Title : HEMODYNAMIC EFFECTS OF ANESTHESIA AND SURGERY IN RENAL HYPERTENSIVE PATIENTS RECEIVING LARGE DOSES OF β - RECEPTOR ANTAGONISTS.

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Patients with severe hypertension due to acute renal ischemia, under treatment with huge doses of β receptor antagonists, present the anesthesiologist with considerable problems. Ten such patients aged between 12 and 62, with pre-operative arterial pressure averaging 232 ± 8 (SEM) mm Hg systolic, 136 ± 6 mm Hg diastolic, despite receiving propranolol (or equivalent doses of oxprenolol or labetalol) in doses ranging from 10 to 36 mg/kg/day in addition to other antihypertensive therapy, were studied before, during and after anesthesia and renal arterial bypass surgery. All patients had accurate diagnosis of unilateral renal artery occlusion by angiography, selective renal vein renin assays, a positive response (decrease of arterial pressure) to saralasin, and biochemical indices of renal function.

Despite the elevated systolic and diastolic arterial pressures (SAP : 201 ± 10 , DAP : 104 ± 6 mm Hg) and the β-receptor antagonist therapy, cardiac outputs (5.05 ± 0.51 l/min/70kg) in the awake, morphine-scopolamine premedicated patients were well within normal limits for normotensive patients of the same age[1]. These were associated with mildly elevated (10.7 ± 1.1 mm Hg) pulmonary arterial wedge pressures (PAWP) , and aortic dP/dt (1087 ± 90 mm Hg /sec) values in the normal range.

Anesthesia was induced with fentanyl 10 mcg/kg intravenously, Althesin 6-12 mg and pancuronium 0.1 mg / kg , and maintained with nitrous oxide 66% and oxygen supplemented with fentanyl (3 mcg/kg/hr) with volume controlled ventilation in circle system without a soda-lime absorber to maintain a normal $Paco_2$.

During established anesthesia before surgery systolic arterial pressure (SAP) decreased to 122 ± 12 mm Hg (- 39%) associated with a 17% decrease of cardiac output (4.21 l/min/70kg) and a decrease of PAWP to 8.1 ± 0.8 mm Hg.

During surgery, complete or partial cross - clamping of the aorta caused modest increases of SAP (+ 25 mm Hg), systemic vascular resistance (+ 550 dyn sec cm^{-5}) and PAWP (+ 1.0 mm Hg) and a decrease of cardiac output (- 0.3 l/min/70kg).

Two hours postoperatively, all patients were awake and not complaining of pain. At this time their SAP and DAP averaged 183 ± 8 mm Hg and 97 ± 6 mm Hg respectively, and cardiac output was 4.59 ± 0.40 l/min/70kg . PAWP (8.7 ± 1.0 mm HG) and central venous pressure (5.0 ± 0.8 mm Hg) were lower than the preoperative values. An average of 1100 ml of whole blood had been transfused to compensate for measured blood loss.

Laryngoscopy and endotracheal intubation produced negligible increases of SAP (+ 4 mm Hg), heart rate (+ 3 beats per min) and PAWP (+ 1.0 mm Hg), but the rate-pressure product increased by 7% (not significant). There were no dysrhythmias nor was there evidence of subendocardial ischemia in response to this stimulus [2].

Heart rate and aortic dP/dt responses to graded increments of isoproterenol were observed during surgery (4 hours after last dose of β-receptor antagonist), and in the ITU 24 and 48 hours post-operatively. To achieve a 20 beats/min increase of heart rate during surgery , an average dose of 440 mcg of isoproterenol was required. By 24 hours post-operatively a dose of 80 mcg was still necessary, and at 48 hours a dose of 40 mcg was needed.

These patients had markedly inhibited heart rate responses to atropine during surgery. Atropine 0.6 mg and 1.2 mg caused average increases of heart rate of only 2.3 and 7.2 beats/min respectively. Despite atropinisation, neostigmine consistently reduced heart rates to below 45 beats/min.

Discussion. Despite the pre-existing severe hyper - tension and therapy with huge doses of β-receptor antagonists, the hemodynamic responses of these patients to anesthesia and surgery were unremarkable and not dissimilar to those of hypertensive patients receiving much smaller doses of β- receptor antagonists [3]. Nevertheless, the markedly inhibited response to isoproterenol during surgery and the first 24 hours after is noteworthy, and indicates the duration of clinically important degrees of β-receptor blockade. More surprising was the lack of heart rate response to atropine, and the enhanced response to neostigmine, neither of which has been observed in patients taking lower doses of any β-receptor antagonist.

With careful intra- and post-operative monitoring of arterial pressure, heart rate, cardiac output and PA wedge pressure, patients with very severe hypertension, and those receiving huge doses of β -receptor antagonists can be safely managed during major surgery.

References

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