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Haemodynamic effects of subarachnoid block in elderly patients

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Summary

We have studied the haemodynamic effects of subarachnoid block in elderly patients. Thirty patients were undergoing elective transurethral surgery and 18 non-elective orthopaedic surgery, predominantly fractured neck of femur. Systolic arterial pressure (SAP) was measured by automated oscillotonometry, central venous pressure (CVP) by manometer and cardiac index (CI), stroke index (SI) and heart rate (HR) by transthoracic electrical bioimpedance. Systemic vascular resistance index (SVRI) was derived. SAP decreased by more than 25% in 33 patients and SVRI showed similar decreases ($P = 0.0001$). CVP decreased (2.5 (SD 1.5) cm H_2O) in all patients. CI was unaffected because a decrease in SI in some patients (13 (19)%; $P = 0.01$) was compensated for by an increase in HR (13 (13)%; $P = 0.01$). Decreases in SAP of 25% were treated initially with colloid solution 8 ml kg^{-1}, which restored SAP in 19 patients. CVP, SI and HR were all restored to baseline values, however, SVRI was decreased further ($P < 0.05$). Fourteen patients required additional treatment with metaraminol which restored SVRI to baseline values. Patients with systolic hypertension were more likely to require treatment with metaraminol ($P = 0.04$). (*Br. J. Anaesth.* 1994; **73**: 464–470)

Key words

Anaesthetic techniques, subarachnoid Cardiovascular system, effects. Measurement techniques, transthoracic electrical impedance.

Subarachnoid block is used frequently for anaesthesia for lower abdominal and lower limb surgery. Hypotension is common during subarachnoid block and episodes of severe hypotension can be detrimental to the patient, especially the elderly. Current practice is to treat systolic arterial pressure (SAP) of less than 80 mm Hg or a decrease of greater than 20–30% [1–6]. However, the haemodynamic changes that underlie the decrease in SAP have not been investigated fully [4].

The main factor causing hypotension is sympathetic block, which results in arterial and venous vasodilatation [7]. Current accepted practice is to administer a vasopressor to counteract arterial dilatation and fluids to compensate for venous dilatation [1–6]. However, at present there is no consensus on how these two methods should be combined to give the best results, partly because of the lack of good data on the primary haemodynamic effects of subarachnoid block [4]. Published work to date has mostly involved obstetric patients [4] and the few studies that have examined elderly patients examined only the effects of treatment [2,3,5]. Green [7] has reviewed the current literature, but his conclusions were based mainly on studies in young healthy adults.

Our objective was to determine the haemodynamic effects of subarachnoid block in elderly patients. Arterial pressure, central venous pressure (CVP) and cardiac output were recorded during the first 30 min of subarachnoid block. Transthoracic electrical bioimpedance (TEB) was used to measure cardiac output non-invasively. Two groups of patients were studied, one undergoing elective transurethral surgery and the other non-elective orthopaedic surgery.

Patients and methods

We studied 48 ASA II or III elderly Chinese patients who required subarachnoid block for elective transurethral surgery or non-elective surgery for predominantly traumatic orthopaedic problems, mainly internal fixation of the neck of the femur. Local Ethics Committee approval and informed consent from each patient were obtained. We included patients with untreated isolated systolic hypertension and hypertension treated with angiotensin converting enzyme inhibitors, calcium channel blockers, methyldopa, alpha blockers or thiazide diuretics. Our definition of isolated systolic hypertension was a baseline SAP greater than 160 mm Hg. Patients were excluded if the New York Heart Association dyspnoea class was III or IV, heart rate was irregular or if they were receiving beta adrenergic receptor blockers or digoxin.

Oral diazepam 5–10 mg was given 1 h before surgery as premedication. After the patient arrived in the induction room, 10 min were allowed for stabilization before obtaining haemodynamic data.

