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## Correspondence

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### Time course of neurone-specific enolase and S-100 protein release during and after coronary artery bypass grafting

Editor—Gao, Harris and Sapsed-Byrne's article carefully maps out S-100 protein release during and after coronary artery surgery and raises some interesting points.<sup>1</sup> Intriguingly, they found that concentrations of S-100 began to increase even before cardiopulmonary bypass (CPB) had begun. A possible explanation for this early increase may have been the inclusion in their study of patients with previous neurological damage as such patients have increased concentrations of S-100 after CPB.<sup>2</sup>

However, their most striking finding is peak concentrations of S-100 at the end of rewarming on CPB. This differs from Von Knobelsdorff and co-workers who found peak concentrations at the end of CPB rather than at the end of rewarming.<sup>3</sup> Differences in sampling times or rewarming strategies between the studies may account for this discrepancy. Tonninger and colleagues were unable to detect any difference in S-100 concentrations 30 min after CPB between patients undergoing normothermic or mild hypothermic CPB.<sup>4</sup> However, active warming of the normothermic group during CPB and the late sampling time (missing an earlier peak) may account for the failure to detect any difference. For these reasons, inclusion of the rate of increase of S-100 in relation to the rate of rewarming in Gao, Harris and Sapsed-Byrne's study would have been of value.

Most importantly, recent literature does not substantiate their conclusion that S-100 samples should be obtained at the end of CPB for investigation of cerebral damage.<sup>2 5 6</sup> Grocott and co-workers used the maximum S-100 concentrations of four samples (during and after operation) to investigate the association between brain damage and cardiac surgery.<sup>5</sup> S-100 showed a weak, albeit significant correlation with cognitive ( $P = 0.0118$ ,  $r^2 = 0.047$ ) and neurological ( $P = 0.026$ ,  $r^2 = 0.039$ ) outcomes on univariate analysis. Inclusion of intraoperative together with postoperative samples may account for this. Jonsson and colleagues found S-100 concentrations at the end of CPB to be associated with age and duration of perfusion but not cerebral outcome, whereas concentrations 5–48 h after operation were associated with neurological damage.<sup>2</sup> Moreover, Sandstrom and colleagues found that patients with memory impairment had significantly higher S-100 concentrations 7 h after termination of CPB but there were no significant differences at the end of CPB.<sup>6</sup>

Thus the emerging picture suggests that the causes of S-100 release towards the end of CPB may well differ from

those in the early postoperative period and it is far from clear whether this peak concentration of S-100 simply represents neuronal damage. Increased permeability of the blood–brain barrier or washout of S-100 as cerebral vasodilatation occurs during rewarming may contribute to peak concentrations and perhaps it is a case of 'not being able to see the wood for the trees'. While the peak concentration at the end of CPB may be most striking, it appears to have less prognostic value than postoperative increase in S-100. Further investigation is required to establish the pattern of S-100 release in the early postoperative period and its association with cerebral damage.

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- 3 Von Knobelsdorff G, Paris A, Tonner PH, Scholz J. Mild hypercapnoea during rewarming of hypothermic cardiopulmonary bypass does not effect S-100 protein release. *Br J Anaesth* 1998; **80**: A70
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Editor—I read with great interest the article by Gao, Harris and Sapsed-Byrne.<sup>1</sup> We have recently studied the relationship between S-100  $\beta$  protein concentration and neurophysiological outcome in 10 patients undergoing coronary artery bypass grafting (CABG). Serious carotid stenosis was excluded by Doppler echocardiography before operation in all patients. Anaesthesia was induced with etomidate, fentanyl and vecuronium, and maintained with isoflurane and nitrous oxide in oxygen. Cardiopulmonary bypass (CPB) was performed under moderate hypothermia (28°C) using a membranous oxygenator, arterial line filter and non-pulsatile perfusion. A neurologist performed a neurological examination on all patients before surgery and

on the third postoperative day. The neuropsychiatric test battery consisting of the mini mental state examination (MME) and the visual aural digit span test (VADST) was applied to patients before operation and on days 3 and 6 after operation. Blood samples for analysis of concentrations of S-100  $\beta$  protein were obtained before induction of anaesthesia, before CPB, after 15 min of CPB, after CPB and 24 h after operation.

Postoperative neurological examination of all patients was normal. The MME revealed minimal deterioration on the third postoperative day. S-100  $\beta$  protein concentrations increased at initiation of CPB, reached maximal concentrations after CPB and declined to basal levels 24 h after operation. VADST performance declined significantly on the third day and returned to baseline values on day 6. While S-100  $\beta$  protein concentration showed a significant strong correlation with both cross-clamp time (CCT,  $r = +0.67$ ) and CPB time ( $r = +0.74$ ), VADST performance showed a mild correlation with CCT ( $r = -0.39$ ) and CPB time ( $r = -0.43$ ). A moderate correlation was observed between S-100  $\beta$  and VADST performance ( $r = -0.43$ ).

We concluded that because of its specificity, known kinetics and good correlation with neuropsychiatric tests, S-100  $\beta$  protein may be a useful biochemical marker for cerebral injury in patients undergoing CABG.

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Editor—Drs Kanbak and Öcal found a similar time course to ours for S-100 release during CABG, and have related it to neuropsychological tests. It is interesting that they found a correlation with operative times in only 10 patients, as most authorities would suggest 50+ patients per group because of the high variability. While the specificity of increased serum concentrations of S-100 after stroke is well established, recent studies are questioning the significance of *intraoperative* S-100. We would suggest that their conclusion may be optimistic: while the possibility of an *intraoperative* marker for cerebral damage is very attractive, the role of S-100 requires more clarification.

We thank Drs Robson and Alston for their comments. We find it strange that there is an increase in S-100 concentrations before CPB, even if patients have previous cerebral damage, unless anaesthesia alone is thought to worsen existing damage. With our procedure, patients come off bypass when they are fully warmed (38°C), therefore 'end of rewarming' and coming off bypass are almost simultaneous, whereas 'rewarming' for Von Knobelsdorff and colleagues was at 36°C. Our next measurement was 15 min after bypass as cerebral perfusion during weaning from bypass is variable and unstable. Unlike Tonninger and

colleagues, we found a significant difference in S-100 and neurone-specific enolase (NSE) between bypass at 28°C and 37°C (although less at 32°C), with peaks at the end of bypass.<sup>1</sup> Tonninger and colleagues made no measurements between 60 min of bypass and 30 min after bypass and therefore would probably have missed peak concentrations. We did not obtain measurements at 30 min after bypass, but if our data can be interpolated, by 30 min S-100 concentrations would already have fallen sharply and any difference might be missed. This was the reason why we felt that delineating the time course of S-100 release was important.

We agree that there is now a suggestion that intraoperative increases in neuroproteins may not be related to postoperative neuropsychological deficits, but there are few data at present. Large studies are needed for such a comparison, and we await them with interest. However, increased S-100  $\beta$  has been regarded previously as specific for glial (not neuronal) cell damage, and we found that both S-100 and NSE increased around CPB. The pathophysiology of cerebral damage is complex, and it is equally possible that intraoperative increases reflect cerebral 'stunning' rather than infarction, in line with  $SjO_2$  data suggesting cerebral ischaemia during rewarming.<sup>2</sup> We agree that the significance of intraoperative increases in neuroproteins needs further investigation.

