Methoxamine and blood loss in prostatic surgery

Sir,—I wish to comment on the significance of the findings of Dr Chambers and colleagues [1] in investigating the effects of methoxamine on blood loss during transurethral prostatic resection. A brief review of their references and a further literature search succeeded in finding only one paper where postoperative blood loss was considered. Clarke and colleagues, in studying patients receiving total hip arthroplasty, found that only 55% of measured blood loss occurred in theatre; the rest of the blood loss occurred in the postoperative period [2].

As all patients who undergo transurethral prostatic resection in this district undergo bladder irrigation overnight, one can only assume a similar picture in this group of patients, and therefore perhaps continuing the study into the postoperative period may have added to the relevance of the findings. Also, if indeed glycerine was maintained at a height of 500 cm H₂O above the patient, then the operating theatres must have impressively high ceilings! M. ALEXANDER-WILLIAMS
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Sir,—I agree that postoperative blood loss could be calculated in a similar fashion and add to the relevance of our findings. However, I feel that it would not be accurate to assume comparisons between patients undergoing total hip arthroplasty and transurethral prostatic resections. The height of the glycerine should have been 200 cm H₂O instead of 500 cm H₂O, as you correctly observed.

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SIR,—I was interested to read of the use of methoxamine to reduce prostatic volume and hence vascular capacitance in patients undergoing transurethral resection of the prostate under spinal anaesthesia in an effort to reduce blood loss [1]. The effect of methoxamine given as an α agonist to reverse hypotension, lasts 60-90 min [2] and the effect on the prostate could be expected to be the same. As the effects of methoxamine decline, then previously uncoagulated vessels would dilate, exacerbating blood loss at a time when the patient is either in the recovery room or back on the surgical ward with less haemodynamic monitoring in progress.

It would have been interesting to compare the admission and discharge haemoglobin concentrations between the groups to see if an overall difference existed. Alternatively, if bladder irrigation was continued after operation, the same photometric technique [3] and method of calculating blood loss could have been used to compare perioperative blood loss rather than the pure intraoperative losses as measured in the study.

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Sir,—I agree with Dr Howells that the effect of methoxamine could be expected to last approximately 90 min, but its effect is not directly on the prostatic blood vessels, as he suggests, but on α receptors in the prostatic adenoma and capsule causing an in vivo contractile response [1]. As most of the prostatic adenoma was resected during surgery, it is unlikely that the remaining blood vessels would dilate after the effects of methoxamine had declined. A comparison of admission and discharge haemoglobin concentrations would be difficult to interpret as changes in haemoglobin concentration could occur because of haemodilution and could not be attributed to blood loss alone. I agree that total perioperative blood loss could be calculated in a similar fashion, and would have provided us with additional information.

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Posture and autonomic cardiac control

Sir,—In a recent issue of the journal [1], McHugh, Robinson and Galletly reported on the effect of the Trendelenburg position and leg elevation on autonomic control of sinus node function. They used three different body postures in healthy volunteers: the supine position, head-down tilt position (−10°) and supine position with 50-cm leg elevation from the hip. In these settings they failed to find any significant alterations in the time and frequency domain indices of heart rate variability.

We studied the effect of postural changes on cardiac vagal control in healthy subjects in the supine position with head-up (70°) and head-down (−30°) tilts. We found that postural changes from supine to head-up tilt and from head-up tilt to head-down tilt positions resulted in changes in cardiac autonomic tone. The postural changes from the supine to the head-up tilt position caused a significant reduction in parasympathetic indices of the time and frequency domain measures of short-term heart rate variability (i.e. mean normal RR interval, percent of consecutive normal RR interval differences > 50 ms, root mean square successive difference, high frequency power), and an increase in the low frequency/high frequency ratio, indicating a shift towards sympathetic tone, was also seen. Similar results were reported elsewhere [2, 3]. These observations could be explained by the decreasing load on the arterial baroreceptors. This hypothesis was substantiated further by our findings that in the head-down tilt position, parasympathetic tone increased and sympathetic tone decreased, as reflected in the variables mentioned above. The differences between our findings and those of McHugh, Robinson and Galletly are probably related to different designs. In the −10° head-down tilt position there is presumably very little change in the loading conditions of the arterial baroreceptors. The low pressure receptors might have been triggered; however, in the absence of arterial and/or intracardial pressure recordings, no data were available on the real extent of the baroreceptor challenge. The same holds for the leg elevation manoeuvre.

We feel that further studies with continuous pressure recordings are warranted to clarify the issue.

