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- No letter should be more than 400 words.
- For letters on scientific subjects we normally reserve our correspondence columns for those relating to issues discussed recently (within six weeks) in the *BMJ*.
- We do not routinely acknowledge letters. Please send a stamped addressed envelope if you would like an acknowledgment.
- Because we receive many more letters than we can publish we may shorten those we do print, particularly when we receive several on the same subject.

## Health education in a time of adversity

SIR,—As the author of the report on food and low income which the Health Education Authority refused to publish recently I read Dr Spencer Haggard's letter with great interest.<sup>1</sup> He claims that the review was not published because "it exceeded its brief and needed more editing than we had time to undertake." This is nonsense and it does the Health Education Authority no credit to gloss over its failings by publishing misleading information.

I was asked to write the review after submitting a very detailed proposal to the authority. This was considered, along with four or five other submissions, and I was offered the work. At the time I was complimented on the detail of the proposal by the commissioning officers, who knew exactly what level of detail to expect. The report was written exactly in line with the proposal.

I understand that the review was then refereed by both the Department of Health and an academic nutritionist, neither of whom appeared to be happy with the conclusions. I offered to look at their comments and consider making justifiable alterations but was told that there was not time before the publication of the Health Education Authority's own research, for which my report was to have been an introduction. That was in August. It is now mid November and nothing has been published.

This is a serious issue and the Health Education Authority should not be allowed to fob it off in such a light hearted manner. There is no doubt that the independence of the authority has been undermined since its reconstruction as a special health authority under the direct control of the Department of Health. If it is to have any useful role in promoting good health and preventing ill health it must work much harder to refine its independence and be prepared to discuss and publish what, to some, may be controversial evidence. If it cannot do this but has to continue acting as a government propaganda machine I for one, as a taxpayer, am very unhappy about supporting it.

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<sup>1</sup> Haggard S. Health Education Authority in a time of adversity. *Br Med J* 1989;299:1222. (11 November.)

SIR,—Dr Spencer Haggard states that the "Learning About AIDS" pack is widely available.<sup>1</sup> It was, however, published by a commercial publisher rather than by the Health Education Authority (HEA).

The development of "Learning About AIDS" was financed by the charity the Aids Education and Research Trust (AVERT), and in June 1988 the

HEA agreed to publish it. This agreement was then withdrawn after AVERT refused to allow the Department of Health to "review" the pack. An expert advisory group, which included representatives from the HEA, had supervised the development of the pack and had approved the final text. It was unacceptable to AVERT for the text to be subsequently altered.

Even if the Department of Health review had not resulted in any alterations it would have caused a considerable delay. In any case such a review was unacceptable as a matter of principle to AVERT, an independent charity that had entered into a collaborative venture with an independent authority. AVERT finally and reluctantly agreed to publication by a commercial publisher as this was the only way the pack could be made available in its original form.

In explaining further about the review process Dr Haggard has since stated that "the Department of Health has made known its requirement to have the opportunity to comment on AIDS materials produced by this authority prior to publication." Where does this leave the concept of an independent HEA?

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<sup>1</sup> Haggard S. Health Education Authority in a time of adversity. *Br Med J* 1989;299:1222. (11 November.)

## Brain stem death and organ donation

SIR,—Two recent papers provide important data about the factors that influence the yield of organ donors from intensive care units.<sup>1,2</sup>

There are several reasons why there are fewer organs available for transplantation than might be expected. The report from Cambridge shows that the coroner withheld consent in eight of 52 suitable cases,<sup>1</sup> which happened in the Glasgow neurosurgical unit in only one of 42 cases when the

relatives had already consented. That 23% of organs offered by relatives in Cambridge were not actually transplanted is noteworthy, given the proximity to kidney, heart, and liver transplant teams. It is ironic that lack of postoperative intensive care facilities was the reason why some organs could not be used, because unwillingness to commit intensive care resources in donor units is sometimes cited as a reason for reluctance to identify donors.

A review of 403 deaths in this neurosurgical unit over three years shows many similarities with the careful audit of English intensive care units (table). It is not surprising that more deaths in a neurosurgical unit occur in patients undergoing ventilation (31% v 14%). The Cambridge figures are difficult to compare because cases of circulatory arrest are included, and in some consent was obtained only for corneal donation. In both the recent Glasgow and Cambridge reviews head injuries accounted for half the cases of brain stem death, the same proportion as in our review of the 1228 United Kingdom donors in 1977-80.<sup>1</sup> That survey showed that 39% of donors came from the 14% of hospitals that had a neurosurgical unit; in 1987, 46% of donors came from the 12% of hospitals with a neurosurgical unit, according to the United Kingdom Transplant Service figures for 1988.

