Haemodynamic evidence for cardiac stress during transurethral prostatectomy

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Abstract

Objective—To compare haemodynamic performance during transurethral prostatectomy and non-endoscopic control procedures similar in duration and surgical trauma.

Design—Controlled comparative study.

Setting—London teaching hospital.

Patients—33 men aged 50-85 years in American Society of Anesthesiologists risk groups I and II undergoing transurethral prostatectomy (20), herniorrhaphy (eight), or testicular exploration (five).

Main outcome measures—Percentage change from baseline in mean arterial pressure, heart rate, Doppler indices of stroke volume and cardiac output, and index of systemic vascular resistance, and change from baseline in core temperature.

Results—In the control group mean arterial pressure fell to 11% (95% confidence interval -17% to -5%) below baseline at two minutes into surgery and remained below baseline; there were no other overall changes in haemodynamic variables and the core temperature was stable. During transurethral prostatectomy mean arterial pressure increased by 16% (5% to 27%) at the two minute recording and remained raised throughout. Bradycardia reached -7% (-14% to 1%) by the end of the procedure. Doppler indices of stroke volume fell progressively to 15% (-24% to -6%) below baseline at the end of the procedure, and the index of cardiac output fell to 21% (-32% to -10%) below baseline by the end of the procedure. The index of systemic vascular resistance was increased by 28% (17% to 38%) at two minutes, and by 46.8% (28% to 66%) at the end of the procedure. Core temperature fell by a mean of 0.8 (-1.0 to -0.6) °C. Significant differences existed between the two groups in summary measures of mean arterial pressure ($p<0.05$), Doppler indices of stroke volume ($p<0.005$) and cardiac output ($p<0.005$), index of systemic vascular resistance ($p<0.0005$), and core temperature ($p<0.0001$).

Conclusions—Important haemodynamic disturbances were identified during routine apparently uneventful transurethral prostatectomy but not during control procedures. These responses may be related to the rapid central cooling observed during transurethral prostatectomy and require further study.

Introduction

Transurethral prostatectomy is considered the best treatment for relieving obstruction of the bladder outflow.¹ The reported hospital mortality for transurethral prostatectomy ranges from 0.2% to 2.5%.² Most specialist centres expect a hospital mortality of between 0.5% and 1%. Hospital mortality and morbidity tend to be higher in small or non-specialised units and are related to medical state, presence of infection or uraemia, acute retention, age, and size of resection.³⁻⁷ Most of the morbidity and mortality is associated with the cardiovascular system, with myocardial infarction, cardiac arrest, heart failure,

or cardiac arrhythmia occurring in up to 2.5% of patients.^{4,5}

As transurethral prostatectomy is considered a low risk procedure, it should have a similar mortality and morbidity to other operations, of similar duration and surgical trauma, in men in their middle and late years. Recent studies of elective hernia repair in elderly patients report no deaths.^{8,9} Haemorrhoidectomy has a hospital mortality of about 0.001%, and there were no deaths in the 1242 patients in the Hospital In-Patient Enquiry sample in 1983.¹⁰ This disparity implies that important differences exist between endoscopic and non-endoscopic surgery.

In addition, recent epidemiological data have suggested an increased intermediate and long term risk of cardiovascular mortality after transurethral prostatectomy compared with open prostatectomy.^{3,11,12} In the largest such study the researchers were convinced that "the differences in mortality would be explained by confounding influences of differences in the preoperative severity of illness."¹² This preconception, based on the premise that open surgery is rarely offered to unfit patients, was not confirmed. Although patients who had transurethral prostatectomy tended to be less healthy, the increased risk associated with transurethral prostatectomy was present in all subgroups analysed, including the healthiest. This study raised serious questions regarding the safety and efficacy of transurethral prostatectomy, demanding reappraisal of every aspect of this technique.^{2,13}

The incidence of cardiovascular complications after transurethral prostatectomy is higher than expected for a non-invasive procedure. This could be due to the medical state of the patients or transurethral prostatectomy itself could be a cardiac risk factor. There are many factors associated with transurethral prostatectomy that could result in adverse cardiovascular effects including hypervolaemia, hypovolaemia, hypothermia, hypotension, glycine absorption, hyperammonaemia, hyponatraemia, and sepsis.² The increased risk of death from cardiovascular causes could be explained by perioperative cardiovascular disturbances resulting in myocardial damage. A study of cardiospecific isoenzymes indicated that subclinical myocardial damage can occur during routine transurethral prostatectomy.¹⁴

If transurethral prostatectomy is a cardiovascular insult this should be reflected by perioperative changes in haemodynamic performance. Little work has been done on this subject. Radioisotope dilution techniques performed before and after transurethral prostatectomy under spinal anaesthesia showed a postoperative fall in cardiac output of 17.5%.¹⁵ Raised pulmonary artery wedge pressure and changes in systemic vascular resistance have been identified in high risk patients undergoing transurethral prostatectomy.¹⁶ In our pilot study using oesophageal Doppler ultrasonography to measure aortic blood flow during routine transurethral prostatectomy we showed significant, early, and progressive haemodynamic responses.¹⁷ Bradycardia and a reduced Doppler index of stroke volume resulted in mean perioperative reductions in cardiac output of

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25%. As mean arterial pressure also rose, this indicated a significant rise in left ventricular afterload. Large falls in core temperature were noted and may have contributed to the adverse haemodynamic responses that we observed.

