It is clear that these are markedly contrasting findings and as such we feel that reference to this earlier study is imperative. The issue is an important one, as many centres administer large doses of methylprednisolone during operation to patients undergoing heart and lung transplantation.

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1. Inaba H, Kochi A, Yorozu S. Suppression by methyl-
prednisolone of augmented plasma endotoxin-like activity
and interleukin-6 during cardiopulmonary bypass. British
2. Andersen L, Baek L, Thomsen B, Rasmussen J. Effect of methyl-
prednisolone on endotoxemia and complement ac-
tivation during cardiac surgery. Journal of Cardiothoracic

Sir,—Our article was published as a short communication, the number of references was limited and therefore we were unable to include the report by Andersen and colleagues [1]. In this study, plasma concentration of endotoxin was measured using a con-
vencional LAL assay which demonstrated that methyl-
prednisolone augmented the increase in plasma endotoxin during
cardiopulmonary bypass. Despite minor differences in our study, we cannot provide a clear explanation for the contrasting findings. A different disagreement may exist between the latest study by Jansen and colleagues [2] and ours. They demonstrated that concentrations of tumor necrosis factor (TNF) increased
significantly after release of the aortic cross-clamp and that dexamethasone treatment effectively inhibited the increase in
TNF. In our study, cardiopulmonary bypass did not significantly
increase concentrations of TNF.

The effect of corticosteroids on pathological alterations during
cardiopulmonary bypass is still controversial. A randomized study in a large number of subjects is necessary to determine the influence of corticosteroids. In addition, standardization of the assays for plasma concentrations of endotoxin, endotoxin-like activity and cytokines will improve comparability between studies, as the reported concentrations of endotoxin and cytokines may be dependent on the particular method used.

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ACTH treatment for post-lumbar puncture headache

Sir,—In a recent letter [1], Dr Collier described his use of ACTH in relieving post-lumbar puncture headache. As he noted, it is odd that such a simple and effective technique seems entirely unknown and there is no reference to it in any recent review. I have used this method successfully since 1987. In a personal series of about 20 patients, the method has produced at least as good results as are claimed for blood patch, being 70% effective. The diagnostic criteria that I applied before starting treatment included bilateral headache starting within 72 h of a dural puncture, which was diminished by lying supine and bilateral jugular compression. Photothobia, neck stiffness, nausea and vomiting, and signs of dehydration may be present. Papilloedema and fever contra-
dicate ACTH as a primary therapy. All patients had tried supine bed rest, forcing oral fluids and various analgesics, including i.v. caffeine, without improvement. ACTH has been effective after failed blood patch. Similarly, a second reinfusion of the original dose for recurrent headache after 24 h has been effective in two patients.

The method finally adopted differs from Dr Collier's in that ACTH 1.5 u. kg"^"^ in is infused over 1 h in 1 or 2 litres of lactated
Ringer's solution. The infusion gives temporary headache relief so that the overall response tends to be biphasic. There is usually complete relief of symptoms at the end of infusion, which may wear off gradually if the patient walks immediately after the procedure. During the next 6–12 h final relief is established. This suggests a delayed onset for ACTH which would be consistent with its known pattern of action.

One may only speculate on any mechanism. It is possible that spinal puncture headache is found in a minority group with a different metabolic response to stress or trauma. One assumes that the release of aldosterone and mineralocorticoids is somehow involved in producing fluid and salt retention, that glucocorticoids have analgesic properties and that long-term glucocorticoids are used to lower intracranial pressure. It may be that active CSF secretion and absorption processes are affected by ACTH or some of the drugs used in spinal anaesthesia. Does the delay in onset of headache support a hormononal mechanism at work? Whatever the cause, intuitively a single dose of ACTH should be less hazardous than injecting blood into the extradural space. The only side effect that I have seen is an altered requirement for hypoglycaemic agents in diabetic patients.

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Hypertonic saline prehydration before spinal anaesthesia

Sir,—I read with interest the article by Baraka and colleagues [1] on the role of hypertonic saline prehydration in patients undergoing transurethral resection of the prostate under spinal anaesthesia. The authors have shown that prehydration with 3 % saline 7 ml kg"^-1 decreased the incidence of hypotension after spinal anaesthesia. This particular observation is encouraging. However, are the authors justified in using hypertonic saline prehydration in the population of elderly patients (mean age > 60 yr) with age-associated diminished cardiac reserve [2]? Is it likely that several of these patients were suffering from hyper
tension and coronary artery disease, and were prone to congestive cardiac failure? The authors themselves observed a
significant increase in CVP after hypertonic saline prehydration.

Patients in this study were premedicated with atropine 0.4 mg i.m. and diazepam 5 mg orally. It would have been preferable to ensure that the patients were adequately sedated rather than administering atropine, which is associated with undesirable side effects, such as tachycardia and central nervous system excitation [3], especially in elderly patients.

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Sir,—Our report has shown that in patients undergoing transurethral resection of the prostate, prehydration with 3 %
saline 7 ml kg⁻¹ decreased the incidence and degree of hypotension after spinal anaesthesia [1].

Hypotension after spinal anaesthesia results from functional sympathetic denervation, not only of the arterial and arteriolar circulation, but also of the large veins and venules. Venodilation can increase significantly venous capacitance with a consequent decrease in venous return and cardiac output [2]. The rationale of prehydration is to expand the plasma volume. However, isotonic crystalloid solutions, in the volumes commonly used, may not be effective, as about 75% of the solution diffuses extravascularly into the interstitial space. In contrast, hypertonic saline is more effective as it can induce instantaneous mobilization of endogenous fluid along the osmotic gradient from the intracellular to the extracellular space [3]. However, I agree that the technique must be used carefully in hypertensive patients, and also in patients who have, or are prone to develop, congestive heart failure. I agree also that patients should be adequately sedated and we have found that the administration of diazepam 5 mg orally achieves this aim.