The recent audit of English intensive care units certainly provides a more accurate estimate of the frequency of brain death than we were able to make in 1980. It would be interesting to know the diagnostic and hospital mix for these data. If half the cases were head injuries, as in several other studies, the estimate of 1700 possible cases of brain death in England a year would include 850 head injuries. In 1980 there were 90 head injury deaths per million population in England, 55% of them in hospital, making 2475 per year.<sup>1</sup> We reported that half the deaths from head injury in the Glasgow and Cambridge neurosurgical units in 1980 occurred in patients undergoing ventilation.<sup>1</sup> If head injuries elsewhere are similarly treated, this would mean 1240 patients with head injuries a year dying on a ventilator in England, rather than 850. But

*Comparative data from Glasgow neurosurgical unit, English intensive care units, and Cambridge intensive care unit. Results are percentages*

	Glasgow neurosurgical unit	English intensive care units <sup>1</sup>	Cambridge intensive care unit <sup>1</sup>
No of deaths	403	2853	—
Brain stem death possible	31	14	—
Brain stem death tests:			
Of all deaths	22	11	—
Of deaths on ventilator	70	69	—
Medically unsuitable (among those tested)	23	17	25 (10/42)
Consent requested	84	94	96
Refused	26	30	10
Donors:			
Among tested	47	50	38
Among suitable	60	61	55

perhaps fewer are ventilated in other hospitals. As computed tomographic scanners become available in more general hospitals, fewer hopeless cases of head injury (and cases of intracranial haemorrhage) are likely to be referred to neurosurgeons. That could mean more potential donors in other hospitals. International comparisons of brain stem death and organ donation should allow for the fact that the incidence of fatal head injuries is two to three times greater in the United States, Canada, Australia, France, and Germany than in Britain and that the number of intensive care beds per head is 10 times greater in the United States. We hope that the further planned audits of deaths in intensive care units in England may throw more light on the factors that influence the yield of organ donors.

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- 1 Bodenham A, Berridge JC, Park GR. Brain stem death and organ donation. *Br Med J* 1989;299:1009-10. (21 October.)
- 2 Gore SM, Hinds CJ, Rutherford AJ. Organ donation from intensive care units in England. *Br Med J* 1989;299:1193-7. (11 November.)
- 3 Jennett B, Hesse C. Brain death in Britain as reflected in renal donors. *Br Med J* 1981;283:359-68.
- 4 Jennett B, MacMillan R. Epidemiology of head injury. *Br Med J* 1981;282:101-4.
- 5 Jennett B, Gleave J, Wilson P. Brain death in three neurosurgical units. *Br Med J* 1981;282:533-9.

SIR,—Dr A Bodenham and his colleagues undertook a prospective audit of potential organ donors in Addenbrooke's Hospital, Cambridge, and found only three cases where consent to a donation was not sought.<sup>1</sup> They concluded that required request would not increase the supply of donor organs.

Several points make this conclusion invalid. The survey was conducted in a large teaching hospital heavily committed to liver, kidney, and pancreatic transplantation and therefore more aware than most of the needs of transplantation. The survey was not carried out by a group that was independent of the donation process, and the fact that it was prospective and known to be in progress could well have influenced decisions. No attempt was made to see whether the number of brain stem dead individuals identified within the hospital was complete. This could have been done by examining the case notes of all patients dying within the hospital.

A point of concern is that a pair of kidneys was not removed because of lack of a suitable recipient. Cambridge is a signatory to the national sharing agreement, and if there were no suitable local recipients there would have been plenty nationally.

We believe that potential organ donors are frequently overlooked, particularly in smaller, non-teaching hospitals where intensive care facilities may be limited. Required request could well have an impact in such hospitals, and it is unfortunate that the authors should attack the concept using questionable data obtained within one specialised institution.

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- 1 Bodenham A, Berridge JC, Park GR. Brain stem death and organ donation. *Br Med J* 1989;299:1009-10. (21 October.)

## Organ donation from intensive care units in England

SIR,—We wish to point out that the first sentence in the conclusion of our abstract can be misinterpreted if read without the qualifications given

in the discussion.<sup>1</sup> This sentence could be read as implying that we recommend formal testing of brain stem function whenever brain stem death is a possible diagnosis. We do not. Although the results of the audit showed that tests for brain stem death were not performed in 26% of those in whom this was a possible diagnosis, we suspect that in many of these cases formal testing of brain stem function was, for various reasons, clearly inappropriate. Accordingly, a supplementary inquiry has been initiated to determine the reasons why tests for brain stem death were not performed in these cases and to estimate the proportion of these patients who could have been potential donors.

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- 1 Gore SM, Hinds CJ, Rutherford AJ. Organ donation from intensive care units in England. *Br Med J* 1989;299:1193-7. (11 November.)

## Acute renal failure after infusion of gelatin

SIR,—We wonder whether the article by Drs S F Hussain and P J T Drew could be more simply entitled "Acute renal failure after aortobifemoral graft surgery."<sup>1</sup>

The data provided are incomplete and do not confirm their contention that Gelofusine was a causative agent. The only circumstantial evidence is one measurement of plasma oncotic pressure, which could relate to accumulation of any fluid at a time when renal failure was more or less established.

