pre-clamp data : ** p < 0.01

TABLE 2 : Renal hemodynamics and function (x ± SD)(n = 8)

PERIOD	UO (ml/min)	C _{EDTA} (ml/min)	C _{HIP} (ml/min)	E _{HIP}	RBF (ml/min)	FF	RVR (units)
PRE	4.62	124	798	0.69	1884	0.16	3792
CLAMP	± 1.87	± 41	± 273	± 0.03	± 687	± 0.02	± 1592
PER	3.65	79	403*	0.74	860	0.20	10094
CLAMP	± 1.46	± 33	± 178	± 0.05	± 415	± 0.02	± 5875
POST	3.49	81	388**	0.74	845**	0.22**	10029
CLAMP	± 1.02	± 27	± 163	± 0.06	± 373	± 0.04	± 5505

Statistical difference from pre-clamp data : * p < 0.05, ** p < 0.01

Reference :

1. Gamulin Z et al : Anesthesiology (in press)