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2 Groughwell N, Smith LR, Quill T, et al. The effect of temperature on cerebral metabolism and blood flow in adults during cardiopulmonary bypass. *J Thorac Cardiovasc Surg* 1992; **103**: 549–54

D. N. F. Harris

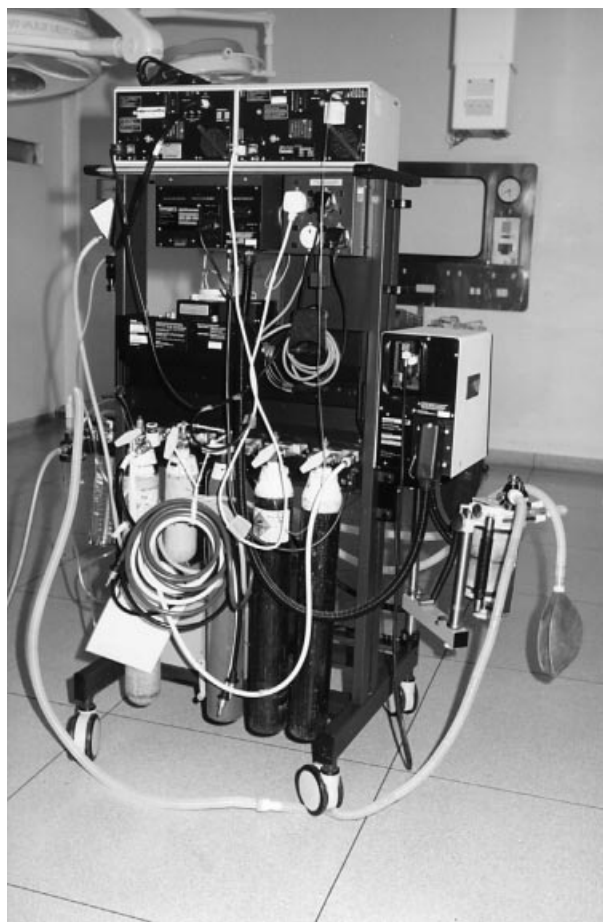
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### Hidden hazards of scavenging

Editor—While we appreciate all the benefits of scavenging systems for personnel and the environment, the potential hazards to our patients must not be forgotten. I was reminded of this recently during a critical incident involving a 31-year-old female undergoing an appendectomy.

After induction of anaesthesia in the anaesthetic room, the patient was transferred from the bed onto the operating table, during which time the anaesthetic machine was moved to make space for equipment. On starting artificial ventilation, I immediately noticed peak inflation pressures of 41 cm H<sub>2</sub>O, returning to a baseline of 17 cm H<sub>2</sub>O, despite having no positive end-expiratory pressure (PEEP)



**Fig 1** The wheel of the anaesthetic machine can be seen occluding the scavenging transfer tubing.

selected on the Ohmeda 7900 ventilator. The same difficulties were encountered during hand ventilation, even with the adjustable pressure-limiting valve completely open. Checking the patient and breathing system elicited no clues as to the cause of the problem. Disconnecting the expiratory limb scavenging hose relieved the pressure and on close inspection, I found the wheel of the anaesthetic machine to be occluding the scavenging transfer tubing (Fig. 1). The obstruction was removed quickly and the patient sustained no harmful pulmonary barotrauma.

Despite the efficient safety mechanisms installed on the Ohmeda AGSS evacuation system receiving unit, neither the reservoir nor the safety valves and pressure balancing devices protect against problems developing in the proximally situated collecting tubing. The hazards of scavenging are well-established.<sup>1-3</sup>

In this case, it was not the scavenging system that was defective but the assembly of the evacuation system. Transfer tubing must be stowed so that it does not trail on the floor. If this is not the case, the anaesthetist must correct this immediately and notify the maintenance technician.<sup>4</sup> Such obstruction to a system can be prevented by the use of kink-resistant rather than standard plastic tubing and

by exercising caution whenever the anaesthetic machine is moved.

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- 1 Gray WM. Scavenging equipment. *Br J Anaesth* 1985; **57**: 685-95
- 2 Mantia AM. Gas scavenging systems. *Anesth Analg* 1982; **61**: 162-4
- 3 Tavakoli M, Habeeb A. Two hazards of gas scavenging. *Anesth Analg* 1978; **57**: 286-7
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### **Analgesic efficacy of paracetamol and diclofenac in children receiving PCA morphine**

Editor—Regarding the article by Morton and O'Brien,<sup>1</sup> I believe a few comments are warranted. Analgesic requirements for appendicectomy are variable. Two major determinants of the degree of pain experienced by a child are the degree of inflammation of the appendix, including the presence of peritonitis, and the nature of the surgery (difficulty, length of incision, surgical expertise, etc.). Neither of these was mentioned by the authors and in a study with only 20 patients per group, a predominance of severe disease in any one group would affect the results. Indeed, a straightforward appendicectomy often requires very little opioid and allows for discharge home the following day. Two of the four groups in this study, for example, averaged less than one bolus per hour of PCA morphine.

The age range of the study was 5-13 yr. Although there is evidence of PCA use in younger children,<sup>2</sup> I would question the validity of morphine consumption data with this technique in children less than 7 yr of age.

The authors did not blind the observer of the pain assessment to the technique used and did not mention who was the observer or scorer of the pain. If it was the child, the validity of the score in the younger age group must also be questioned.

Paracetamol, as stated correctly by the authors, is now used in a larger, more appropriate, dose. What the authors failed to mention was that rectal paracetamol may not reach peak therapeutic concentrations until 2 h after administration,<sup>3</sup> potentially increasing the number of high pain scores in these groups in the early postoperative period. In a study with very low pain scores, low morphine consumption and low numbers, the timing of an adjunctive therapy is critical. To the authors' recommendation that further study of the appropriate paracetamol dose is warranted, I would add that the surgical group studied should be one that would show benefit from both opioid and non-opioid analgesia (i.e. one with moderate to high opioid requirements).

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- 1 Morton NS, O'Brien K. Analgesic efficacy of paracetamol and diclofenac in children receiving PCA morphine. *Br J Anaesth* 1999; **82**: 715–17
- 2 Gaukroger P. Patient controlled analgesia in children. *Anaesth Intensive Care* 1989; **17**: 264–83
- 3 Anderson BJ, Holford NHG. Rectal paracetamol dosing regimes: determination by computer simulation. *Paediatr Anaesth* 1997; **7**: 451–5

Editor—We thank Dr Ragg for his interest in our paper. He makes several points, both in his letter and also between the lines!

I agree with his implication that we did not have total control over all of the potential variables in this study, in particular in the blinding of the pain scorer. In school-aged children, such as those in our study, the scores are self-reported by the child in response to questioning by the bedside nurse. Children of school-age can do this reliably if they know they are not going to receive an i.m. injection of analgesia. Dr Ragg implies that younger children cannot do this, but literature more recent than the reference from 10 yr ago which he cites, suggests otherwise. From previous studies using similar methodology, we were able to differentiate between PCA regimens in terms of efficacy and adverse effects using 15–20 children per group. For each patient we have a large number of datum points from the hourly assessments carried out. The low morphine consumption values mentioned by Dr Ragg were in those groups who received diclofenac, and this is really the point of the study. The variability in morphine requirements between patients was nearly 20-fold ( $4\text{--}80\ \mu\text{g kg}^{-1}\ \text{h}^{-1}$ ) which demonstrates the advantage of PCA in allowing self-titration. I agree that straightforward removal of a normal appendix tends to be less painful but this is not always the case; patients' pain thresholds vary approximately 10-fold after abdominal surgery. Dr Ragg implies that PCA is an overkill for appendectomy but our experience is that if analgesia is titrated against movement pain scores, a significant proportion of children need significant amounts of opioid. The amount can be reduced but not eliminated by concurrent NSAID administration, but some children still require an average dose of morphine up to  $50\ \mu\text{g kg}^{-1}\ \text{h}^{-1}$  in the first 24 h. The surgical and anaesthetic techniques in our study were standardized and the incidence of perforated appendices with peritonitis was similar across the four groups (data we should have included in our report). We acknowledge that the paracetamol dose used in our study is now regarded as too low and therefore we believe further studies are worthwhile using this methodology with the currently recommended higher doses of paracetamol. Our study proves the analgesic efficacy of NSAID in children

and the methodology can be used to compare multimodal analgesia regimens. The choice of surgical group is open to debate and personal preference. As a result of this study, we feel the ethically acceptable *control* group in future studies of morphine PCA in children should be PCA morphine with concurrent diclofenac or comparable NSAID, unless these agents are contraindicated. A morphine-alone PCA group is less beneficial. How a no-PCA group would perform when assessed in the same detailed way may be worthy of study using similar methodology as clinical impressions can be deceptive!