The report as presented suggests that the only fluids given over the 48 hour perioperative period were blood and Gelofusine to a total of 3.5 litres, together with 330 ml of mannitol. Over the same period the urine output was 2.2 litres, thus leaving 1.3 litres to cover all losses, including significant "3rd space" losses into the areas of surgical dissection. The fall in serum albumin concentration would seem to indicate that the losses into this "3rd space" were considerable. It would seem probable, therefore, that the use of mannitol and frusemide together with a diminished intravascular volume, which this treatment could have worsened, were the major contributory factors to the acute renal failure.

Whatever the cause in this case it is clear that preventing renal failure depends on adequate organ perfusion, and this is best achieved by having an appropriate circulating volume and hence cardiac output. A fall in urine volume should not be treated by a diuretic in patients who are likely to have a reduction in circulating volume after major vascular surgery. Uncertainty over the state of the intravascular compartment needs measurement of the appropriate vascular pressure and trials of fluid. In this context it has been shown that central venous pressure can be unreliable in predicting change in blood volume<sup>2</sup> and that it is better to use pulmonary capillary wedge pressure and urine output as guides to fluid replacement.<sup>3</sup>

We therefore cannot support the contention that Gelofusine caused renal failure in this patient.

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- 1 Hussain SF, Drew PJT. Acute renal failure after infusion of gelatins. *Br Med J* 1989;299:1137-8. (4 November.)

2 Baek SM, Makabali GG, Brown CWB, Kusek JM, Shoemaker WC. Plasma expansion in surgical patients with high central venous pressure (CVP): the relationship of blood volume to haematocrit, CVP, pulmonary wedge pressure, and cardio-respiratory changes. *Surgery* 1975;78:304-15.

3 Clark NJ, Stanley TH. Anaesthesia for vascular surgery. In: Miller RD, ed. *Anaesthesia*. 2nd ed. Edinburgh: Churchill Livingstone, 1986.

SIR,—The implication in this paper that the Gelofusine caused the renal failure in this patient is highly debatable.<sup>1</sup>

The most well documented cause of renal failure after aortic vascular surgery is prerenal in origin. The pronounced haemodynamic changes that occur—particularly with aortic cross clamping and unclamping—cause changes in cardiac output and hence renal blood flow. For instance, infrarenal cross clamping causes redistribution of blood flow and hence cortical ischaemia.<sup>2</sup> Moreover, central venous pressure monitoring is an unreliable indicator of left ventricular end diastolic pressure. Pulmonary capillary wedge pressure is a more accurate indicator for volume expansion,<sup>3</sup> particularly in the face of coronary artery disease, which frequently coexists with aortic atherosclerotic diseases. It is also conflicting to say on the one hand that blood loss was minimal and on the other that 3.5 litres of colloid were required. Finally, the problems of prerenal failure may be compounded by direct nephrotoxins—antibiotics, radiocontrast media, haemoglobinuria (from blood transfusion reactions)—in the pathogenesis of acute tubular necrosis.

No mention is made of the postoperative monitoring of the patient, and it is at this time when hypovolaemia may occur, particularly during re-warming.

The implication that Gelofusine has accumulated is at odds with the known pharmacology of Gelofusine. Gelofusine has a mean molecular weight of 35 000 and even in anephric patients it would redistribute to the entire extravascular space by 48 hours (when plasma oncotic pressure was measured). In any case as it is renally excreted and the urine output was satisfactory for the first two days after operation most of the Gelofusine would have been excreted.

In my department Gelofusine is used precisely for the circumstances in which we are now advised to avoid it—that is, low perfusion pressure and low rates of urine flow—and when titrated accurately against the patient's needs it is most successful at restoring cardiac output and urine flow.

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- 1 Hussain SF, Drew PJT. Acute renal failure after infusion of gelatins. *Br Med J* 1989;299:1137-8. (4 November.)
- 2 Abbott WM, Austen WG. The reversal of renal cortical ischaemia during aortic occlusion by mannitol. *J Surg Res* 1974;16:482-9.
- 3 Bush HL, Huse JB, Johnson WC, O'Hara ET, Nasbeth DC. Prevention of renal insufficiency after abdominal aortic aneurysm by optimal volume loading. *Arch Surg* 1981;116:1517-23.

SIR,—As medical adviser to the manufacturers of Gelofusine, Hausmann Laboratories of St Gallen, Switzerland, I was surprised and dismayed to read a case report suggesting that Gelofusine was potentially nephrotoxic.<sup>1</sup> My surprise was based on the apparent breach of the longstanding practice whereby, before publication of such reports, the manufacturer's comments are invited by the author.

Such a policy of consultation is of value, for, while it is essential that clinically important reactions are speedily drawn to the attention of potential prescribers, it is also essential that the report should discuss other information available so that the risk:benefit equation can be meaningfully reassessed.