We report the findings of a larger study comparing haemodynamic responses during transurethral prostatectomy with those during non-endoscopic general surgical procedures in men of similar age. The objectives were to answer the following clinical questions. What haemodynamic responses occur during these procedures? Did the groups exhibit overall haemodynamic differences? What factors were present that may have been responsible for haemodynamic differences between the groups?

Subjects and methods

Patients aged between 50 and 85 undergoing routine transurethral prostatectomy, herniorrhaphy, or testicular exploration with physical status scores of I and II (American Society of Anesthesiologists) were entered into the study. Approval was granted by the Middlesex Hospital medical ethics committee and fully informed consent was obtained from each patient. A standardised anaesthetic technique and routine monitoring was used for all cases (box). The anaesthetist was not informed of the results of additional haemodynamic monitoring.

Transurethral prostatectomy was performed with the patient in the Lloyd-Davies position and used intermittent irrigation with 1.5% glycine solution delivered through a Uromatic fast flow set (Travenol, Thetford, Norfolk) from a reservoir set at 60 cm above the pubic symphysis. Surgeons performed their usual resection and the irrigating fluid was at ambient temperature (21°C). The length of the operation was the time from the start of resection until the urethral catheter was sited, to the nearest 10 minutes. The prostatic chippings were weighed on an electronic scale immediately postoperatively.

Oesophageal Doppler ultrasonography (Doptek, Chichester, Sussex) was used to record flow velocity waveforms from blood flow in the descending thoracic aorta. Oesophageal Doppler ultrasonography is an accurate method of following trends in cardiac output and has been validated against cardiac output measured by the thermodilution technique.^{18,19} A transducer is inserted orally after inducing anaesthesia and sited 35-40 cm from the incisors to detect aortic blood flow signals. Mean arterial pressure and Doppler waveform parameters were recorded at baseline with the patient in the Lloyd-Davies position and anaesthetically stable, before instrumentation in the transurethral prostatectomy group or with the patient prepared, draped, and anaesthetically stable immediately before incision in control patients. Further recordings were made at two minutes into the resection or after the incision and at 10 minute intervals until the end of the operation.

Stroke distance refers to the area contained within each flow velocity waveform and is a linear index of left ventricular stroke volume.^{18,19} The integral software incorporated in the Doppler system measures heart rate and stroke distance from the separation and area of five consecutive flow velocity waveforms, thus minimising measurement error. Minute distance is the product of stroke distance and heart rate and is a linear index of cardiac output. Given that the coefficient of variation for Doppler indices of cardiac output in a steady haemodynamic state was 3.8% whereas that for thermodilution was 6.2%¹⁸ and that a 13% change in cardiac output is required for clinical significance with thermodilution, a change in the Doppler index of cardiac output greater than 10% would be a reliable indication that a change in cardiac output has occurred.

Anaesthetic protocol and monitoring in patients undergoing transurethral prostatectomy, herniorrhaphy, or testicular exploration

Anaesthetic protocol

Premedication	Temazepam 10-20 mg
Induction	Thiopentone 4 mg/kg
Maintenance	Nitrous oxide 60% in oxygen, enflurane 0.5-1%, intermittent positive pressure ventilation. End tidal carbon dioxide 35 (SD 5) mm Hg
Muscle relaxant	Vecuronium 0.1 mg/kg
Analgesia	Papaveretum 0.2 mg/kg intravenously on induction

Anaesthetic monitoring

Blood pressure, electrocardiogram, and oxygen saturation	Patient monitor 78354A (Hewlett Packard Medical, Uxbridge, United Kingdom)
End tidal carbon dioxide	Datex Normocap (S W Vickers, Sidcup, Kent)

An index of systemic vascular resistance was calculated by dividing mean arterial pressure by minute distance and multiplying by 10⁴. In our unit central lines are not routinely placed in patients undergoing transurethral prostatectomy; thus we did not allow for the contribution of the central venous pressure to the pressure gradient over the vascular tree. Nevertheless, its contribution is likely to be small since a fall in central venous pressure of about 7 mm Hg would be required to cause a 10% rise in this non-invasive index of systemic vascular resistance, provided that the cardiac output remained stable. Studies that have monitored central venous pressure during transurethral prostatectomy generally showed small changes if any.^{15,20}

Changes in core temperature were measured in the lower oesophagus (High-Lo general purpose temperature probe, Mallinckrodt Critical Care, New York). This site gives the most satisfactory and repeatable measurements of core temperature.^{21,22}

Blood loss was estimated from the total haemoglobin loss, expressed as the product of the volume of irrigant collected, its haemoglobin concentration (g/l) (OSM2 Hemoximeter, Radiometer, Copenhagen, Denmark), and the preoperative haemoglobin concentration. The limit of accuracy was 70 ml, adequate for the purposes of this study as over 400 ml of blood needs to be lost before large compensatory cardiovascular responses are initiated.