SAP and mean arterial pressures were measured every 1 min using an automated oscillotonometer (Dinamap 1846SX, Critikon, Florida, USA) and data were recorded on the attached printer. CVP measurements were made using a manometer at-

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tached to a 16-gauge cannula inserted into the right internal jugular vein, with the zero taken as the mid-axillary line and the 4–6th intercostal space. Cardiac output was monitored non-invasively by TEB using the BoMed NCCOM3-R7S (BoMed Medical Manufacturing Ltd, Irvine, CA, USA). This was connected to the patient according to the manufacturer's instructions, using four neck and four lower thoracic electrodes with two chest electrodes for detecting the ECG [8]. The method used to derive cardiac output has been presented previously [9, 10]. Data were indexed to the patient's body surface area and the average of 16 beats recorded. The variables recorded were cardiac index (CI), stroke index (SI) and heart rate (HR). Using custom-made software, these data were recorded continuously onto a spreadsheet file (Microsoft Excel, version 4.0, Microsoft Corporation, USA) by an IBM-compatible lap-top computer.

Data from the Dinamap and BoMed were collected for 3–5 min before the patient was turned to the lateral position for subarachnoid block and continued for at least 25 min afterwards. CVP measurements were made before turning the patient laterally and at 5-min intervals after starting the subarachnoid block.

Subarachnoid puncture was performed with a 22-gauge spinal needle at the L3–4 or L4–5 interspace with the patient in the lateral position. Then, 0.5% bupivacaine 2.5–3.0 ml (Marcain Spinal 0.5% heavy, Astra, North Ryde, NSW, Australia) was injected over 10 s and the patient returned to the supine position and kept horizontal for the duration of the study. The level of sensory loss to pinprick was assessed 30 min after subarachnoid block by an independent observer who was blinded to the treatment regimen.

If SAP decreased by 25% of baseline on two consecutive measurements, patients received an i.v. infusion of colloid solution 8 ml kg^{-1} given over 3–4 min (Polygeline–Haemaccel, Behring, Marburg, Germany). If SAP remained less than 25% of baseline after administration of the colloid solution, patients received a bolus dose of metaraminol 0.25 mg followed by an infusion of 5 mg h^{-1}. The infusion rate was adjusted to maintain SAP between 75 and 100% of baseline.

DATA ANALYSIS

A Macintosh LCIII computer was used for data analysis and statistics. Haemodynamic data, stored as spreadsheet files, were prepared for analysis using the program Microsoft Excel (version 4.0). The haemodynamic data collected before subarachnoid block were averaged to give baseline values. Subsequent TEB data were averaged to give mean values for each 1 min during subarachnoid block. Systemic vascular resistance index (SVRI) was calculated from the formula: SVRI = (mean arterial pressure − CVP) × 80/CI, where CVP, measured in mm Hg, was substituted for right atrial pressure.

Haemodynamic data from the transurethral and orthopaedic surgery patient groups were combined and patients were classified into three groups according to which treatment was given: (i) no treatment: SAP decreased by less than 25%; (ii) colloid effective: required colloid only; and (iii) colloid ineffective: required metaraminol in addition. Although the starting times for treatment differed between patients, these were aligned for the purpose of data presentation and analysis of the effects of treatment.

To determine the effect of administration of colloids, the time of starting colloid was treated as zero. Data collected 5 min before and 5 min after starting colloid were aligned at this zero point.

In order to compare the magnitudes of the haemodynamic changes after subarachnoid block and treatment, mean percentage changes for SAP, SVRI, CI, SI and HR and the numerical difference for CVP were used. In patients requiring no treatment, baseline data were compared with mean data for the 10–20-min period after the start of block. In patients receiving colloid, baseline data were compared with mean data for the 2 min before starting and the 2 min after completing administration of colloid. In patients receiving metaraminol, mean data were calculated over 3 min after the decrease in SAP had been corrected.

Statistical analysis was performed using the program Statview II (Abacus Concepts, Inc, USA). Patient data were compared using Student's *t*-test or analysis of variance (ANOVA) for continuous data and the chi-square test for categorical data. Haemodynamic data during subarachnoid block were compared within each group and between groups using ANOVA for repeated measures (ANOVAR). Changes in haemodynamic variables after subarachnoid block and after treatment for hypotension were compared using regression analysis. The level of sensory block was treated as non-parametric data and analysed using Kendall's rank correlation test. $P < 0.05$ was regarded as significant. Results are presented as mean (SD or SEM).