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### Central nerve block and thromboprophylaxis

Editor—I read with interest the editorial on central nerve block and thromboprophylaxis.<sup>1</sup> I have just spent a year working in Melbourne where the topic of low-molecular weight heparins (LMWH) in conjunction with neuraxial regional anaesthesia was also creating much debate. In response, guidelines by the Victorian Consultative Council on Anaesthetic Mortality and Morbidity were published.<sup>2</sup> They suggest that *both* insertion and removal of epidural catheters should be performed at least 12 h after a dose of LMWH *and* the subsequent dose of LMWH should be withheld for 6–12 h. Less specific is their recommendation of 'regular' assessment of the patient's neurology for motor block, continuing until the patient is ambulant or for 2–3 days after catheter removal. Quite a commitment!

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- 1 Checketts MR, Wildsmith JAW. Central nerve block and thromboprophylaxis—is there a problem? *Br J Anaesth* 1999; **82**: 164–7
- 2 Scott D. Notes on the use of low molecular weight heparins in conjunction with neuraxial regional anaesthesia. Victorian Consultative Council on Morbidity and Mortality Information Bulletin, December 1998

Editor—Checketts and Wildsmith's editorial<sup>1</sup> revisits an issue which is relevant to everyday practice, and provides an excellent up-to-date review of the literature. I am interested in the subject as at present there are no guidelines and, in particular, no consensus about the timing of neuroaxial block insertion and epidural catheter removal in the presence of low-molecular weight heparin (LMWH) thromboprophylaxis. I was pleased to see that the authors

endorsed the view which I took in my review article<sup>2</sup> regarding the frequency of enoxaparin dosing and a 'safe' interval, namely that 'When a twice-daily regimen has been started, there may never be a 'safe' time in the day either to perform the block or to remove the catheter'.<sup>1</sup>

The authors do not express any views regarding a 'safe' interval when a once-daily thromboprophylactic (low-dose) regimen is in use. Such a regimen should be standard in this country (e.g. enoxaparin 20 mg or 40 mg once daily), and it will be interesting to see if the incidence of vertebral canal haematoma in the USA has decreased now that the FDA has approved the lower European dose schedule. The authors cite Tryba and Wedel's article<sup>3</sup> which was a detailed review of the problems with neuroaxial block and enoxaparin. The review also contained specific guidelines on *thromboprophylactic* enoxaparin which include, among others, the following recommendation: 'enoxaparin and other LMWHs should not be administered within 8 hours before or after neuroaxial block or epidural catheter removal'.

There was a case report<sup>4</sup> in the same issue as the editorial,<sup>1</sup> and another in *Anaesthesia*,<sup>5</sup> concerning epidural haematoma developing after epidural catheter removal in ambulating patients. It should be noted that one patient<sup>4</sup> possibly developed the first small epidural haematoma after epidural catheter removal 3 h after the last dose of unfractionated heparin. Subsequent paraplegia developed several days later after *high-dose (therapeutic)* enoxaparin. The other patient<sup>5</sup> developed an epidural haematoma when receiving therapeutic doses of heparin after being moved out of bed. Such cases indicate that great caution must be exercised when heparin is given (or about to be given) in therapeutic (high) doses. When examining other case reports<sup>6</sup> and the review by Wysowski and colleagues<sup>7</sup>, it appears that moderate- or high-dose LMWH or unfractionated heparin clearly constitutes a substantial risk.

With regard to anaesthetic management, evidence-based guidelines exist<sup>8</sup> for low-dose thromboprophylactic unfractionated heparin given twice daily, recommending a 4–6 h interval between a dose of heparin and neuroaxial block insertion or removal. With regard to low-dose, thromboprophylactic LMWH given once daily, do the authors endorse Tryba and Wedel's recommendation<sup>3</sup> of an 8-h interval, or if not, what interval do they recommend?

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- 1 Checketts MR, Wildsmith JAW. Central nerve block and thromboprophylaxis—is there a problem? *Br J Anaesth* 1999; **82**: 164–7
- 2 Dolenska S. Neuroaxial blocks and LMWH thromboprophylaxis. *Hosp Med* 1998; **59**: 940–3
- 3 Tryba M, Wedel DJ. Central neuraxial block and low molecular weight heparin (enoxaparin): lessons learned from different

dosage regimens in two continents. *Acta Anaesthesiol Scand* 1997; **41**: 100–3

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Editor—Thank you for the opportunity to reply to the correspondence arising from our recent editorial. There are no absolutes in this area, but we would make the following comments. In principle, we agree with the American Society of Regional Anesthesia Consensus statement,<sup>1</sup> which recommends an interval of *at least* 10–12 h after LMWH thromboprophylactic dosing before performing a central nerve block or removing an indwelling epidural catheter. The Australian guidelines quoted by Dr Gorton are similar, although it may not be necessary to wait 6–12 h after catheter removal before administering the next LMWH dose. Our practice is to remove an epidural catheter 2 h before the next LMWH dose when anti-Xa activity is low, remembering that peak activity occurs 3–4 h after s.c. injection. As Dr Dolenska reminds us, *therapeutic* doses of LMWH or unfractionated heparin (UH) greatly increase the risk of vertebral canal bleeding, and central nerve block is virtually contraindicated. If there are very strong clinical reasons to commence therapeutic LMWH or UH when an epidural catheter is *in situ*, there are two options: either remove the catheter a minimum of 2 h before heparin is started or, if this is not feasible, leave the catheter undisturbed until all anticoagulants have been discontinued and clotting has returned to normal. This means at least 24 h later if a therapeutic dose of LMWH has been given. If UH has been given, check the APTT 4–6 h after discontinuation and before catheter removal. If coagulation is not normal, the 'commitment' to regular neurological assessment is essential.

We have no experience of thrombelastography, but it may be a useful technique to assess coagulation before central nerve block or catheter removal in high-risk patients, as suggested by Wilkes and colleagues.<sup>3</sup> However, our understanding is that the equipment is not cheap or readily available, and it remains to be proved as a reliable method of measuring LMWH activity in clinical practice.

Appropriate thromboprophylaxis must be administered to surgical patients, but it is important to look critically at the evidence of advocating LMWH over low-dose UH. The only setting in which LMWH has been shown to be unequivocally superior to UH is total hip joint replacement

surgery<sup>2</sup> and this point should be considered when devising local thromboprophylaxis protocols.

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- 1 Horlocker TT, Wedel DJ. Neuraxial block and low-molecular weight heparin: balancing perioperative analgesia and thromboprophylaxis. *Reg Anesth Pain Med* 1998; 23: 164–77
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### Acute normovolaemic haemodilution vs controlled hypotension for blood conservation

Editor—Having read with interest the well-designed study by Boldt and colleagues,<sup>1</sup> which addresses the important issue of perioperative blood conservation, we wish to comment on the analysis and interpretation of the results.

First, it appears that patients in the hypotension group received significantly less non-cellular fluids (mean 2230 ml) in the perioperative period compared with patients in the ANH group (mean 6510 ml) and control group (mean 6800 ml) (see Table 1), which would not account for the difference in blood loss (see Fig. 3). Thus patients in the hypotensive group were less haemodiluted and were losing more concentrated blood than patients in the ANH and control groups.