Total fluid balance was determined by transducers (accurate to 50 g), placed under the operating table and connected to a microcomputer programmed to give a continuous readout of weight. The volume of irrigant transfused and extravasated was determined by first adding the estimated blood loss and the weight of resected prostate. The weight of the fluid given intravenously was then added to this negative figure to give the expected change in weight. The difference between the expected and actual change in weight was accounted for by irrigation fluid loading.¹⁷ The system gives an adequate degree of accuracy since an irrigation fluid load greater than 2 litres is required to produce symptoms of the transurethral resection syndrome.²³ The anaesthetist and surgeon were informed when weight gains indicating an irrigation fluid load over 1000 ml were observed.

STATISTICAL ANALYSIS

The group size required was estimated at 13 by

standard methods and data from our pilot study.^{17 24} Statistical analysis was performed after 13 control procedures and 20 transurethral prostatectomies had been performed. Significant differences between the groups in haemodynamic performance and core temperature were shown and the study was halted. Percentage change from the baseline recording had been selected as the feature of the haemodynamic data of most interest. This measure takes account of differences in the baseline reading between individuals, emphasises the relative importance of subsequent responses in individual patients, and is clinically meaningful, widely used, and easily interpreted. The mean percentage change from baseline at each recording was calculated for both groups and plotted against time to give a graphic indication of overall trends within each group.

Overall trends in haemodynamic performance were analysed by summary measures.²⁵ Plots of the variables versus time were constructed for each patient and the patients separated into groups depending on the size and direction of response. Trends within groups were identified by using a one tailed sign procedure. If preliminary analysis of data suggested reduction in a haemodynamic variable the null hypothesis that

summary measures of that variable were zero or positive was tested against the alternative hypothesis that the summary measures were negative. If the variable seemed to increase on preliminary analysis the hypothesis was adjusted appropriately.

The summary measures did not conform to a normal distribution so non-parametric testing was used to assess differences between the groups (Mann-Whitney U test). Reductions in core temperature were analysed by the same methods. Preoperative haemodynamic readings and core temperature data were compared non-parametrically to look for differences in baseline recordings between the groups (Mann-Whitney U test). The clinical operational research unit at University College London supervised the design and statistical analysis of the study.

Results

Twenty patients undergoing routine transurethral prostatectomy and 13 control patients undergoing either hernia repair (n=8) or testicular exploration (n=5) were entered into the study. Ten of the transurethral prostatectomies were performed by a senior registrar, nine by a registrar under supervision, and one by a consultant. Table I shows the details of the patient's age, duration of operation, core temperature readings, and, for the transurethral prostatectomy group, weight of resected tissue, blood loss, and irrigation fluid load. There were no significant differences in terms of age and duration of operation. Patients undergoing transurethral prostatectomy showed a wide range of blood loss and irrigation fluid loading and the distributions of these measures were positively skewed. One patient in the transurethral prostatectomy group developed atrial fibrillation on the first postoperative day. There was no excess morbidity in the control group.

Table II shows the results of preliminary interpretation of individual plots and one tailed sign tests and indicates the general trends within the groups in terms of direction and size. Table III shows the baseline

TABLE I—Details of men and operations

	Mean	Median	Standard deviation
Transurethral prostatectomy (n=20):			
Age (years)	71.05	74.00	8.98
Duration of operation (min)	38.25	40.00	8.63
Weight of resected tissue (g)	22.65	20.00	10.54
Irrigant used (l)	11.59	10.75	4.87
Intravenous fluids (ml)	140.0	50.0	237.1
Blood loss (ml)	226.6	145.8	243.8
Irrigation fluid load (ml)	727	340	1170
Baseline core temperature (°C)	36.0	35.9	0.665
End of operation	35.2	35.2	0.867
Mean change	-0.8	-0.8	0.410
Control procedures (n=13):			
Age (years)	71.00	69.00	11.28
Duration of operation (min)	42.31	40.00	11.66
Baseline core temperature (°C)	36.3	36.2	0.415
End of operation	36.2	36.2	0.451
Mean change	-0.1	-0.1	0.14

TABLE II—Results of preliminary assessment of haemodynamic parameters and one tailed sign test of summary measures in men undergoing transurethral prostatectomy and control procedures