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Sir,—In commenting on our paper [1], Kardos, Rudas and Simon described their own use of the analysis of short-term heart rate variability to assess the effect of varying posture on cardiac autonomic control. Essentially they have confirmed that head-up tilt causes sympathetic activation (as evidenced by an increase in the low frequency variability) in comparison with supine and head-down positions. This phenomenon is well described [2, 3]. Their letter also indicates that increased parasympathetic tone was found when their subjects went from head-up tilt (+70°) to head-down tilt (−30°). However, they give no indication of any comparison between the supine and head-down positions, which is the finding in which we were interested. Without this comparison we cannot comment further on their letter as their results seem consistent with our own.

We would be very happy to reply to the letter of Kardos, Rudas and Simon if a statistical comparison can be given between the flat supine position and head-down tilt (with exclusion of order bias).

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Post-reperfusion coagulopathy

Sir,—The case reports of Bayly and Thick [1] on reversal of post-reperfusion coagulopathy during orthotopic liver transplantation are verified by our experience in Birmingham. Three patients have each received protamine sulphate 0.75 mg kg⁻¹ in the presence of a “straight line” thrombelastogram, 5 min after reperfusion of the new liver; an empirical dose was used because of the fact that the patients were still receiving veno-venous bypass without the use of therapeutic anticoagulation or heparin bonded tubing.

In each case, coagulation was re-established with a minimally prolonged reaction time. However, in each case, fibrinolysis was apparent and the resultant thrombelastogram produced the “Taj Mahal” pattern. The combination of reformation of coagulation following administration of protamine and the more usual presence of a prolonged reaction time after reperfusion led us to believe that either heparin itself or a heparin-like substance released from the newlyperfused liver was responsible for the coagulopathy consistently seen after reperfusion. In support of this, the partial thromboplastin time (PTT) was also specifically prolonged at this stage of the procedure.

Since our more standardized use of aprotinin (a bolus of 2 Mu. followed by 50 ml per hour until after reperfusion) severe coagulopathy at this stage is now unusual, even in the “at risk” groups, namely alcoholic liver disease, long standing autoimmune disease and regrafts.

While protamine may have a place in the diagnosis of reperfusion coagulopathy, I would advise caution in its use for complete correction of a heparin-like effect. Not only is the bypass tubing at risk of clot formation, but the hepatic artery and portal vein may also be at risk of occlusion.

We accept an International Normalized Ratio (INR) of < 2 and a PTT of < 90 s at the end of the procedure in order to protect the vascular anastomoses.

J. Huggins
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Sir,—It is gratifying that our observations have been confirmed by others, who also appear to have reached the same conclusions on the cause of the phenomenon.

In contrast with Dr Huggins’ experience, the appearance of fibrinolysis, as detected by thrombelastography, is rare in our unit. In fact, I have seen evidence of fibrinolysis only twice in the last 35 liver transplants. In neither of the reported cases was fibrinolysis apparent, as evidenced by the thrombelastogram in figure 2. On the other hand, experience over the past year has shown that a heparin-like effect is demonstrable in up to 50% of all liver transplants, although not all of these required treatment with protamine.

I agree with Dr Huggins that severe coagulopathy after reperfusion is becoming less common, although we do not routinely use aprotinin as prophylaxis. When treating a coagulopathy, it remains important to aim to stop unacceptable bleeding, rather than to normalize numbers. Therapy should be directed at the cause of the problem: thus if a heparin-like effect is suspected, then protamine would seem to be the logical treatment. If this is used injudiciously, then there are major risks of vascular thromboses, just as if platelets or other procoagulants are used inappropriately. We have had experience while still running veno-venous bypass, as we do not wish to risk clot formation in the bypass tubing, and resultant emboli.

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Hyperventilation-induced unconsciousness during labour

Sir,—We read with interest the case report by Burden, Janke and Brighouse [1], describing the management of hyperventilation-induced unconsciousness during labour with extradural block. Although we applaud the fact that the outcome of this case was positive, we question the wisdom of initiating a major conducting block in an unconscious parturient who presumably had an unprotected airway. The authors reported that at the time of arrival in the hospital, this patient was “unconscious” and scored 7 on the Glasgow coma scale. They were so sure that the patient’s problems were secondary to pain-related hyperventilation that they opted to treat her unconsciousness with an extradural anaesthetic. This patient’s principal problem was not hyperventilation, but rather the potential for aspiration pneumonitis. If she was indeed unconscious, her airway should have been the paramount consideration. Additionally, we question the advisability of initiating an extradural anaesthetic in a patient who has just lost consciousness. If her altered mental state was caused by increased intracranial pressure, a “wet-tap” could have had devastating consequences; additionally, in her comatose state, the patient could not have identified paresthesiae, and a total spinal or caudal would presumably have been misdiagnosed.

We suggest that this case report sends the wrong message to your readers. Parturients at term are known to have “full stomachs” and are at risk of Mendelson’s syndrome. This is evidenced by the fact that aspiration and failed intubation remain major causes of anaesthesia-related maternal death in England and Wales [2]. It is our opinion that in unconscious parturients, airway management should precede other diagnostic and therapeutic measures.

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