It is the low concentration of cellular components in blood lost during operation, and consequently overall reduction in the loss of red cell mass, that is the essence of ANH and its blood saving efficiency.<sup>2–4</sup> Therefore, the statement that a loss of 1260 ml of (concentrated) blood in the hypotension group is significantly lower than a loss of 1820 ml of (haemodiluted) blood in the ANH group (see Fig. 3) must be questioned, as the precise calculations depend on the packed cell volume (PCV) of the aspirates. It would be more accurate to express blood loss as a percentage of red cell mass rather than absolute volume.

Second, the hypotension patients were reported to receive overall significantly fewer units of packed red cells (14 vs 21 in the ANH group and 28 in the control group). However, analysis of the use of allogeneic blood (see Fig. 3) reveals a similar number of units transfused during operation in the hypotension and ANH groups (5 and 6, respectively), the difference occurring between day 1 and day 6. Was there any difference in the transfusion trigger between the three groups in the postoperative period?

The efficacy of ANH as a blood conservation method is proportional to the amount of blood withdrawn.<sup>2–4</sup>

Withdrawing 15 ml kg<sup>-1</sup> of blood for ANH, regardless of the initial PCV, may be practical but it will not achieve the maximum blood saving benefit, particularly in patients with a higher preoperative PCV who can afford a greater ANH deposit. The most frequently used and recommended formula for the volume of blood to be withdrawn as ANH is:  $V = EBV \times (Ho - Ht)/Hav^{2.3.5}$  (where EBV = estimated blood volume, and Ho = initial, Ht = desired and Hav = average haematocrit).

Finally, having demonstrated the value of controlled hypotension in reducing both intraoperative blood loss and requirements for allogeneic blood transfusion, one can speculate that a combination of two not mutually exclusive methods, namely controlled hypotension and ANH, can produce even greater blood savings, as pointed out in the related editorial.<sup>6</sup>

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- 1 Boldt J, Weber A, Mailer K, Papsdorf M, Schuster P. Acute normovolaemic haemodilution vs controlled hypotension for reducing the use of allogeneic blood in patients undergoing radical prostatectomy. *Br J Anaesth* 1999; 82: 170–4
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- 5 British Committee for Standards in Haematology Blood Transfusion Task Force. Guidelines for autologous transfusion. II. Perioperative haemodilution and cell salvage. *Br J Anaesth* 1997; 78: 768–71
- 6 Levack ID, Gillon J. Intraoperative conservation of red cell mass: controlled hypotension or haemodilution—not necessarily mutually exclusive? *Br J Anaesth* 1999; 82: 161–3

Editor—In the ANH patients, significantly more gelatin was infused than in the hypotension or control group. Use of crystalloids was similar in all groups. Unfortunately, in the hypotension group an incorrect value was printed in Table 1 (960 ml instead of 3960 ml, see corrected version of Table 1).

Table 1

	ANH group (n = 20)	Hypotension group (n = 20)	Control group (n = 20)
Perioperative volume infusion (ml)			
Crystalloids	4060 (670)	3960 (490)	4210 (890)
Gelatin	2450 (550)*	1270 (530)	1590 (540)

The total amount of transfused allogeneic blood was significantly lower in the hypotension group. When evaluating the use of blood conservation techniques, it is important to consider not only the intraoperative period, but also the days after surgery. There were no differences in the transfusion triggers during and after operation. The concentration of haemoglobin was more stable in the hypotension patients compared with the control and ANH groups. Wolowczyk, Lamont and Nevin are correct in stating that withdrawing 15 ml kg<sup>-1</sup> of blood will not achieve the maximum blood saving benefit. Adjusting the amount according to the initial packed cell volume (PCV) would be more effective. But study conditions should be comparable for all patients and thus we decided to select a fixed amount of withdrawn blood (15 ml kg<sup>-1</sup>) instead of an individual amount calculated according to the patient's initial PCV.

The comment on the additional use of both techniques is also correct and a study combining both techniques is already in process at our institution.

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### **Glycopyrrolate reduces nausea but is dry mouth acceptable?**

Editor—We were interested to read the article by Ure and colleagues,<sup>1</sup> describing administration of glycopyrrolate 200 µg i.v. before spinal anaesthesia for elective Caesarean section. The dose resulted in a significant reduction in the frequency and severity of nausea and was apparently harmless to the neonate, as evidenced by absence of an adverse effect on Apgar scores. This is an elegantly described and simple technique, seemingly devoid of complications, that can help to abate a common problem.

In our efforts to use this evidence-base, we copied the technique for a small number of patients presenting for Caesarean section. We were immediately impressed not by the reduction in nausea but by the recurring complaint of dry mouth. Glycopyrrolate is a potent antisialogogue,<sup>2</sup> which we unintentionally confirmed using 200 µg in the manner described. Unfortunately, the fasted, awake patient undergoing Caesarean section, who has no immediate prospect of a sip of water, can find this dry mouth a considerable irritation. In one case the patient's tongue was sufficiently adhered to the roof of her mouth so as to make her speech unintelligible. We are surprised that the authors made no report of this unpleasant and perhaps predictable side effect.<sup>3</sup>

Nausea continues to be a problem in this group of patients and the authors have demonstrated a novel technique that

can be helpful. However, this technique, in common with others described,<sup>4</sup> has its potential complications.

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- 1 Ure D, James KS, McNeill M, Booth JV. Glycopyrrolate reduces nausea during spinal anaesthesia for Caesarean section without affecting neonatal outcome. *Br J Anaesth* 1999; **82**: 277–9
- 2 *British National Formulary*. London: British Medical Association and the Royal Pharmaceutical Society of Great Britain 1999; No. **37**: 551
- 3 Mirakhur RK, Dundee JW, Jones CJ. Evaluation of the anticholinergic actions of glycopyrrolate bromide. *Br J Clin Pharmacol* 1978; **5**: 77–84
- 4 Lussos SA, Bader AM, Thornhill ML, Datta S. The antiemetic efficacy of prophylactic metoclopramide for elective Caesarean delivery during spinal anesthesia. *Reg Anesth* 1992; **17**: 126–30

Editor—Thank you for the opportunity to reply to Drs Marples and Wrench. We were interested in their description of problems related to dry mouth when they used glycopyrrolate in the manner we described. We did not report this complication because we did not find that dry mouth was a problem during our study. We have had no problems with this side effect while continuing to use the drug in our routine practice, although we agree it would not be unexpected given the antisialogogue action of glycopyrrolate. Our preoperative fasting policy is evidence-based and allows patients to drink water for up to 2 h before operation. Perhaps a more restrictive fasting policy may explain the difference between their patients and our own. If a patient complains of a dry mouth while having a procedure under regional anaesthetic, it is our normal practice to give them ice to suck while the procedure is ongoing and we find that this reliably cures the complaint. When the patient enters the recovery room they can have a drink of water and most patients get 'tea and toast' soon after returning to the post-natal ward.