Results

Data from 48 patients are presented. Patient and pre-block haemodynamic data are presented in tables 1 and 2. Patients undergoing transurethral surgery were younger ($P = 0.01$), predominantly male ($P =$

Table 1 Patient characteristics (mean (range or SD)), incidence of hypertension (untreated or treated), dose of bupivacaine and sensory level (thoracic dermatome) to pinprick after 30 min of subarachnoid block (median (range)) in transurethral and orthopaedic patients. Significant differences between groups: $*P < 0.05$, $**P < 0.01$

	Transurethral ($n = 30$)	Orthopaedic ($n = 18$)
Age (yr)	71 (53–84)**	78 (61–96)**
Weight (kg)	55 (9)	51 (8)
Height (cm)	163 (7)	161 (9)
Sex (M/F)	27/3**	7/11**
ASA status (II/III)	22/8	11/7
Hypertension (untreated/treated)	6/2*	5/5*
Bupivacaine (ml)	2.9 (0.2)**	2.7 (0.3)**
Sensory level	6 (3–11)	5.5 (4–10)

Table 2 Patient haemodynamic variables (mean (SD)) in transurethral and orthopaedic patients. SAP = systolic arterial pressure, SVRI = systemic vascular resistance index, CVP = central venous pressure, CI = cardiac index, SI = stroke index and HR = heart rate. Significant differences between groups: * $P < 0.05$, ** $P < 0.01$

	Transurethral ($n = 30$)	Orthopaedic ($n = 18$)
SAP (mm Hg)	143 (21)*	157 (25)*
SVRI (dyn s cm^{-5} m^{-2})	3176 (859)	3145 (974)
CVP (cm H_2O)	6.4 (2.3)	6.3 (2.2)
CI (litre min^{-1} m^{-2})	2.57 (0.65)	2.90 (0.65)
SI (ml min^{-1} m^{-2})	37 (12)	34 (9)
HR (beat min^{-1})	72 (15)**	84 (11)**

Table 3 Patient characteristics (mean (range or SD)), dose of bupivacaine and sensory level (thoracic dermatome) to pinprick after 30 min of subarachnoid block (median (range)) in the three treatment groups. Significant difference between groups: ** $P < 0.01$

	No treatment ($n = 15$)	Colloid effective ($n = 19$)	Colloid ineffective ($n = 14$)
Age (yr)	71 (61–81)	73 (53–87)	77 (61–96)
Weight (kg)	55 (8)	55 (9)	49 (7)
Height (cm)	164 (6)	161 (8)	160 (7)
Sex (M/F)	12/3	15/4	7/7
ASA status (II/III)	11/4	12/7	10/4
Bupivacaine (ml)	2.9 (0.2)	2.8 (0.2)	2.9 (0.2)
Sensory level	8 (3–11)**	6 (4–9)**	4 (3–6)**

0.0006), received a larger dose of bupivacaine ($P = 0.01$) and had a lower baseline HR value ($P = 0.005$). There were more hypertensive patients, treated and untreated, in the orthopaedic group ($P = 0.04$). No patient with a baseline diastolic arterial pressure greater than 100 mm Hg presented for the study. There was no difference in the level of sensory block