	No of men showing				No with summary measure >0	No with summary measure <0	p Value	Median of summary measure
	Increase	Stable*	Decrease	Both				
Transurethral prostatectomy:								
Mean arterial pressure	11	4	3	2	11	9	0.41	189
Heart rate	4	8	8		5	15	<0.05	-178
Stroke distance	2	3	15		5	15	<0.05	-444
Minute distance	2	1	17		3	17	<0.005	-748
Index of systemic vascular resistance	18	2			19	1	<0.0001	1292
Control group:								
Mean arterial pressure	2	4	7		3	10	<0.05	-178
Heart rate	4	2	7		5	8	0.29	-138
Stroke distance	8	2	3		9	4	0.13	65
Minute distance	4	3	4	2	6	7	0.5	-4
Index of systemic vascular resistance	4	3	5	1	4	9	0.13	-142

*Changes in mean arterial pressure, heart rate, stroke distance, and minute distance within 10% of baseline and changes in index of systemic vascular resistance within 20% of baseline.

TABLE III—Mean (95% confidence interval) percentage change from baseline in haemodynamic parameters during transurethral prostatectomy and control procedures

	Baseline recording	% Change at							Mean of summary measures
		2 Minutes	10 Minutes	20 Minutes	30 Minutes	40 Minutes	50 Minutes	End of procedure	
Transurethral prostatectomy group:									
No of men	20	20	20	20	12	4		20	20
Mean arterial pressure	79 (73 to 85) mm Hg	16 (5 to 28)	9 (-2 to 20)	8 (-4 to 20)	8 (-2 to 18)	9 (-1 to 20)	15 (-20 to 51)	10 (0 to 20)	404 (34 to 773)
Heart rate	69 (63 to 75) beats/min	0 (-5 to 4)	-2 (-9 to 4)	-6 (-11 to 0)	-5 (-12 to 3)	-6 (-12 to 1)	-6 (-21 to 8)	-7 (-14 to 1)	-140 (-341 to 62)
Stroke distance	5.52 (4.78 to 6.26) cm	-7 (-14 to -1)	-10 (-16 to -3)	-10 (-17 to -2)	-14 (-21 to -8)	-21 (-34 to -9)	-16 (-34 to 3)	-15 (-24 to -6)	-468 (-738 to -198)
Minute distance	378 (322 to 433) cm/min	-8 (-15 to -1)	-13 (-20 to -5)	-15 (-23 to -7)	-18 (-28 to -8)	-27 (-37 to -16)	-21 (-42 to 0)	-21 (-32 to -10)	-606 (-898 to -314)
Index of systemic vascular resistance	2269 (1909 to 2630)	28 (17 to 38)	26 (14 to 39)	29 (17 to 41)	36 (23 to 50)	57 (30 to 84)	49 (-14 to 112)	47 (28 to 66)	1305 (822 to 1788)
Control group:									
No of men	13	13	13	13	8	6		13	13
Mean arterial pressure	87 (79 to 94) mm Hg	-11 (-17 to -5)	-5 (-12 to 2)	-4 (-12 to 4)	-7 (-15 to 0)	-2 (-14 to 10)	-7 (-25 to 11)	-9 (-17 to 0)	-210 (-464 to 43)
Heart rate	66 (57 to 74) beats/min	-1 (-7 to 4)	-4 (-12 to 3)	-3 (-12 to 6)	-5 (-14 to 3)	-5 (-9 to 1)	-10 (-16 to -5)	-5 (-11 to 0)	-179 (-436 to 78)
Stroke distance	5.26 (4.1 to 6.42) cm	0 (-9 to 9)	6 (-11 to 23)	7 (-11 to 26)	16 (-5 to 37)	21 (-8 to 49)	26 (-16 to 68)	14 (-7 to 35)	466 (-308 to 1240)
Minute distance	344 (260 to 428) cm/min	-2 (-11 to 7)	0 (-16 to 16)	2 (-13 to 18)	7 (-9 to 23)	15 (-12 to 41)	12 (-21 to 45)	7 (-12 to 26)	204 (-444 to 852)
Index of systemic vascular resistance	2937 (2156 to 3717)	-8 (-18 to 3)	-2 (-14 to 10)	-3 (16 to 10)	-9 (-22 to 4)	-10 (29 to 10)	13 (-38 to 12)	-10 (-25 to 6)	-243 (-701 to 216)

FIG 1—Haemodynamic responses during transurethral prostatectomy and control procedures

haemodynamic recordings, the subsequent mean percentage changes, and the mean summary measures. There were no significant differences in baseline recordings between the groups (Mann-Whitney U test). Figure 1 shows the haemodynamic trends.

