Changes in haemodynamic variables during transurethral resection of the prostate: comparison of general and spinal anaesthesia

P. M. S. Dobson, L. D. Caldicott, S. P. Gerrish, J. R. Cole and K. S. Channer

SUMMARY

We have compared changes in haemodynamic variables before and during transurethral resection of the prostate in 22 patients under general or spinal anaesthesia. In the general anaesthetic group there was a significant decrease in cardiac output (mean 32% (SEM 5%)) and mean arterial pressure (14% (3%)) after induction of anaesthesia and a significant decrease (27% (3%)) in heart rate before the start of resection. Otherwise, variables remained close to baseline values. In the subarachnoid block group, mean arterial pressure decreased after induction (16% (2%)), but overall variables remained unchanged. We conclude that with both these anaesthetic techniques the greatest changes in haemodynamic variables occurred shortly after induction, and that these changes were greater during general than spinal anaesthesia. The resection period was not associated with significant haemodynamic changes. (Br. J. Anaesth. 1994; 72: 267-271)

KEY WORDS
Anaesthetic techniques • subarachnoid. Surgery: urological

Roos and colleagues [1] suggested that medium and long-term mortality increased after transurethral resection of the prostate (TURP) compared with open prostatectomy. This could not be explained by known risk factors. In particular, they highlighted an increased incidence of cardiac complications, including increased mortality from myocardial infarction, in the TURP group. Evans and colleagues [2, 3] have observed marked haemodynamic changes associated with the resection period of TURP during general anaesthesia and concluded that these changes may be detrimental to the myocardium, although the validity of their conclusions has been questioned [4].

In a preliminary report [5], Lawson and co-workers compared the haemodynamic response to TURP during general and subarachnoid anaesthesia. Their findings did not agree with those of Evans and colleagues, but they used a bioimpedance method to estimate cardiac output and this may partly explain the differences.

In this study we have used transcutaneous Doppler aortovelography (a similar method to that used by Evans and colleagues) as an index of cardiac output and have compared the changes in cardiovascular variables during TURP under spinal or general anaesthesia.

PATIENTS AND METHODS

After obtaining local hospital Ethics Committee approval and informed consent, we studied 22 patients undergoing elective TURP; they were allocated randomly to receive either subarachnoid or general anaesthesia. Patients whom the anaesthetist did not consider suitable for both types of anaesthesia were excluded. Patients with pre-existing cardiovascular, respiratory or endocrine diseases were not specifically excluded.

Patients were graded for ASA status and Goldman's cardiac risk index [6]. Standard anaesthetic techniques were used. Both groups received premedication with temazepam 20 mg. In the general anaesthetic group, anaesthesia was induced with a dose of etomidate sufficient to obtund the eyelash reflex, followed by vecuronium 0.1 mg kg⁻¹ and fentanyl 1.5 μg kg⁻¹. Manual IPPV was maintained until 3 min after induction when the trachea was intubated orally. The lungs were ventilated to an end-tidal carbon dioxide concentration of 4.5–5.0 %. Anaesthesia was maintained using 0.5–1.0 % enfurane and 65 % nitrous oxide in oxygen. Patients in the spinal group received, in the sitting position, 0.5 % hyperbaric bupivacaine 3.0 ml via a 25-gauge needle at the L2-3 or L3-4 interspace. Immediately after injection, the patient was laid supine until the block, as judged by loss of cold sensitivity, had reached a level of T10, when the patient was placed in the lithotomy position. I.v. sedation was not used in the spinal group.

Both groups received a loading dose of Hartmann's solution 500 ml administered over the first 10 min after induction of anaesthesia. Otherwise fluid input (Hartmann's solution) during the study period was determined by the anaesthetist based on clinical criteria (arterial pressure, heart rate and observation of patient).

