Waldeyer[1]. According to Waldeyer, the pelvic sympathetic innervation consists of a paired truncus sympathicus pelvinus, a primary sympathetic pelvic plexus and a secondary sympathetic plexus. The paired truncus sympathicus pelvinus is identical to what we call the hypogastric nerves. The primary sympathetic pelvic plexus corresponds to sacral sympathetic trunk described by Dr Liang and colleagues. The primary sympathetic plexus is closely related to the hypogastric vessels and is located posterior to the parietal pelvic fascia. As the function of this sympathetic plexus is not clear, it is not known what symptoms are associated with intraoperative damage to these nerves. Damage to these nerves is unlikely during total mesorectal excision as the area posterior to the parietal fascia close to the iliac artery and vein is not entered. The superior rectal plexus as described by Dr Liang *et al.* corresponds to the superior rectal plexus as part of the secondary sympathetic plexus as described by Waldeyer. As these nerves only innervate the rectum we agree they are not relevant in rectal cancer surgery.

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Randomized controlled trial of the effect of mannitol on renal reperfusion injury during aortic aneurysm surgery

Sir

We were interested in the paper by Mr Nicholson and colleagues (*Br J Surg* 1996; **83**: 1230–3) reporting the protective effect of mannitol on renal reperfusion injury following infrarenal aortic surgery. We would like to comment on several points raised in this study.

The mechanism of renal injury in this situation is undoubtedly complex. Cross-clamping of the aorta has significant haemodynamic effects resulting in relative ischaemia of the kidney. A recent review in this journal[1] suggested that renal ischaemia results in damage to the proximal convoluted tubule. This is in contrast to other vascular beds, in which reperfusion with oxygenated blood is responsible for most of the tissue injury. The results of this study show that mannitol reduces urinary *N*-glucosaminidase excretion, suggesting that mannitol prevents the initial renal ischaemic injury, rather than by acting as a free radical scavenger.

The authors also suggested that the urinary albumin:creatinine ratio (ACR) was a useful marker of glomerular damage. In our experience these data are significantly skewed to the right and, therefore, should not be presented as mean(standard errors) which may give misleading results. However, non-parametric statistics were correctly used to analyse the data. Microalbuminuria is useful as a marker of renovascular permeability, which has been shown to accurately reflect the short-lived reversible change in systemic vascular permeability resulting from ischaemia–reperfusion injury. It is therefore incorrect to use the ACR as a measure of structural glomerular damage. The earliest clinical manifestation of ischaemia–reperfusion injury following aortic surgery is usually colonic ischaemia[2], which in severe cases may progress to adult respiratory distress syndrome (ARDS) and multisystem organ failure (MSOF). Previous work has shown an increase in ACR in all patients within 3 h of aortic surgery, returning to normal within 24–48 h. Those patients who later developed respiratory dysfunction had persistently higher levels of ACR, suggesting that this may be a strong predictor for the development of MSOF[3]. Mannitol acts by scavenging hydroxyl radicals produced on reperfusion and may therefore protect against the development of splanchnic ischaemia and ARDS[4].

The results of this paper show that in patients given mannitol there were no deaths from MSOF, whereas there was a single death from MSOF in the control group. With such small numbers, it is impossible to conclude that mannitol protects against renal reperfusion injury. One could, in fact, argue that in the mannitol group the two deaths from perioperative myocardial infarction may be a result of thromboxane A_2 release on reperfusion, with a reduction in cardiac index and hence an increase in myocardial ischaemia[5]. We agree with the authors that further work is needed to clarify both the aetiology of this renal injury and the mechanism of action of mannitol.

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Author's reply

Sir

I thank Messrs Tisi and Shearman for their interest and comments. The most important point is that the published work is a preliminary study which suggests that mannitol is protective against subclinical renal injury during infrarenal abdominal aortic aneurysm repair. This finding was of sufficient interest to prompt a paper for publication but we did point out that further studies are required to evaluate the mechanism of action of mannitol and these are currently ongoing.

We agree that the urinary albumin:creatinine ratio (ACR) is a marker of renovascular permeability and as such can also be described as a function marker of glomerular damage[1]. We have not stated in our manuscript that ACR is a measure of structural glomerular damage. Urinary *N*-acetyl-glucosaminidase (NAG) excretion was also measured as a marker of proximal tubular damage and this showed a similar trend to the ACR data.

Messrs Tisi and Shearman raise an interesting hypothesis in the final paragraph of their letter that can only be tested by further clinical studies.

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