Preliminary analysis of data suggested that in the control group mean arterial pressure and heart rate tended to fall, stroke distance was usually increased, and the minute distance and the index of systemic vascular resistance showed no overall changes (table II). Patients in the transurethral prostatectomy group showed increases in mean arterial pressure and the index of systemic vascular resistance with reductions in heart rate, stroke volume, and minute distance. Non-parametric analysis of summary measures within the control group showed that mean arterial pressure tended to fall; 10 patients had a negative summary measure and three had a positive value ($p < 0.05$, median -178.0). Analysis of the other haemodynamic variables showed no such trends (table II).

During transurethral prostatectomy the sign test showed equal numbers of patients with positive and negative summary measures of mean arterial pressure; the increases in mean arterial pressure were greater than the decreases, explaining the overall rising trend (table II). Heart rate tended to fall, 15 patients having a negative summary measure and five a positive measure ($p < 0.05$, median -178), and stroke volume was reduced, 15 patients having negative summary measures and five positive measures ($p < 0.05$, median -440). Minute distance was also reduced, with 17 patients having a negative summary measure and three a positive measure ($p < 0.005$, median -748). The index of systemic vascular resistance was decreased in one patient and increased in 19 ($p < 0.0001$, median 1292). Comparing non-endoscopic procedures with

transurethral prostatectomy there were significant differences between the groups in summary measures of mean arterial pressure ($p < 0.05$), stroke distance ($p < 0.005$), minute distance ($p < 0.005$), and the index of systemic vascular resistance ($p < 0.0005$).

The core temperature recordings at baseline were not significantly different in the two groups (table I). Although core temperature was stable in control patients, it fell rapidly in patients having transurethral prostatectomy (fig 2). At the end of the procedure there was a significant difference between the groups (Mann-Whitney U test, median temperature 36.2°C (control) v 35.2°C transurethral prostatectomy; $p < 0.005$). The mean reduction in core temperature was 0.8 (95% confidence interval -1 to -0.6) $^{\circ}\text{C}$. Summary measures analysis comparing changes in core temperature in the two groups (Mann-Whitney U test) confirmed a significant reduction in core temperature during transurethral prostatectomy ($p < 0.0001$).

Discussion

We have presented haemodynamic evidence of cardiovascular disturbances during routine, apparently uneventful transurethral prostatectomy. These responses were initiated early in the procedure and were independent of irrigation fluid loading or blood loss. The overall haemodynamic trends identified during transurethral prostatectomy were reductions in Doppler indices of stroke volume and cardiac output, together with bradycardia and increases in mean arterial pressure and index of systemic vascular resistance. These trends were not seen during non-endoscopic procedures of similar duration, surgical stimulus, and anaesthetic protocol. We believe that these results are caused by differences between the surgical procedures because an identical anaesthetic protocol was used. The groups were comparable in terms of age and had physical status scores of I and II (American Society of Anesthesiologists). Differences between the groups include blood loss, irrigation fluid loading, operating position, and the rapid central cooling during transurethral prostatectomy.

The mean duration of transurethral prostatectomy, mean weight of resected tissue and volume of irrigation fluid used, blood lost, and irrigation fluid load are comparable with those from other series, indicating that our data are representative of transurethral prostatectomy.^{4,7,26-29} All of these variables differ widely from one transurethral prostatectomy to another; the range of values obtained in our study indicates that a sample representative of transurethral prostatectomy as a whole was obtained. The unequal group size arose because access to patients having transurethral prostatectomy was easier and in the time it took to monitor 13 control patients, 20 transurethral prostatectomies had been monitored.

The haemodynamic findings were unlikely to have been caused by an error in measurement or to have occurred by chance. Not only do Doppler measurements of change in cardiac output compare well with thermodilution over wide changes in flow, blood pressure, and temperature but they also have a smaller coefficient of variation when repeated measurements are made on patients in a steady haemodynamic state.¹⁸ We therefore believe that a true representation of haemodynamic performance has been obtained. The consistent differences in Doppler indices between control and transurethral prostatectomy groups support our confidence in this method of haemodynamic monitoring.

There is no operation which represents a satisfactory control for transurethral prostatectomy. The control operations were selected with the intention of identifying the haemodynamic effects of general anaesthesia

FIG 2—Perioperative core temperature in men undergoing transurethral prostatectomy and control procedures. Bars represent standard error

and minor surgical stimulation during low risk procedures in men of similar age. Any additional cardiovascular disturbances caused by transurethral prostatectomy could contribute to the excess cardiovascular morbidity seen after this procedure.

General anaesthetic drugs have cardiovascular actions, including cardiodepression with bradycardia and reduced contractility, which are caused by central effects and direct actions on the myocardium. The main peripheral effects are vasodilatation and reduced left ventricular afterload. The cardiac output is maintained by more efficient left ventricular ejection, a potentially beneficial effect in patients with myocardial ischaemia provided that the effective coronary artery perfusion pressure is maintained.^{30, 31} These predictions assume that cardiovascular control mechanisms have not been modified by disease processes, age, or therapeutic interventions. At least one such factor was likely to have been present in most patients and might explain some of the differences in haemodynamic responses between patients having the same operation.