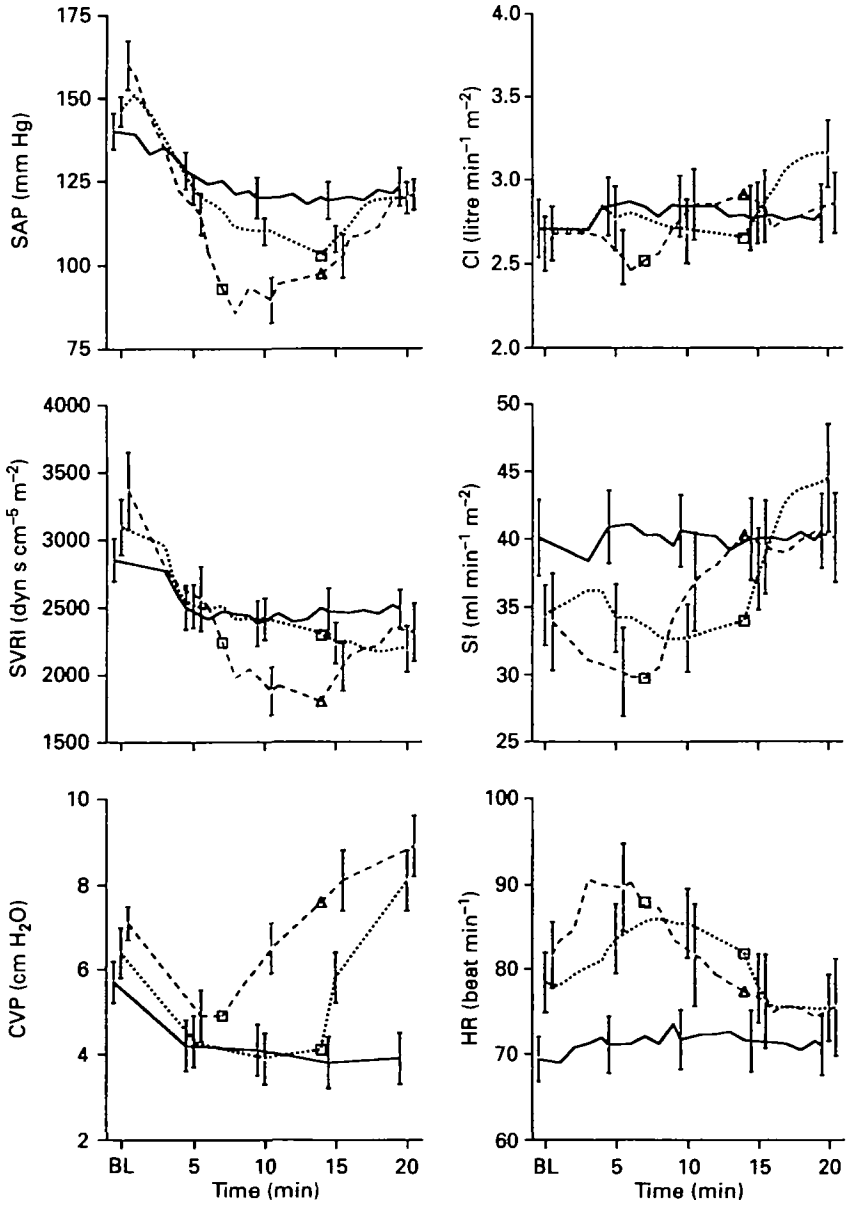

Figure 1 Mean (SEM) values for haemodynamic variables at baseline (BL) and during the first 20 min of subarachnoid block for the three groups of patients. —— = no treatment, ... = colloid effective and -- = colloid ineffective. □ = Administration of colloid; △ = administration of metaraminol.

Table 4 Percentage changes (Δ) and numerical difference (diff.) compared with baseline values for haemodynamic variables before treatment of hypotension or during established subarachnoid block when no treatment was required (mean (SD)). SAP = systolic arterial pressure, SVRI = systemic vascular resistance index, CVP = central venous pressure, CI = cardiac index, SI = stoke index and HR = heart rate. Significant changes compared with baseline: * $P < 0.05$; ** $P < 0.01$

	No treatment ($n = 15$)	Colloid effective ($n = 19$)	Colloid ineffective ($n = 14$)
ΔSAP (%)	−14 (6)**	−27 (6)**	−29 (11)**
ΔSVRI (%)	−14 (9)**	−24 (12)**	−23 (17)**
CVP diff. (cm H_2O)	−2.2 (1.5)**	−2.5 (1.3)**	−2.5 (1.5)**
ΔCI (%)	+3 (8)	0 (11)	−3 (29)
ΔSI (%)	0 (10)	−7 (13)*	−13 (19)**
ΔHR (%)	+3 (7)	+8 (8)**	+13 (13)**

