Hypertonic saline prehydration in patients undergoing transurethral resection of the prostate under spinal anaesthesia

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SUMMARY
Thirty-three patients undergoing elective transurethral resection of the prostate were allocated randomly to receive either 0.9% isotonic saline 7 ml kg\(^{-1}\) (16 patients), or 3% hypertonic saline 7 ml kg\(^{-1}\) (17 patients) as a preload before spinal anaesthesia. After spinal anaesthesia, the incidence of systolic arterial pressure < 75% of control value was greater in the normal saline group than in the hypertonic saline group. Also, the mean dose of phenylephrine required to maintain arterial pressure > 75% of the baseline value was significantly greater in the normal saline group than in the hypertonic saline group. (Br. J. Anaesth. 1994; 72: 227–228)

KEY WORDS
Anaesthetic techniques: subarachnoid. Complications. TURP syndrome.

In 1980, Velasco and co-workers demonstrated, in dogs subjected to severe haemorrhagic shock, that 7.5% saline infused in a volume equivalent to only 10% of the shed blood volume increased systemic pressure rapidly, restored cardiac output and yielded 100% long-term survival [1]. Beneficial results have been reported in patients suffering from hypovolaemic shock refractory to conventional treatment [2].

We have compared the influence of prehydration with hypertonic saline or isotonic saline on the haemodynamic changes and serum sodium concentrations in patients undergoing transurethral resection of the prostate (TURP) under spinal anaesthesia.

METHODS AND RESULTS
The investigation was conducted in 33 male patients (ASA I–III, aged 42–87 yr) undergoing TURP. The investigation was approved by the institutional Ethics Committee.

All patients were premedicated with atropine 0.4 mg i.m. and diazepam 5 mg orally. Central venous pressure (CVP) was monitored via a 16-gauge catheter inserted in the right antecubital fossa. Patients were monitored also by ECG (V\(_6\)), and an automated, non-invasive arterial pressure monitor (Omega 1400). Baseline arterial pressure (AP), heart rate (HR) and CVP were recorded and a baseline venous blood sodium concentration was measured.

Patients were allocated randomly to two groups. Group I comprised 16 patients who received as a preload 0.9% normal saline 7 ml kg\(^{-1}\) (equivalent to 310 mosmol litre\(^{-1}\)). Group II comprised 17 patients who were given 3%, hypertonic saline 7 ml kg\(^{-1}\) (equivalent to 1025 mosmol litre\(^{-1}\)). The crystalloid solutions were infused via the peripheral cannula over 10 min. Following prehydration, AP, HR and CVP were recorded and serum sodium concentration was measured. Spinal anaesthesia was performed at the L3–4 interspace with a 22-gauge spinal needle using 0.3% amethocaine 4 ml and the level of anaesthesia was determined by pinprick. AP, HR and CVP were recorded at 5-min intervals for 1 h, then every 10 min for 2 h in the operating room and the recovery room. Serum concentrations of sodium after spinal anaesthesia were measured every 1 h in both groups. The smallest systolic AP achieved after spinal anaesthesia was recorded and the mean of these values compared with control values and with those obtained after prehydration. Also, the mean CVP and HR values recorded at the time of at least AP were compared with the corresponding control values and the values after prehydration.

Hypotension after spinal anaesthesia was defined as a decrease in systolic AP to less than 75% of the baseline value and was treated by an i.v. bolus of phenylephrine 100 μg. Phenylephrine was repeated at 5-min intervals until the arterial pressure was increased to > 75% of baseline. The number of patients developing hypotension and the mean dose of phenylephrine required for treatment were noted. The mean dose of phenylephrine was calculated as the total dose of phenylephrine administered divided by the total number of patients treated.

All data other than the incidence of hypotension are expressed as mean (SD). Statistical analysis was by Student's \(t\) test and ANOVA for repeated measurements for quantitative data. Chi-square analysis with Yates' corrections was used for qualitative data. \(P < 0.05\) was considered significant.

Age, weight, height of sensory block, duration of surgery and total volume of urological irrigating fluid (sorbitol, mannitol solution) did not differ...
significantly between the two groups (table I). Mean control systolic AP and heart rate values were not significantly different between the groups and did not show a significant change after prehydration. After spinal anaesthesia, AP and heart rate decreased significantly in both groups. The maximal decrease in arterial pressure was observed at 31.0 (19.0) min in group I and at 40.0 (16.0) min in group II. The mean value of the smallest systolic AP after spinal anaesthesia was significantly less in the isotonic saline group than in the hypertonic saline group. In addition, the incidence of systolic arterial pressure values <75 % of control value was greater in group I than in group II. The mean dose of phenylephrine required to treat hypotension was significantly greater in the isotonc saline group.

Prehydration with hypertonic saline resulted in a significant increase in CVP and less hypotension after spinal anaesthesia compared with prehydration with isotonic saline. The incidence of systolic arterial pressure values <75 % of control value was greater in group I than in group II. Also, the mean dose of phenylephrine required to treat hypotension was significantly greater in the isotonic saline group.

The improved maintenance of arterial pressure in the hypertonic saline group cannot be attributed to blood loss (table I), to preoperative fluid administration (same volume (7 ml kg⁻¹) of hypertonic saline or isotonic saline) or to different doses of phenylephrine (less phenylephrine administered in the hypertonic saline group).

Hypotension after spinal anaesthesia results from functional sympathetic denervation, not only at the arterial and arteriolar circulation, but also at the large veins and venules. Venodilatation can increase significantly the venous capacitance with a consequent decrease in venous return and cardiac output [3]. The mechanism by which hypertonic saline counteracts haemodynamic changes after spinal anaesthesia may be attributed primarily to the instantaneous mobilization of endogenous fluid along the osmotic gradient from the intracellular to the extracellular space [4]. In addition, hypertonic saline may result in direct myocardial stimulation and venoconstriction [1, 2, 5].

Patients undergoing TURP may develop dilutional hyponatraemia secondary to systemic absorption of the irrigation fluid. The degree of absorption is related to the time of resection, degree of bleeding and type, volume and pressure of irrigating fluid [5]. Prehydration with hypertonic saline may decrease the degree of dilutional hyponatraemia.

**REFERENCES**