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The operation was performed in the lithotomy position using continuous irrigation with a 1.5% glycine solution, warmed to body temperature. Arterial pressure (automated non-invasive), heart rate and peripheral oxygen saturation were measured (Kontron \textquoteleft Supermon\textquoteright). Cardiac output was estimated using suprasternal Doppler aorto velocography (Sci-Med PC Dop 842). This method of measurement of cardiac output has been shown to accurately reflect changes in cardiac output when compared with other standard techniques [7]. Mean velocity was obtained from the first moment of the Doppler power spectrum which was computed automatically. Cardiac output was derived by computer program from the product of the time-averaged mean velocity and cross-sectional area of the aorta, which was assumed to be 30 mm². The mean of at least three readings measured over 60 s was used and all measurements were performed by the same observer. The patient’s ECG was monitored from lead CM5. Systemic vascular resistance index (SVRI) was derived by dividing mean arterial pressure by cardiac output estimations. This calculation does not take into account central venous pressure (CVP), but other studies have found that CVP alters little during TURP [8]. Also, 100% increase in CVP (e.g. from 4 to 8 cm H₂O) with IPPV would have little effect on SVRI if mean arterial pressure is in the “normal” range.

Variables were recorded before induction, immediately before and after intubation in the general anaesthetic group, at 5 min after injection in the spinal anaesthesia group and immediately after the patients were placed in the lithotomy position in both groups. This latter time was taken as time zero and resection began within 5 min in all patients. Measurements were then recorded every 10 min from time zero throughout the resection period. Results of haemodynamic monitoring are presented from time zero throughout the resection period. Measurements were then recorded every 10 min from time zero throughout the resection period. Results of haemodynamic monitoring are presented from time zero throughout the resection period. Results of haemodynamic monitoring are presented from time zero throughout the resection period. Results of haemodynamic monitoring are presented from time zero throughout the resection period.

Blood loss was estimated at the end of the operation by photocolorimetry of the irrigation fluid using a Haemocue colorimeter. The weight of prostate resected, volume of i.v. (including the loading dose) and irrigating fluid administered up to the end of surgery, and grade of operator and duration of operation were recorded. Serum concentrations of sodium and osmolalities were measured before induction and immediately after operation.

Data were analysed by one-way analysis of variance, paired t tests and unpaired t tests, as appropriate. P < 0.05 was considered significant.

### RESULTS

There was no significant difference between the two groups in age, weight, ASA status, cardiac risk index or pre-existing disease (table I). One patient in each group had a history of hypertension (both treated with a diuretic), and one patient in the spinal group had a history of ischaemic heart disease (stable, treated with atenolol and nifedipine). Cardiovascular variables are presented as percentage change from baseline and as absolute values (figs 1–3). It should be noted that the resection period did not last 60 min for all patients; for the general anaesthetic group it lasted up to 30 min (n = 11), 40 min (n = 8), 50 min (n = 7) and 60 min (n = 4); for the spinal group it lasted up to 30 min (n = 11), 40 min (n = 6), 50 min (n = 5) and 60 min (n = 4).

In the general anaesthetic group there was a significant decrease in cardiac output (mean 4.6 (sd 0.7) litre min⁻¹ to 3.1 (0.82) litre min⁻¹) after induction of anaesthesia and the onset of manual IPPV (figs 1, 2). This was associated with a reduction in mean arterial pressure (104 (13) mm Hg to 88 (12) mm Hg) and heart rate (73 (11.2) beat min⁻¹ to 69 (11.1) beat min⁻¹) and paralleled a significant increase in systemic vascular resistance index (SVRI) of 41% (17%). Laryngoscopy and intubation were associated with a return in cardiac output and heart rate to baseline values, with mean arterial pressure increasing to above baseline. After placement of the patient in the lithotomy position, heart rate decreased significantly (73 (12.9) beat min⁻¹ to 54 (9.2) beat min⁻¹), mean arterial pressure and SVRI were within baseline limits and cardiac output was less than baseline.

In the spinal group, induction of anaesthesia was associated with a significant reduction in mean arterial pressure (116 (12) mm Hg to 98 (15) mm Hg) at 5 min, which then remained unchanged, and an initial reduction in cardiac output, but no change in heart rate or SVRI (figs 1, 3). No patient received a vasopressor agent at any time.

During resection, cardiac output in the general anaesthetic group remained unchanged for the first 30 min and returned to preinduction baseline values by 40 min into the resection period. Mean arterial pressure changed little during resection and SVRI decreased towards the end of the resection period. Heart rate increased gradually back to baseline values during resection (figs 1, 2).