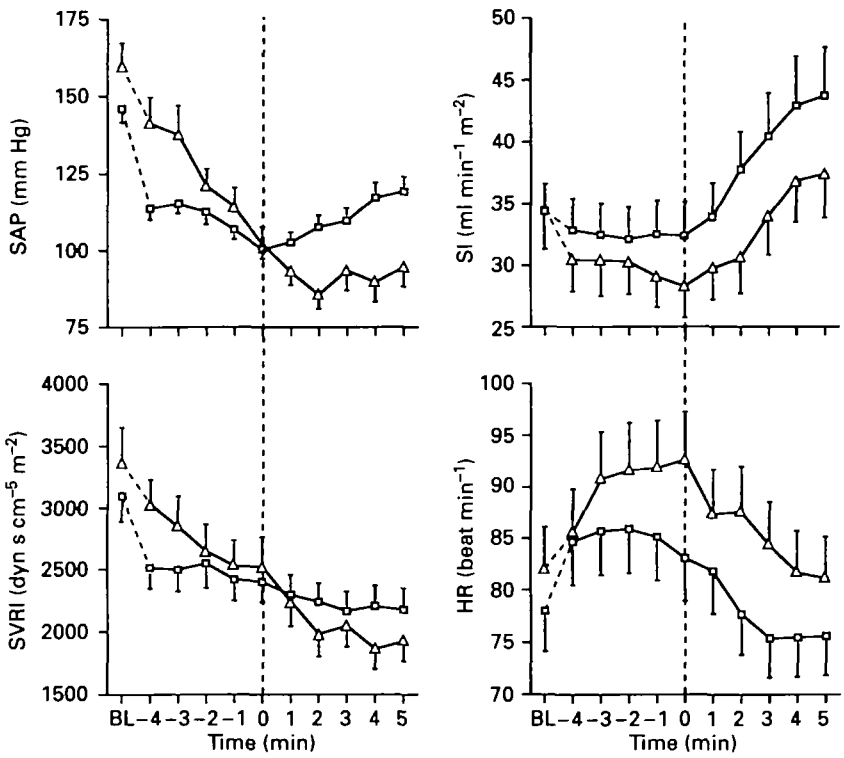

Figure 2 Mean (SEM) values for haemodynamic variables at baseline (BL) and before and after administration of colloid for the two groups of treated patients. □ = Colloid effective; △ = colloid ineffective.

to pinprick at 30 min between the transurethral and orthopaedic patients (table 1). Patients were grouped also according to management of hypotension (table 3 and fig. 1).

The haemodynamic changes after subarachnoid block and treatment of hypotension are presented in figure 1. The overall effect of subarachnoid block was a significant decrease in SAP, SVRI and CVP and a significant increase in HR (all ANOVAR; $P = 0.0001$). However, the magnitude and onset of these changes varied among patients (table 4).

Thirty-one percent of patients required no treatment for hypotension and in these patients CI, SI and HR did not change significantly (table 4, fig. 1). Sixty-nine percent of patients required treatment for hypotension. The less severe cases were managed with colloid alone and in this group the median time to treatment was 14 (range 8–17) min after the block. In more severe cases, where colloid and eventually metaraminol were required, the onset of hypotension was more rapid and initial treatment with colloid was started after only 7 (6–9) min.

There was no change in CI before treatment and SI decreased significantly only in those patients where colloid administration was found to be ineffective (ANOVAR; $P = 0.002$). HR increased significantly before treatment (ANOVAR; $P < 0.02$) (table 4).

The percentage decreases in SAP (table 4) were related closely to decreases in SVRI ($P = 0.0001$; $r = 0.6$) and to the height of sensory block after 30 min ($P = 0.001$). The percentage decreases in SAP were unrelated to the changes in CI and CVP. The percentage decreases in SI were related closely to the increases in HR ($P = 0.003$; $r = 0.5$).

The administration of colloid had a variable effect on SAP, caused a decrease in SVRI and HR (both $P < 0.05$) and increased CVP, CI and SI (all $P < 0.01$) (table 5). Figure 2 shows the changes in SAP, SVRI, SI and HR before and after administration of colloid.