Auto-PEEP, high frequency jet ventilation

Sir,—I refer to the article by Mansfield, Pugh and Brockway [1] where the authors felt that most of the problems encountered by the patient were attributed directly to the tracheotomy, rather than the ventilatory technique.

Of particular concern were the frequency and inspiratory time used for high frequency jet ventilation. The same settings were quoted twice by the authors: driving pressure = 17 bar (or 2465 psi), frequency (f) = 150 b.p.m and inspiratory time = 0.3 s. A simple calculation reveals that at a ventilatory frequency of 150 b.p.m., each ventilatory cycle has a duration of 0.4 s. If the inspiratory time is 0.3 s, then the expiratory time is 0.1 s. This gives an inspiration to expiration (I:E) ratio of 3:1. Tidal volume delivered during high frequency jet ventilation is dependent upon several factors, including driving pressure, the dimensions and arrangement of the connecting tubes, injector cannula size, I:E ratio, the nature of gas entrainment and the impedance of the respiratory system [2]. With an I:E ratio of 3:1, undoubtedly gas trapping occurs; this can increase alveolar pressure without affecting proximal airway pressure, as a result of impedance in expiratory flow [3]. The difference between alveolar pressure and proximal airway pressure at end-expiration has been called auto-PEEP.

Some of the findings of the study [3] were that when the driving pressure was held constant, longer inspiratory time values were associated with significant increases in auto-PEEP and the gas trapping effect was maximized as the time constant lengthened. Even though there was no mention of the condition of the patient's lungs [1] at the time of tumour resection, as he used to smoke 20 cigarettes per day, it is possible that he suffered from chronic obstructive pulmonary disease (COPD). If indeed he had COPD, high frequency jet ventilation with a ventilatory frequency of 150 b.p.m. and a reverse I:E ratio of 3:1 could have resulted in auto-PEEP. If the presence of auto-PEEP goes unnoticed and the level is allowed to increase, the alveolar membrane disrupts as a result of overtensioning. It would come as no surprise that the patient developed pneumomediastinum and pneumothorax, first left-sided then right-sided. The bronchoscopic findings of "a grossly abnormal trachea...marked ballooning of the (tracheal) mucosa" could well have been evidence of gas tracking up along the tracheobronchial tree as a result of air leak from the alveoli. The tracheotomy could have been the cause of this, but the cuff was still inflated and the tube was positioned correctly, as confirmed by fibrescopy.

The patient had high frequency jet ventilation for another 4 days with a break from assisted ventilation for 24 h. Even though the patient had cheat tubes inserted to drain the pneumothoraces, a constant auto-PEEP was probably maintained in the damaged alveoli causing a leak. This would be of no help in terms of regeneration and repair of the alveolar membrane and probably led to the development of bilateral bronchopleural fistula in the patient.

Do other users of high frequency jet ventilation share the same opinion as to the probable cause of ventilatory complications?

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Sir,—Thank you for the opportunity to respond to Dr Hwang's letter. Unfortunately, the inspiratory time in our article was twice quoted incorrectly as 0.3 s, instead of a 0.3 fraction of the respiratory cycle. We apologise for this error which implies that Dr Hwang's correctly stated points regarding reverse I:E ratio ventilation are no longer applicable.

Despite the patient's smoking habit he did not exhibit the clinical manifestations of chronic obstructive airways disease. The time constant of his lungs can therefore be assumed to lie within normal limits. This would suggest that the development of significant auto-PEEP or air trapping was unlikely [1].

The tracheotomy tube was seen to be located in the trachea on fibrescopic examination. The report states however that we were unsure if the cuff was in the trachea and also that the patient could breathe past the tracheotomy (thus probably saving his life at this stage). The airway above the tracheotomy was therefore in direct communication with that below, potentially exposing both to the same ventilatory pressures and patterns. It is also conceivable that the tip of the tracheotomy tube interfered with the lower margins of the extended tracheostoma. Peak inspiratory pressures were measured on the expiratory limb of the T-piece. We therefore stand by our original opinion that the problems which occurred were related primarily to the tracheotomy rather than the ventilatory technique, although a contribution from high frequency jet ventilation cannot be excluded.

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The extradural sieve

Sir,—We read with interest the report by Tehan [1] and the letter by Rashiq and Huston [2] on the abolition of the extradural sieve by administration of extradural opioids. We believe that it would be incorrect to conclude that the use of extradural opioids is unsafe in women with a scarred uterus undergoing "trial of labour" for two reasons. First, we feel that the emphasis on pain as the sentinel sign of uterine rupture is incorrect. In a review of 14 studies of trial of labour from 1980 to 1989 [3], uterine rupture presented with signs and symptoms other than abdominal pain in 76% of patients, with fetal distress presenting by itself in 52% of patients. Thus, symptom was the most sensitive sign of uterine rupture rather than abdominal pain. Furthermore, five of 14 complete ruptures in patients who had received extradural anaesthesia presented with abdominal pain compared with four of 23 in patients who had not received extradural anaesthesia. Second, the abolition of pain described by Tehan and by Rashiq and Huston occurred after bolus doses of