DIFFERENCES BETWEEN GROUPS

The major differences between transurethral prostatectomy and the control operations were in the operating position, blood loss, irrigation fluid loading, surgical site, and core temperature. All of these factors could affect the cardiovascular system and could cause predictable haemodynamic responses. The large increases in mean arterial pressure and index of systemic vascular resistance seen at two minutes into resection (table III) imply that a factor which has profound cardiovascular effects is present early during transurethral prostatectomy. Subsequent haemodynamic changes, caused by blood loss, irrigation fluid loading, or level of anaesthesia could be attenuated because of this previous response. Most patients in the transurethral prostatectomy group had an increased mean arterial pressure, a tendency to bradycardia, reduced stroke distance, reduced minute distance, and a raised index of systemic vascular resistance, all of which were present early in the procedure (table III).

Cardiac output will fall at any stage during transurethral prostatectomy if preload or contractility are reduced or if left ventricular afterload is increased. The early haemodynamic responses were unlikely to have been caused by reductions in preload because there was no apparent reason, such as haemorrhage, for large changes in intravascular volume to occur early in the procedures; irrigation fluid loading could not have been sufficient to be an important contributory factor early in the procedure. Cardiovascular effects of general anaesthetic drugs are unlikely to be responsible because similar responses would have been seen in the control group and the effects attributed to anaesthetic drugs are generally in the opposite direction to those seen in transurethral prostatectomy.

An alternative explanation is that there is an early vasoconstrictor response during transurethral prostatectomy which increases left ventricular afterload and depresses cardiac output. A pressor response to surgical stimulation in the form of pain, bladder distension, or the different surgical site is unlikely as no appropriate sympathetic responses such as tachycardia, sweating, and lacrimation were observed. Resection could release vasoactive substances into the circulation, which might cause myocardial depression or vasoconstriction. Significant haemodynamic effects have been noted when prostatic extracts were injected into dogs.³²

Three patients lost over 400 ml of blood, all of whom showed large responses at two minutes. Irrigation fluid loading of more than 300 ml occurred in 10 patients, one of whom was haemodynamically stable; of the others, all but one had large haemodynamic responses

at the two minute recording, before appreciable irrigation fluid loading could have occurred. In addition, the typical haemodynamic trends were seen by two minutes into resection in patients who had minimal blood loss or fluid loading. The haemodynamic differences between the groups must be the result of a factor peculiar to transurethral prostatectomy which was present early in the procedure.

The haemodynamic change in the transurethral prostatectomy group represents increased left ventricular afterload. Since left ventricular afterload rather than stroke work is the main determinant of cardiac work, transurethral prostatectomy could increase myocardial oxygen demand and possibly reduce the effective coronary perfusion pressure.^{33, 34} This has serious implications when operating on patients with coronary occlusive disease and could precipitate ischaemia, especially in the subendocardium, where tissue pressures are highest.³¹

The most remarkable difference between the two groups was the rapid central cooling during transurethral prostatectomy (fig 2). Previous studies have reported reductions in core temperature during transurethral prostatectomy, and our findings closely agree with them.³⁵⁻³⁹ The possibility that the cardiovascular system could be adversely affected by perioperative reductions in core temperature and the metabolic demands of rewarming has also been noted.^{35, 36, 39}

Hypothermia causes bradycardia, reduced cardiac output, and peripheral vasoconstriction, exactly the responses observed early during transurethral prostatectomy; there are additional adverse effects on every other body system.⁴⁰ The cardiovascular system is stressed as a result of increased afterload, effects on myocardial excitability and conduction, and the metabolic demands of rewarming.^{40, 41} Rapid reductions in core temperature start with bladder irrigation during transurethral prostatectomy as unheated irrigant is used and could be a significant aetiological factor in the production of the haemodynamic responses observed. The mechanisms which could result in increased afterload include neurovascular reflexes and increases in whole blood viscosity.⁴²

CONCLUSIONS

Our results support the hypothesis that routine transurethral prostatectomy is a cardiac stress. Perioperative factors associated with this procedure that could cause these haemodynamic responses include haemorrhage, irrigation fluid loading, and reductions in core temperature. Finding major cardiovascular disturbances during apparently uneventful routine endoscopic procedures but not in non-endoscopic surgery has serious implications in terms of patient selection and which procedure to perform. Patients with severe intercurrent cardiorespiratory disease may be more appropriately managed by a minimally invasive approach such as prostatic stenting.^{43, 44} Measures to prevent reductions in core temperature and other physiological insults during transurethral resection, especially of larger glands or in high risk patients, are a sensible precaution. The role of haemorrhage, irrigation fluid loading, and body temperature changes in the aetiology of adverse perioperative cardiovascular responses is being investigated in an ongoing study of transurethral prostatectomy.