Table 5 Percentage changes (Δ) and numerical difference (diff.) compared with values immediately before treatment, for haemodynamic variables immediately after treatment with colloid and during established treatment with metaraminol (mean (SD)) (scale: baseline = 100%). SAP = systolic arterial pressure, SVRI = systemic vascular resistance index, CVP = central venous pressure, CI = cardiac index, SI = stoke index and HR = heart rate. Significant changes compared with the value before treatment: $^{*}P < 0.05$; $^{**}P < 0.01$

	After colloid		After metaraminol ($n = 14$)
	Effective ($n = 19$)	Ineffective ($n = 14$)	
ΔSAP (%)	+7 (10)**	−12 (18)*	+33 (16)**
ΔSVRI (%)	−10 (13)**	−21 (20)**	+23 (14)**
CVP diff. (cm H_2O)	+4.5 (1.8)**	+3.4 (2.2)**	+2.6 (2.5)**
ΔCI (%)	+18 (15)**	+11 (19)*	+3 (8)
ΔSI (%)	+31 (20)**	+23 (21)**	+8 (16)
ΔHR (%)	−12 (10)**	−13 (11)**	−3 (11)

Table 6 Distribution of transurethral and orthopaedic patients and normotensive and hypertensive patients (untreated or treated) between the three treatment groups for hypotension

	No treatment	Colloid effective	Colloid ineffective	Total
Transurethral (elective)	12	12	6	30
Orthopaedic (non-elective)	3	7	8	18
Normotensive	13	12	5	30
Hypertensive (untreated:treated)	2 (2:0)	7 (3:4)	9 (6:3)	18

SVRI decreased, but this was significant only in patients in whom treatment was ineffective (ANOVAR; $P = 0.016$). In both groups of patients, SI increased and HR decreased (both ANOVAR; $P = 0.0001$). Administration of metaraminol caused an increase in SAP, SVRI and CVP (all ANOVAR; $P < 0.006$), without significant changes in CI, SI and HR (table 5).

A higher proportion of orthopaedic patients required treatment with colloid and metaraminol ($P = 0.04$) (table 6) and orthopaedic patients had a higher baseline SAP ($P = 0.04$) and HR ($P = 0.01$). Patients who were hypertensive had a greater requirement for treatment of hypotension after block ($P = 0.04$) (table 6). The height of sensory block to pinprick after 30 min was related to the extent of the patients' treatments ($P = 0.001$) (table 3).

Discussion

We found that during subarachnoid block, 69% of elderly patients required treatment for hypotension. The non-elective orthopaedic trauma patients were at more risk than those undergoing elective transurethal surgery. SAP decreased by 25% as early as 6–9 min after block in some patients. SVRI showed similar decreases as SAP. There was a modest reduction in CVP in all patients. Cardiac output was unaffected by subarachnoid block, because a decrease in SI in the more severe cases of hypotension was compensated by an increase in HR.

The BoMed provided a simple and non-invasive method of assessing changes in stroke volume and cardiac output. The BoMed follows pharmacologically induced changes in stroke volume with a coefficient of variation of 7.8% [11] and has been used successfully in several studies to measure changes in stroke volume and cardiac output during anaesthesia [12–16].

Non-invasive arterial pressure monitoring was used in preference to direct intra-arterial pressure monitoring. Data were recorded at 1-min intervals, which was sufficient to show the changes in SAP. Individual Dinamap readings have been shown to have a ±10% variability [17] and in order to minimize errors, treatment was started after only two consecutive low readings.

The choice of 8 ml kg^{-1} of the colloid Haemaccel to correct hypotension was based on the findings of previous studies [2, 3] and its relatively long half-life in the circulation [18]. Coe and Revanas found that severe hypotension requires treatment with a vasoconstrictor [2] and we chose to use metaraminol which was the only pure vasoconstrictor commercially available in our hospital. Delivery by infusion improved control over dose and prevented unnecessary episodes of hypertension.

