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 β 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 β 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 β 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 β and VADST performance (\( r = -0.43 \)).

We concluded that because of its specificity, known kinetics and good correlation with neuropsychiatric tests, S-100 β 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.1 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 β 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 \( S_j O_2 \) data suggesting cerebral ischaemia during rewarming.2 We agree that the significance of intraoperative increases in neuroproteins needs further investigation.


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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-yr-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\(_2\)O, returning to a baseline of 17 cm H\(_2\)O, despite having no positive end-expiratory pressure (PEEP)
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.\(^1\)-\(^3\)

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.\(^4\) 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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**Analgesic efficacy of paracetamol and diclofenac in children receiving PCA morphine**

Editor—Regarding the article by Morton and O’Brien,\(^1\) 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,\(^2\) 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,\(^3\) 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).