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- 1 Chisholm GD. Benign prostatic hyperplasia: the best treatment. *BMJ* 1989;299:215-6.
- 2 Neal DE. Prostatectomy—an open or shut case. *Br J Urol* 1990;66:449-54.
- 3 Wennburg JE, Roos N, Sola L, Schori A, Jaffe R. Use of claims data systems to evaluate health care outcomes. Mortality and re-operation after prostatectomy. *JAMA* 1987;257:933-6.
- 4 Holtgrewe HL, Valk WL. Factors influencing the mortality and morbidity of transurethral prostatectomy: a study of 2015 cases. *J Urol* 1962;87:450-9.
- 5 Melchior J, Valk WL, Foret JD, Mebust WK. Transurethral prostatectomy: computerised analysis of 2223 consecutive cases. *J Urol* 1974;112:634-42.
- 6 Blandy JP. Benign prostatic enlargement. *BMJ* 1971;i:31-5.
- 7 Sach R, Marshall VR. Prostatectomy: its safety in an Australian teaching hospital. *BMJ* 1977;64:210-4.
- 8 Allen PIM, Zager M, Goldman M. Elective repair of groin hernias in the elderly. *Br J Surg* 1987;74:987.
- 9 Tingwald GR, Cooperman M. Inguinal and femoral hernia repair in geriatric patients. *Surg Gynecol Obstet* 1982;154:704-6.
- 10 Office of Population Censuses and Surveys. *Hospital inpatient enquiries* 1983. London: HMSO, 1985.
- 11 Roos N, Ramsay EW. A population based study of prostatectomy: outcomes associated with differing surgical approaches. *J Urol* 1987;137:1184-8.
- 12 Roos NP, Wennburg JE, Malenka DJ, Fisher ES, McPherson K, Andersen TF, et al. Mortality and re-operation after open and transurethral resection of the prostate for benign prostatic hyperplasia. *N Engl J Med* 1989;320:1120-4.
- 13 TU or not TU [Editorial]. *Lancet* 1989;i:1361-2.
- 14 Coppinger SWV, Hudd C. Risk factor for myocardial infarction in transurethral resection of the prostate? *Lancet* 1989;ii:859.
- 15 Mebust WK, Brady TW, Valk WL. Observations on cardiac output, blood volume, central venous pressure and electrolyte changes in patients undergoing transurethral prostatectomy. *J Urol* 1970;103:632-6.
- 16 De Angelis L, Chang P, Kaplan JH, Kudish H, Sacks S, Wender R, et al. Hemodynamic changes during prostatectomy in cardiac patients. *Crit Care Med* 1982;10:38-40.
- 17 Evans JWH, Singer M, Chapple CR, Macartney N, Coppinger SWV, Milroy EJG. Haemodynamic evidence for per-operative cardiac stress during transurethral prostatectomy: a preliminary communication. *Br J Urol* 1991;67:376-80.
- 18 Singer M, Clarke J, Bennett ED. Continuous hemodynamic monitoring by esophageal Doppler. *Crit Care Med* 1989;17:447-52.
- 19 Singer M. Continuous haemodynamic monitoring by oesophageal Doppler (MD thesis). London: University of London, 1990.
- 20 Kunin SA, Limbert DJ. Central venous pressure monitoring during transurethral prostatectomy. *J Urol* 1969;102:469-72.
- 21 Cork RC, Vaughan RW, Humphrey LS. Precision and accuracy of intra-operative temperature monitoring. *Anesth Analg* 1983;62:211-4.
- 22 Imrie MM, Hall GM. Body temperature and anaesthesia. *Br J Anaesth* 1990;64:346-54.
- 23 Ghanem AN, Ward JP. Osmotic and metabolic sequelae of volumetric overload in relation to the TUR syndrome. *Br J Urol* 1990;66:71-8.
- 24 Armitage P. *Statistical methods in medical research*. Oxford: Blackwell Scientific 1971:185.
- 25 Matthews JNS, Altman DG, Campbell MJ, Royston P. Analysis of serial measurement in medical research. *BMJ* 1990;300:230-5.
- 26 Perkins JB, Miller HC. Blood loss during transurethral prostatectomy. *J Urol* 1969;101:93-7.
- 27 Flechner SM, Williams RD. Continuous flow and conventional resectoscope methods in transurethral prostatectomy: comparative study. *J Urol* 1982;127:257-9.
- 28 Heathcote PS, Dyer PM. The effect of warm irrigation on blood loss during transurethral prostatectomy under spinal anaesthesia. *Br J Urol* 1986;58:669-71.
- 29 Mebust WK, Holtgrewe HL, Cockett ATK, Peters PC. Transurethral prostatectomy: immediate and postoperative complications. A cooperative study of 13 participating institutions evaluating 3,855 patients. *J Urol* 1989;141:243-7.
- 30 Schulte-Schasse U, Hess W, Tarnow J. Haemodynamic response to induction of anaesthesia using midazolam in cardiac surgical patients. *Br J Anaesth* 1982;54:1053-7.
- 31 Merin RG. Is anesthesia beneficial for the ischemic heart? *Anesthesiology* 1980;53:439-40.
- 32 Verrilli RA, Uhlman RC, Vick NF, Hunsicker WC Jr. The hypotensive effect of a prostatic extract. *J Urol* 1962;87:184-6.
- 33 Braunwald E. Control of myocardial oxygen consumption. Physiologic and clinical considerations. *Am J Cardiol* 1971;27:416-32.
- 34 Berne RM, Rubio R. Coronary circulation. In: Berne RM, Sperekalis N, Geiger SR, eds. *Handbook of physiology. Section 2: the cardiovascular system*. Baltimore: Williams and Wilkins, 1979:873-952.
- 35 Rabke HB, Jenicek JA, Khouri E Jr. Hypothermia associated with transurethral resection of the prostate. *J Urol* 1962;87:447-9.
- 36 Allen TD. Body temperature changes during prostatic resection as related to the temperature of the irrigation solution. *J Urol* 1973;110:433-5.
- 37 Kulatilake AE, Roberts PN, Evans DF, Wright J. The use of cooled irrigating solution during transurethral resection of the prostate. *Br J Urol* 1981;53:261-2.
- 38 Rawstron RE, Walton JK. Body temperature changes during transurethral prostatectomy. *Anaesth Intens Care* 1981;9:43-6.
- 39 Carpenter AA. Hypothermia during transurethral resection of prostate. *Urology* 1984;23:122-4.
- 40 Carli F. Metabolic disturbances of hypothermia. In: *Ballière's clinical anaesthesiology* 1989;3:405-21.
- 41 Reuler JB. Hypothermia: pathophysiology, clinical settings and management. *Ann Intern Med* 1978;89:519-27.
- 42 Keatinge WR, Coleshaw SRK, Cotter F, Mattock M, Murphy M, Chelliah R. Increase in platelet and red cell counts, blood viscosity, and arterial pressure during mild surface cooling: factors in mortality from coronary and cerebral thrombosis in winter. *BMJ* 1984;289:1405-8.
- 43 Williams G, Jager R, McLoughlin J, El Din A, Machan L, Gill K, et al. Use of stents for treating obstruction of urinary outflow in patients unfit for surgery. *BMJ* 1989;298:1429.
- 44 Chapple CR, Milroy EJG, Rickards D. A permanently implanted urethral stent for prostate obstruction in the unfit patient: preliminary report. *Br J Urol* 1990;66:58-65.