D. S. Ure

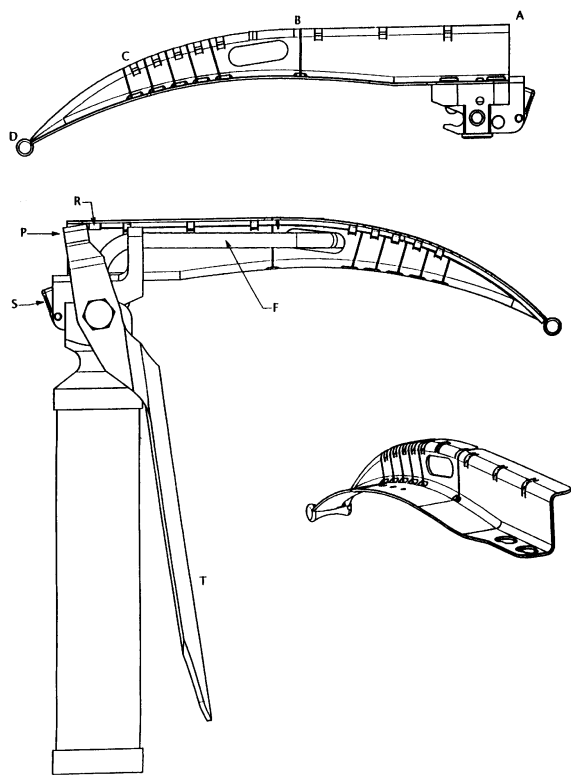
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### **A new laryngoscope with flexible adjustable rigid blade**

Editor—A new laryngoscope that combines a rigid and flexible blade (prototype made by Arco Medic Ltd, Omer,



**Fig 1** Design of the Flexiblade. BC=Flexible intermediate portion of the blade, R=rod, F=fiberoptic, P=pusher and T=trigger.

Israel; US patent application No. 09/125,449) has been designed and tested. This device, the Flexiblade, was designed to overcome the limitations of existing conventional laryngoscopes by providing a dynamically bending yet instantaneously rigid blade. The Flexiblade is composed of two basic parts: a blade with an adjunct trigger and a handle (Fig. 1). The dynamically bending blade combines the features of a flat bending spring and a pushing rod. This combination makes it possible to maintain the rigidity of the blade at any point during its flexion.

The blade comprises a rigid rear portion (AB), a flexible intermediate portion (BC), both L-shaped, and a rigid front portion (CD), which terminates at a tip of small radius. In the intermediate portion BC, the vertical is segmented by six slots. Each slot has an opening at the top and extends downwards to end at the horizontal part of the L. The blade holds a pushing rod (R) whose front end is fixed to the front portion of the blade, CD, and its rear end is free. A 'pusher' (P), which is bearing directly on its free end, achieves the forward movement of the rod. This pusher is the distal part of the trigger (T) that acts as a lever about a fulcrum on the base of the blade. Pulling the trigger drives the pusher and the rod forward causing flexion of the blade. The Flexiblade handle has a standard fitting for a conventional blade and it includes a convector for a fiberoptic light source.

The device is designed to facilitate orotracheal intubation with the patient's head in the neutral position. Insertion is



**Fig 2** X-ray laryngoscopy with the Flexiblade in full curvature. The blade tip is behind the body of the hyoid and the epiglottis is pulled forward.

similar to the insertion of a standard Miller or Macintosh blade. The concave-shaped tip of the blade works as a non-traumatic 'clip' which enters the vallecula and fits its anatomical shape. Squeezing the trigger gently changes the blade curvature from nearly a straight Miller blade into a curved Macintosh blade with unlimited angles between  $9 \pm 1^\circ$  to  $30 \pm 2^\circ$ . This flexibility allows depression of the tongue and the hyoepiglottic ligament, with forward movement of the epiglottis, allowing the vocal cords to be seen without the need for anterior lifting of the mandible or changing the axis of the laryngoscope in the patient's mouth, a manoeuvre which frequently results in damage to the upper teeth (Fig. 2).

Because of the flexibility of the blade, it is easier to introduce into the mouth as its shape can be fitted to the opening of the mouth and the position of the incisors. After using the trigger, the best position may be found for the blade during intubation without seeking the bearing point for the laryngoscope (upper teeth or gum). This feature overcomes one of the disadvantages of the old and newer rigid fibrescopes such as the Upsher, Wu and Bullard that force the patient's upper airway anatomy to conform with the particular shape of the blade.<sup>1</sup> Compared with the McCoy laryngoscope that has a complex levering system of six components, the Flexiblade has only two components: the trigger and the rod.<sup>2</sup>

Movements of the McCoy blade are limited to the last 25 mm from the tip, leaving most of the lower lingual surface of the blade to obstruct the line of view. Movement of the Flexiblade uses six intermediate points (between 3.5 and 10 cm from the tip's end) that change the shape of the entire blade with parallel changes in the view angles.



We have used the Flexiblade successfully in patients with: a small mandibular space (short thyromental distance); a large tongue and small narrow mouth (Mallampati grade III); those with protruding incisor teeth; short-necked patients; and in the morbidly obese, without the need for changing blades.

Comparative studies and design evaluation are now underway. The potential advantages of the Flexiblade were tested in two clinical studies. The preliminary results were presented as a free paper at WARC 99 (Western Anesthesia Residents Conference) in Seattle, April 1999, and as a poster discussion at the 7th ESA Meeting in Amsterdam, May 1999.

Additional advantages include the short learning curve and almost complete elimination of torque movement. The Flexiblade does not replace the flexible fiberoptic laryngoscope, but it reduces the number of occasions where it may be needed. Because of its simplicity and ease of handling, we believe it has a useful place in modern day anaesthesia.

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1 Roberts JT. *Fiberoptic Intubation and Alternative Techniques for Managing the Difficult Airway*. ASA Annual Refresher Course Lectures, 243, Oct 1997

2 McCoy EP, Mirakhur RK. The levering laryngoscope. *Anaesthesia* 1994; **81**: 516–19

## Gelatin may not be the cause of hypercoagulability

Editor—We read with interest the article by Karoutsos and colleagues<sup>1</sup> on the thrombelastogram, revealing hypercoagulability after administration of gelatin solution. Karoutsos and colleagues concluded that gelatin was responsible for the hypercoagulability observed. These findings, which compared the coagulation effects of 3.5% modified gelatin (GEL), 200/0.6 hetastarch (HES) and 5% albumin (ALB) are different from previous similar studies. Egli and colleagues<sup>2</sup> compared the *in vitro* effects of haemodilution with HES, GEL and ALB, and found that all three agents compromised coagulation, especially HES and ALB. Mortelmans and colleagues<sup>3</sup> compared HES and GEL for volume replacement in acute normovolaemic haemodilution and found no difference in coagulation

between the two agents but a slightly higher incidence of abnormal bleeding time and increased bleeding in the HES group. Mortier and colleagues<sup>4</sup> compared *in vitro* haemodilution with HES and GEL and found a minimal change with GEL which was related predominantly to impaired clotting (reduced  $\alpha$  and MA, and prolonged  $k$  on the TEG). Treib and colleagues<sup>5</sup> have suggested a mechanism for altered coagulation reported on prolonged administration of HES.

We would like to postulate that the difference between the results of the previous studies with those of Karoutsos and colleagues is the natural tendency for a hypercoagulable state to develop with blood loss and tissue trauma,<sup>6</sup> irrespective of fluid replacement. In all studies where haemodilution was performed *in vitro*, this natural hypercoagulability was absent and a net impairment of coagulation was observed with HES and ALB, and to a lesser degree with GEL.<sup>2–4</sup> The study of Mortelmans and colleagues only sampled blood for coagulation testing in the first 45 min after induction of anaesthesia and therefore could not show this tendency.<sup>3</sup>

In summary, we suggest that Karoutsos and colleagues' results more likely reflect the inability of GEL to suppress the normal hypercoagulability that develops with blood loss and tissue trauma, instead of GEL inducing hypercoagulability *per se*. In contrast, this normal tendency was suppressed more by HES and ALB, both of which have intrinsic anticoagulant effects, as shown by *in vitro* studies.