Hemmingsen, Poulsen and Risbo [3] studied 48 patients receiving subarachnoid block with 0.5% bupivacaine 3 ml. They used a preload of isotonic sodium–glucose solution 7 ml kg^{-1} and ephedrine to prevent hypotension. Their conclusion was that ASA III patients required prophylaxis against hypotension. Unfortunately, neither the type of surgery nor the age of patients was stated. Taivainen [5] studied elderly patients with fractured hips receiving subarachnoid block and ephedrine or etilefrine to prevent hypotension. Only patients with decreases in mean arterial pressure of more than 25% were included and hence the incidence of hypotension was not available. However, Coe and Revanas [2] found that significant hypotension occurred in 60% of healthy elderly patients receiving subarachnoid block.

In the only comprehensive review of the haemodynamic effects of subarachnoid block, in mainly healthy adults, Green [7] reported a consistent decrease in CVP of 1.5–2.0 cm H_2O when sensory block was higher than the sixth thoracic dermatome, a slowing of HR (although this may vary by ±10 beat min^{-1}) and a reduction in cardiac output ranging from 0–40%. We found a decrease in CVP of similar magnitude. However, in contradiction to Green's [7] conclusions, we found that cardiac output did not

decrease and HR increased by 8–13% in those patients in whom SAP declined by 25%.

The observed decrease in CVP and SI, and increase in HR were reversed by administration of colloid, which suggests that after subarachnoid block, our patients became relatively volume depleted and required fluid administration. We found that 8 ml kg^{-1} of colloid was sufficient to correct the changes in CVP, SI and HR. Hemmingsen, Poulsen and Risbo [3] used a similar volume to prevent hypotension and Coe and Revanas [2] found no advantage in using a larger volume.

Some authors have stressed the importance of decreases in cardiac output and volume depletion in the genesis of hypotension [7, 19]. In our study, there was no large decrease in CVP or cardiac output. Only in those patients where SAP decreased by 25% was a significant decrease in stroke volume seen and in these patients cardiac output appeared to be maintained by a compensatory increase in HR. We did not study the effects of subarachnoid block when SAP decreased by more than 25% and cannot comment on what happens to CVP and cardiac output when hypotension remains untreated. However, we found that the decrease in SVRI, rather than a decrease in cardiac output, was the major factor causing hypotension. The changes in SVRI were related closely to SAP and fluid administration appeared to cause a further decrease in SVRI, particularly in those patients that failed to respond to colloid (fig. 2). There are several theoretical explanations for this decrease in SVRI. Volume loading before subarachnoid block has been shown to cause peripheral vasodilatation and a decrease in SVRI [13], which may have been the result of increased peripheral flow or the effect of volume expansion on baroreceptors and volume receptors in the atria and great vessels [20, 21]. Alternatively, SVRI may have decreased because sympathetic block from the subarachnoid block was still developing.

As was demonstrated by the patients' responses to metaraminol (fig. I, table 5), these findings strongly indicate that a peripheral vasoconstrictor is preferable to giving more fluids in severe or persistent hypotension. Coe and Revanas [2] studied the effects of preloading and found that fluids did not always prevent hypotension and also advocated using a vasopressor.

The decrease in SAP was related to the height of sensory block and this is a well known relationship [22]. The orthopaedic patients were more susceptible to the haemodynamic effects of subarachnoid block. They were older, more frequently hypertensive and their surgery was non-elective. The site of fracture would be expected to cause blood loss and a reduction in blood volume, but this was not supported by lower baseline CVP readings (table 2). Dagnino and Prys-Roberts showed that hypertensive patients were more susceptible to the effects of extradural anaesthesia and this may explain the increased susceptibility to subarachnoid block in the orthopaedic group [23].

The onset time of hypotension using 0.5% heavy bupivacaine for subarachnoid block has not been reported previously. The results of Hemmingsen, Poulsen and Risbo [3] indicate that the main haemodynamic changes occur within 10–15 min of block. We found that changes requiring treatment occurred rapidly within 6–9 min in many patients, indicating that patients should be monitored closely immediately after subarachnoid block.

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