In the spinal group, there was no significant change in any of the variables during the resection period, although the mean values for cardiac output had decreased at 50 and 60 min. This apparent reduction may be caused by the smaller number of patients whose operations lasted these durations (figs 1, 3).

There were no significant differences between the groups in prostatic weight resected, fluid balance or serum sodium concentrations and osmolality values before and after operation (table II). However, there was a trend towards decreased duration of surgery,

### Table I. Patient characteristics (mean (sd or range) or No.) and Goldman’s cardiac risk index (CRI) (mode (range)) in the general and spinal anaesthesia groups

<table>
<thead>
<tr>
<th></th>
<th>General</th>
<th>Spinal</th>
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<tbody>
<tr>
<td>Age (yr)</td>
<td>71.6 (63-85)</td>
<td>76.8 (64-84)</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>75.0 (11.8)</td>
<td>65.7 (8.2)</td>
</tr>
<tr>
<td>Goldman’s CRI</td>
<td>5 (0-5)</td>
<td>5 (0-5)</td>
</tr>
<tr>
<td>ASA I/II</td>
<td>10/1</td>
<td>9/2</td>
</tr>
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GENERAL VS SPINAL ANAESTHESIA IN TURP

**Fig. 1.** Mean (SEM) percentage changes in cardiac output (CO), mean arterial pressure (MAP), system vascular resistance index (SVRI) and heart rate (HR) from baseline (B), in patients in the spinal (---) and general anaesthesia (-----) groups after induction (I), after intubation (IT), at lithotomy (L) and during the 60-min resection period. *P < 0.05 compared with baseline values.

**Table II.** Operation details, and fluid and electrolyte balance for the two groups (mean (SEM))

<table>
<thead>
<tr>
<th></th>
<th>General</th>
<th>Spinal</th>
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<tbody>
<tr>
<td>Prostatic weight (g)</td>
<td>31.4 (18.0)</td>
<td>31.1 (26.5)</td>
</tr>
<tr>
<td>Blood loss (litre)</td>
<td>0.52 (0.39)</td>
<td>0.31 (0.4)</td>
</tr>
<tr>
<td>Irrigation fluid (litre)</td>
<td>17.8 (8.1)</td>
<td>13.3 (8.0)</td>
</tr>
<tr>
<td>I.v. fluid (litre)</td>
<td>0.90 (0.4)</td>
<td>1.11 (0.61)</td>
</tr>
<tr>
<td>Duration of surgery (min)</td>
<td>53 (19)</td>
<td>42 (19)</td>
</tr>
<tr>
<td>Serum sodium (mmol/l)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Preop.</td>
<td>137.4 (2.3)</td>
<td>136.8 (1.9)</td>
</tr>
<tr>
<td>Postop.</td>
<td>131.7 (5.5)</td>
<td>135.4 (2.7)</td>
</tr>
<tr>
<td>Serum osmolality (mmol/l)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Preop.</td>
<td>283.7 (4.8)</td>
<td>283.3 (5.5)</td>
</tr>
<tr>
<td>Postop.</td>
<td>283.0 (3.8)</td>
<td>279.5 (6.5)</td>
</tr>
</tbody>
</table>

decreased volume of irrigation fluid and smaller peroperative changes in serum concentration of sodium in the spinal group.

**DISCUSSION**

We have observed that significant haemodynamic changes occurred around induction of anaesthesia and were more pronounced with general than with spinal anaesthesia. Haemodynamic changes observed during the resection period were not significant clinically.

Several studies have demonstrated a good correlation between changes in cardiac output, as
assessed by Doppler aortoveloangiography (as used in our study), and those by other techniques, including thermodilution [9–12]. Correlation coefficients of between 0.87 and 0.96 have been calculated, with small coefficients of variability of between 5% and 15%. In our department the coefficient of variation of repeated measurements using this technique is <5%. [13].