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1 Karoutsos S, Nathan N, Lahrimi A, et al. Thrombelastogram reveals hypercoagulability after administration of gelatin solution. *Br J Anaesth* 1999; **82**: 175–7

2 Egli GA, Zollinger A, Seifert D, et al. Effect of progressive haemodilution with hydroxyethyl starch, gelatin and albumin on blood coagulation. *Br J Anaesth* 1997; **78**: 684–9

3 Mortelmans YJ, Vermaut G, Verbruggen AM, et al. Effects of 6% hydroxyethyl starch and 3% modified fluid gelatin on intravascular volume and coagulation during intraoperative hemodilution. *Anesth Analg* 1995; **81**: 1235–42

4 Mortier E, Ongenae M, De Baerdemaeker L, et al. In vitro evaluation of the effect of profound haemodilution with hydroxyethyl starch 6%, modified fluid gelatin 4% and dextran 40 10% on coagulation profile measured by thrombelastography. *Anaesthesia* 1997; **52**: 1061–4

5 Treib J, Haass A, Pindur G, et al. Highly substituted hydroxyethyl starch (HES 200/0.62) leads to type-I von Willebrand syndrome after repeated administration. *Haemostasis* 1996; **26**: 210–13

6 Ng KFJ, Lo JWR. The development of hypercoagulability state, as measured by thrombelastography, associated with intraoperative surgical blood loss. *Anaesth Intensive Care* 1996; **24**: 20–5

Editor—Thank you for the opportunity to reply to Drs Ng and Lo. We found a hypercoagulable profile in one group of patients<sup>1</sup> but whether it was gelatin- or stress-mediated was not demonstrated as no tests were carried out to test this hypothesis. However, there are some clues which suggest gelatin-mediated hypercoagulability: gelatin used *in vivo*<sup>2,3</sup> increased erythrocyte aggregates in blood because of increased blood viscosity at a low shear rate, decrease in primary aggregation time and increased partial dissociation threshold. These findings were obtained in a randomized manner, immediately after haemodilution and before surgery (i.e. before tissue damage). In addition, 20% haemodilution with gelatin<sup>4</sup> increased intrinsic coagulability and speed of clot formation when assessed using the thrombelastogram (TEG), as it decreased *r* and *k* and increased  $\alpha$  angle. This *in vitro* study, which excluded extraneous factors such as stress response and tissue damage, seems to favour a hypercoagulable effect of gelatin.

Furthermore, because of the different materials and methods, we believe that it is not possible to compare the studies cited by Ng and Lo and our own. In both *in vitro* studies, there were several methodological differences: (i) no adjustment of pH and calcium concentration occurred to prevent changes caused by haemodilution with plasma substitutes in the study by Mortier and colleagues<sup>5</sup>; (ii) a different low molecular weight heparin was administered the evening before by Mortier and colleagues<sup>5</sup> and Egli and colleagues<sup>6</sup>; (iii) coagulation in the cup was activated with cellite by Egli and colleagues<sup>6</sup> while we used native blood; and (iv) TEG analysis began 6 min after blood sampling, while our TEG analysis always began within 3 min to avoid clot activation in the syringe.<sup>6</sup> Moreover, at least 30% haemodilution of blood volume was performed in both studies, while we replaced, at most, blood loss of 20% of total blood volume (approximately assumed to be 70 ml kg<sup>-1</sup> in an adult).

The *in vivo* study by Mortelmans and colleagues excludes by its design any comparison with our results: body temperature, of prime importance in haemostasis assessment, was not given throughout the study; albumin was used as a plasma substitute in both groups in addition to the studied starch; and blood substitution, including surgical blood loss and acute normovolaemic haemodilution, approached 80% of total blood volume 4 h after the beginning of the study, which was much greater than in our study.

These differences, and the fact that *in vitro* studies poorly reproduce the multiple *in vivo* interactions leading to coagulation, suggest that no comparison can be made between the two sets of results.

Nevertheless, whatever the cause of this hypercoagulable trend after moderate haemodilution with gelatin, our results suggest that the use of this starch in patients known to suffer from a hypercoagulable state, or prone to thromboembolic disease, is not recommended until more data are available.

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- 1 Karoutsos S, Nathan N, Lahrimi A, et al. Thrombelastogram reveals hypercoagulability after administration of gelatin solution. *Br J Anaesth* 1999; **82**: 175–7
- 2 Freyburger G, Dubreuil M, Boisseau MR, Janvier G. Rheological properties of commonly used plasma substitutes during preoperative normovolaemic acute haemodilution. *Br J Anaesth* 1996; **76**: 519–25
- 3 Dewachter P, Laxenaire MC, Donner M, Kurtz M, Stoltz JF. Effets rhéologiques *in vivo* des substituts plasmatiques. *Ann Fr Anesth Réanim* 1992; **11**: 516–25
- 4 Ruttman TG, James MFM, Viljoen JF. Haemodilution induces a hypercoagulable state. *Br J Anaesth* 1996; **76**: 412–14
- 5 Mortier E, Ongenaes M, De Baerdemaeker L, et al. *In vitro* evaluation of the effect of profound haemodilution with hydroxyethyl starch 6%, modified fluid gelatin 4% and dextran 40 10% on coagulation profile measured by thrombelastography. *Anaesthesia* 1997; **52**: 1061–4
- 6 Egli GA, Zollinger A, Seifert D, et al. Effect of progressive haemodilution with hydroxyethyl starch, gelatin and albumin on blood coagulation. *Br J Anaesth* 1997; **78**: 684–9
- 7 Mortelmans YJ, Vermaut G, Verbruggen AM, et al. Effects of 6% hydroxyethyl starch and 3% modified fluid gelatin on intravascular volume and coagulation during intraoperative hemodilution. *Anesth Analg* 1995; **81**: 1235–42

## Metformin and perioperative risk

Editor—Lactic acidosis is a rare but well recognized complication of biguanide therapy, a treatment used commonly in the management of type 2 diabetes. Metformin-associated lactic acidosis (MALA) has an average case incidence of 0.03 per 1000 patient years, which is 10–20 times lower than that of phenformin (which was withdrawn from several countries for this reason in the 1970s).<sup>1</sup> Although rare, MALA remains a serious yet potentially avoidable complication of metformin therapy with a mortality of 50%.<sup>1</sup>

The mechanism whereby metformin causes lactic acidosis is complex but is thought to be mainly a result of a shift in the intracellular redox potential away from aerobic to anaerobic metabolism, leading to an increase in cellular lactate production.<sup>2</sup> As metformin is excreted by the kidneys, renal impairment is the major risk factor precipitating MALA, although other risk factors such as sepsis, acute myocardial infarction, hepatic impairment and respiratory conditions leading to hypoxaemia are also important. Nephrotoxic drugs have been implicated.<sup>3</sup>

Although surgery has never been identified as a specific cause of MALA, metformin-treated patients are at risk of

developing it as a result of perioperative complications. These include hypotension related to induction of anaesthesia or blood loss, and conditions such as myocardial ischaemia and sepsis, which are more common in diabetic patients in the perioperative period. This was highlighted by Mercker and colleagues in a middle-aged diabetic man treated with low-dose metformin 500 mg once daily.<sup>4</sup> After abdominal wall hernia repair, he developed pneumonia, respiratory failure and acute renal failure. This resulted in severe lactic acidosis with a fatal outcome.<sup>4</sup>

There is currently little information in the anaesthesia literature regarding the perioperative management of metformin-treated diabetic patients, although it has been suggested that metformin therapy should be withheld 2 days before surgery.<sup>5</sup> This recommendation is not supported by the pharmacokinetics of this biguanide. Metformin has a short half-life of less than 5.0 h<sup>1</sup> and in the presence of normal renal function, most is excreted in less than 12 h. Hence it may be justifiable that metformin be withdrawn only 24 h before surgery, specifically when general anaesthesia is required. This should limit the hazard of MALA without compromising glycaemic control. During the perioperative period, insulin is the conventional therapy for glycaemic control. After operation, when the patient has resumed full oral intake, metformin can be recommenced provided renal function has remained normal and there are no postoperative complications.