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Extended fetal echocardiographic examination for detecting cardiac malformations in low risk pregnancies

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Abstract

Objective—To improve the rate of prenatal detection of cardiac malformations in a low risk population.

Design—Comparison of extended fetal echocardiography with the standard four chamber view in detecting abnormalities. Extended echocardiography comprised the four chamber view and visualisation of the left ventricular outflow tract, the right ventricular outflow tract, and the main pulmonary artery and its branches. In cases with abnormal results complete echocardiographic studies were performed by a paediatric cardiologist using M mode, Doppler, and colour flow mapping techniques.

Setting—Obstetric ultrasonographic unit at Shaare-Zedek Medical Centre, Jerusalem.

Subjects—5400 fetuses in low risk pregnancies between 18 and 24 weeks' gestation (mean 21 weeks); 53 were lost to follow up.

Main outcome measures—Detection of abnormality before and after birth.

Results—During the study 23 infants (0.4%) were born with cardiac abnormalities, 21 of whom had major structural and functional heart disease. 18 fetuses had heart disease diagnosed prenatally, 11 by the four chamber view alone (sensitivity 48%) and a further seven by extended echocardiography (sensitivity 78%). Five fetal cardiac defects were

missed prenatally (false negative rate 22%). These included coarctation of aorta, persistent truncus arteriosus, tetralogy of Fallot, ventricular septal defect, and pulmonic stenosis. Only one false positive diagnosis (coarctation of aorta) was made (specificity 99.9%, false positive rate 0.1%). The abnormality was correctly identified in 17 out of 18 cases.

Conclusions—The extended fetal heart examination detected 86% (18/21) of major abnormalities in a low risk population. The examination should be incorporated into routine prenatal ultrasonographic investigations.

Introduction

The incidence of congenital heart disease is 8/1000 live births. Half of the cases are considered minor and are easily corrected by surgery. The remainder are more serious and account for about 50% of deaths from lethal malformations in childhood.^{1,2} Congenital heart diseases are 6.5 times more common than chromosomal abnormalities and four times more common than neural tube defects.¹ Unlike chromosomal and neural tube defects, for which there has been extensive prenatal screening, congenital heart diseases are often not identified until the infant is born. The importance of early diagnosis of congenital heart disease is further