The decrease in cardiac output at induction in the general anaesthetic group may reflect a reduction in venous return secondary to manual IPPV, combined with a reduction in heart rate. The large increase in SVRI would appear to be secondary to a reduction in systemic arterial pressure. Etomidate per se is not associated with a decrease in cardiac output [14], although when combined with fentanyl, as in our patients, a significant decrease may occur [15]. The subsequent changes in mean arterial pressure and heart rate after intubation were of a typical "pressor" response [16].

The significant decrease in heart rate which occurred in the general anaesthetic group between the values after intubation and "lithotomy" may be explained in part by loss of the positive chronotropic response to induction and intubation and also to a change in the depth of anaesthesia. The combination of fentanyl and vecuronium may also predispose patients to bradycardia.

In the spinal group, mean arterial pressure decreased by 15–20% when corresponding cardiac output values were 5–15% smaller than baseline values. This paralleled a decrease in SVRI of less than 10% at any time, suggesting that the overall change in SVRI produced by this level of spinal anaesthesia was relatively small. Although the highest level of block was not determined, the dose and technique used would be expected to produce a mid-thoracic block. Thus vasoconstriction of vessels supplied by unaffected sympathetic nerves would counteract vasodilatation of vessels supplied by blocked sympathetic nerves. Also, despite a mean decrease in mean arterial pressure of 15%, patients in the spinal anaesthesia group did not develop reflex tachycardia or increased cardiac output. Both findings may reflect decreased venous return as a result of venodilatation. Reduced venous return leading to a decrease in cardiac filling pressures would tend to decrease stroke volume via the Frank–Starling mechanism.

**Figure 2** Mean (SEM) cardiac output (CO) (■), mean arterial pressure (MAP) (▲) and heart rate (HR) (●) of patients in the general anaesthesia group at baseline (B), after induction (I), after intubation (IT) and during the resection period. *P < 0.05 compared with baseline values.

**Figure 3** Mean (SEM) cardiac output (CO) (■), mean arterial pressure (MAP) (▲) and heart rate (●) of patients in the spinal anaesthesia group at baseline (B), 5 min after injection (Inj.) and during the resection period. *P < 0.05 compared with baseline values.
relationship and low atrial pressures would tend to reduce heart rate via the Bainbridge reflex [17]. Also, sympathetic block may extend several segments higher than sensory block and a high sympathetic block (T1–T4) would reduce cardiac efferent sympathetic activity and thus may prevent an increase in heart rate and inotropy. The relevance of this in our patients is unknown as we did not estimate maximum height of the block.

In studies of patients undergoing TURP under general anaesthesia, Evans and colleagues [2, 3] observed large haemodynamic changes during the resection period. These changes (progressive increase in SVRI and reduction in cardiac output) were not observed in our patients. The difference between anaesthetic techniques (their patients received thiopentone, enflurane, morphine and vecuronium) is unlikely to account for this difference. Evans and colleagues estimated cardiac output using oesophageal Doppler echocardiography, a technique similar to ours, and again this is unlikely to account for the different findings. However, they studied patients in whom bladder irrigation during resection was produced with large volumes (> 11 litre per patient [2]) of fluid at room temperature (20 °C), not body temperature, as in our study. This resulted in a reduction in core temperature which may have significant haemodynamic effects, including an increase in SVRI and a reduction in both heart rate and cardiac output [18] (changes observed in their study). Lawson and colleagues [5], using a bioimpedance technique for estimating cardiac output, found similar haemodynamic changes as those in our study, although they did not state the temperature of their irrigation fluid.

If the haemodynamic changes that occur during anaesthesia and surgery are responsible for the apparent increase in cardiac morbidity and mortality [1], then changes which may tend to increase the ratio of myocardial oxygen demand to supply would be detrimental. In the general anaesthetic group, the only changes in the variables monitored which may promote myocardial ischaemia occurred after intubation, with a mean increase in mean arterial pressure of 15% (SEM 7%). The other haemodynamic changes observed (decrease in cardiac output, decrease in heart rate) would reduce myocardial work [19].

Similarly, in the spinal group the haemodynamic changes would tend to decrease cardiac work throughout the study (decreased cardiac output, heart rate and mean arterial pressure).

In conclusion, from this evidence it would appear that intrathecal anaesthesia is associated with more stable haemodynamic variables compared with this type of general anaesthesia.