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- 1 Chan NN, Brain HPS Feher MD. Metformin-associated lactic acidosis: a rare or very rare clinical entity? *Diabet Med* 1999; **16**: 273–81
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- 5 Alberti KGMM. Diabetes and surgery. *Anesthesiology* 1991; **74**: 209–11

## Acupressure and prevention of nausea and vomiting

Editor—In their study on acupressure and prevention of nausea and vomiting, Harmon and colleagues<sup>1</sup> highlighted the problem of control treatments in acupuncture and acupressure studies. They suggested that the standard control

in acupuncture research is ‘sham’ acupuncture and then explain, quite rightly, why this should not be so. In their study they used sham acupressure. As they admit, they do not know the mechanism of action of acupressure. Could it not be possible that the sham acupressure is having an effect that might be promoting nausea in the control group? They do not mention the site of the sham acupressure. As they were applying the acupressure simultaneously with induction of anaesthesia and removing the bands before recovery, would it not have been better to have had a ‘no treatment’ control group?

I am also puzzled by the data in Table 4. Fifty-two patients were studied in each group but only 44 and 39 patients were scored for nausea in the two groups. Surely as the main aim of the study was to detect nausea and vomiting, all 52 patients should have been scored? If not, then the results are meaningless, if the authors’ power analysis is correct.

The final assertion that acupressure at the P6 point was effective in preventing nausea and vomiting after laparoscopy must be incorrect, as I do not think a 19% nausea and vomiting rate equates with prevention.

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- 1 Harmon D, Gardiner J, Harrison R, Kelly A. Acupressure and the prevention of nausea and vomiting after laparoscopy. *Br J Anaesth* 1999; **82**: 387–90

Editor—Thank you for the opportunity to reply to Dr Coe. The use of a control in acupuncture studies is probably the most debated aspect of acupuncture research. Despite Dr Coe’s assertions, we do consider ‘sham acupuncture’ to be appropriate. Sham acupuncture may have a specific effect,<sup>1</sup> particularly in analgesia research when point location is less important than in nausea and vomiting studies.<sup>2</sup> However, in a letter, Lewith and Vincent have described sham acupuncture as a valid control in nausea and vomiting studies.<sup>3</sup>

In our study, acupressure bands in the control group were placed on the dorsum of the right forearm. Alkaissi, Stalnert and Kalman<sup>4</sup> would disagree with Dr Coe’s assertion that sham acupressure could be responsible for an increased incidence of vomiting. In their study they found no difference in vomiting between the sham acupressure group and a no treatment group. Acupressure, as described in our methods section, was applied before induction of anaesthesia. A ‘no treatment’ group would have prevented blinding of the study.

As described in our methods section, if a patient had both nausea and vomiting, this was scored as vomiting. This method of scoring creates a ‘nausea only’ group. Comparing nausea between the groups of patients who vomited was not included in this study. Power analysis was

based on prevention of nausea and vomiting and not on either symptoms separately. All 52 patients in each group were included in the assessment.

Even the most efficient pharmacological prophylactic treatments only decrease the incidence of PONV by approximately 50%.<sup>5</sup> Our results are of a similar magnitude, with a noteworthy absence of side effects.

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- 1 Richardson PH, Vincent CA. Acupuncture for the treatment of pain: a review of evaluative research. *Pain* 1986; **24**: 15–40
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- 5 Watcha M. Nausea and vomiting: choice of drugs and treatment. *Curr Opin Anaesthesiol* 1996; **9**: 300–5

Editor—The article by Harmon and colleagues<sup>1</sup> on the use of acupressure for the prevention of postoperative nausea and vomiting was of particular interest to me. Some years ago, stimulated by the work of the late Professor John Dundee, I tried Sea-bands on myself for motion sickness with surprising success. I was implored by a gynaecological patient to try the bands when, on my preoperative visit, I mentioned them as we discussed her catalogue of unpleasant experiences with PONV. I applied the bands in the anaesthetic room to both wrists and on seeing her the next morning she was enjoying breakfast as never before after an operation. Having only one set of Sea-bands, I marked a cross on the P6 acupressure point on her wrists and suggested that she rubbed them whenever the nausea welled up. Later that day, she commented how effective this manoeuvre had been. Subsequently, I was often asked if my Sea-bands could be borrowed for a patient with PONV when drug treatment had failed, and commonly a good effect was achieved.

Having written to the manufacturers of my experiences, I was asked if I would conduct a study, Sea-bands vs neutral bands (with the stud reversed). However, I felt one could never be sure that the stud would not be rotated to the usual position by the patient at some point during the study. Thus it was interesting to note that the authors had used,

for control, the standard bands with pressure being applied at a non-acupoint site, and that application was used only during the anaesthetic itself, admittedly in short cases. My patients always had a poor history of PONV and kept the bands in position from immediately before induction until they felt confident to remove them after operation (up to 48 h later). As far as I know, no work has been done to ascertain if the non-dominant or dominant arm is preferable, so I always used the bands bilaterally.

While accepting that the enthusiasm of the anaesthetist in offering the bands might well have a placebo effect, the technique is non-invasive and there is nothing to lose in using this method in what can be a very difficult area of prevention or treatment. Finally, in describing the P6 acupressure point, I note the tendon of the *extensor carpi radialis* is mentioned; surely flexor was intended.

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- 1 Harmon D, Gardiner J, Harrison R, Kelly A. Acupressure and the prevention of nausea and vomiting after laparoscopy. *Br J Anaesth* 1999; **82**: 387–90

Editor—Thank you for the opportunity to reply to Dr Bowie. His previous experience with acupressure in the prevention of PONV is very interesting. We apologize for our error; the P6 point is between the tendons palmaris longus and flexor carpi radialis, not extensor carpi radialis as alluded to in the text. Professor Dundee, as Dr Bowie correctly points out, studied the efficacy of non-pharmacological techniques in the prevention of PONV. He examined if the use of the dominant (right or left) or the right hand had an influence on the efficacy of this technique. His conclusion was that there was no clinically significant difference and it is appropriate to use the right hand in all patients.<sup>1</sup> To our knowledge, no study has examined the specific question of the use of the dominant vs the non-dominant hand in acupuncture antiemesis.

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- 1 Fitzpatrick KTJ, Dundee JW, Ghaly RG, Patterson CC. Is it necessary to always use the right forearm for acupuncture antiemesis. *Br J Anaesth* 1988; **61**: 117–18P

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## Book reviews

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*Problems of Anesthesia. Economic Issues in Current Anesthesia Practice, Vol. 10, No. 3.* F. Arens and D. S. Prough. Published by Lippincott, Williams and Wilkins, Philadelphia. Pp. 113; indexed.

A quotation within the text, 'we in anesthesia, as a profession, will be judged by medical students and other physicians by the way we treat our graduates', underlines how anaesthetists must consider the issues of manpower, quality of care, society's health needs and the overriding effects of limited finance. Failure to do so will result in the over supply of anaesthetists in the job market and anaesthesia will be less attractive to new graduates. This risks a future loss of quality of anaesthetists and the practice of anaesthesia. As the title suggests, this book considers these issues from an economic standpoint. The nine chapters are written by American doctors, and therefore certain sections are not apposite to British anaesthetic practice but substantial parts of the book are immediately applicable or may illustrate a possible future.

The initial two chapters of the book discuss the broad economic issues and cost effectiveness in the fields of pharmaco-economics and technology. The arguments for and against drug budget savings are well considered from a historical background through to the actual experience at Duke University Medical Center. An example shows how small savings per patient can result in a major effect on the profitability of an organization and that it can be attained and maintained through the use of education and practice guidelines. Assessment of cost effectiveness in the USA is prominent because of the change in reimbursement for anaesthetic practice from a pay-per-item to a prospective payment system which is based on diagnostic related groups. The result is that the use of expensive technology no longer increases income, but risks expensive overheads for a predetermined fee. Measurement of outcome, economic analysis and decision analysis needed to manage anaesthetic departments in the new systems are considered logically in the second chapter. Clinical directors under budgetary pressures would do well to start here in the development of strategies to meet fiscal targets.

The next two chapters discuss the process of contracting, capitation, reimbursement and the impact of managed care on American anaesthesia. It is complex and difficult reading, particularly as the process is alien to the UK system. However, the introduction of primary care groups and new government initiatives will radically change the contracting process for healthcare in the near future, thus increasing the relevance of this section. The chapters outline the difficulties in defining the financial risk implicit in accepting the treatment of large, heterogeneous patient populations. The need to develop better information technology to identify the population risks is clearly made; any clinical director who has agreed to a waiting

list initiative only to discover that patients are far sicker than expected may have avoided the mistake having read this section.

The next three chapters consider the American anaesthetic workforce from the perspective of healthcare team structure and the mechanisms of manpower planning and how these two factors influence the size and number of anaesthetists in training. The current discussion in the UK over the role of non-medical anaesthetists, the need to maintain a high quality of care and concerns over unemployment in anaesthesia makes the content of these three chapters appropriate and thought provoking. The introduction of Calman training has created a conveyor belt system producing fully certified doctors who now need locum consultant posts as temporary positions before obtaining a permanent appointment. The parallels with the over production of graduates in the USA recounted in the third chapter gives pause for thought regarding training within the UK.

The penultimate chapter deals with the medico-legal management and risk adjustment systems in place in one hospital in the USA. The introduction of clinical governance in the UK is proving a difficult process. This chapter concisely discusses the experience of a department dealing with this issue and offers a useful read for those concerned with implementing clinical governance. The last chapter is presumably in that position because many hours have been wasted by many hospitals in failing to resolve the longstanding issue of theatre efficiency. This chapter considers the complex man/management issues that bedevil the issue of theatre efficiency and on reading it, it may avoid people reinventing the wheel in their efforts to improve patient throughput. The author details the fundamental problems that need to be identified and solved but also admits to their failure to deal with the most important one, that of dysfunctional consultant behaviour.

The book is well written with only a small degree of overlap between chapters. It is informative and challenges the reader to re-examine the working practice of their anaesthetic department and hospital. It is, however, a read for the interested rather than the passing browser.

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*Problems in Anesthesia — Ambulatory Anesthesia, Vol. 11, No. 1.* B. K. Philip and B. S. Epstein (eds). Published by Lippincott, Williams and Wilkins, Philadelphia. Pp. 155; indexed; illustrated.

This short book consists of several interesting reviews on various aspects of day-case anaesthesia. The authors are entirely from North America and the various chapters reflect practice in the USA. A theme which runs through the book relates to the influence which insurers and managed care are having on the delivery of ambulatory care in the USA.

Despite the bias towards American practice, there is much of interest for anaesthetists in the UK, and with the continuing increase in day-case surgery there can be very few anaesthetists who have no involvement with such cases. Individual chapters are well referenced and generally easy to read. They cover the whole spectrum of problems relating to anaesthesia for ambulatory or day-case surgery. The short summaries of the individual chapters in the contents section at the beginning of the book is a useful feature of the *Problems in Anaesthesia* series. Tables listing various requirements and scoring systems for management in ambulatory anaesthesia are helpful in stressing important points but at times there is wasteful repetition of the same information in the text.

The chapters on preoperative screening and unacceptable patients for ambulatory anaesthesia provide useful, up-to-date reviews of this important area. The difficult problem of defining the limits of procedures that can be performed as day-cases is also well reviewed and indicates how far these limits are being pushed in the USA in the drive to reduce costs. The role of regional anaesthesia for adults undergoing outpatient anaesthesia is reviewed in detail as are the logistics and techniques of monitored anaesthesia care. The chapters on paediatrics, post-anaesthesia care unit assessment and discharge, outcomes and quality assurance, and anaesthesia in remote locations are good reviews which give common sense practical advice and make interesting reading.

The final chapter on office-based anaesthesia demonstrates a very worrying development. This is clearly driven by the pressure to reduce the cost of medical treatment in the USA, but at what cost? For decades, anaesthetists in the UK have strived to centralize general anaesthesia to areas which are well staffed and equipped. Only recently the General Dental Council and the Royal College of Anaesthetists issued regulations for dental anaesthesia which limit such work to premises meeting a minimum standard for staffing and equipment. This effectively limits the work to a small number of practice premises. A return to a situation where general anaesthesia is performed on isolated, poorly equipped, poorly staffed sites compromises safety and is a retrograde step for anaesthesia.

Overall, the book is very readable and gives a good update on the problems associated with day-case anaesthesia. It is not appropriate for trainees preparing for the FRCA examinations who would be better served by reading appropriate chapters in standard textbooks. However, I would recommend it to career grade anaesthetists who anaesthetize day-cases and it would be a useful addition to the shelves of postgraduate medical libraries.

*E. Moss*

*Pain and Suffering.* W. K. Livingston. Published by IASP Press, Seattle. Pp. 250.

This is an unusual book and is published for the benefit of those interested in the broader aspects of pain.

As a new publication it is unusual because the author, William K. Livingston, died in 1966. He began the book 10 yr earlier and worked at it throughout his retirement but never completed it. After his death, the manuscript was held successively by his son, the Oregon Health Sciences University Library and latterly in the History of Pain Collection at the Louise M. Darling Biomedical Library at UCLA. John Liebeskind, together with Marcia Meldrum, realized at least the historical value of the text and persuaded Howard L. Fields to edit it and IASA to publish it in its revised form.

To believe this book is only of historical value would be wrong, although it provides a fascinating insight of the beginnings of pain management as a specialty in the USA. Certainly the clinical descriptions of patients with various neuropathic syndromes are as dramatic as they are instructive. An injured soldier who was found sitting with his foot in a bucket of water to control the neuropathic 'fire' of major causalgia was 'cured' by a chemical lumbar sympathectomy. He describes the 'physiological' effects of missile injury and in particular the effects of nerve damage so produced, and the problems of treating phantom pain.

Dr Livingston's contributions to an understanding of the perception of pain were to relate the carefully observed clinical picture with neural mechanisms. Yet he fully recognized the importance of psychology in pain perception.

In the last chapter he proposed pain to be a 'perceptual process'.

1. Pain is a perception and as such is subject to the influence of associated ideas, apperceptions, and fears.

2. The impulses that subserve it are not pain but are merely a part of its underlying and alterable mechanisms.

3. The impulses may be initiated by a wide variety of stimuli.

4. When they enter the spinal cord they are subject to modification by the internuncial pool of central neurones, whose activity is determined from moment to moment by other sensory impulses and by influences from other parts of the CNS.

5. In their ascent to higher centres the impulses are subject to further modification . . . etc.'

His view of the integration, and especially the modification, of pain pathway activity has a very modern ring to it. '4' above is surely the gate control theory, and Ronald Melzack accepts that Livingston was for him an important mentor. The modification concepts suggest that the current paradigm of neural plasticity was being considered theoretically 40 yr ago.

This book is a valuable contribution to our heritage but significantly adds to our knowledge of some of today's most difficult clinical problems. It is written in an easy style and for me had the 'unable to put down' characteristic of a good thriller. I can strongly recommend it to all who have an interest in chronic pain.

*F. R. Ellis*