service. We would not have the tragedy of 1200 haemophiliacs infected with the human immunodeficiency virus, almost all of whom caught the virus from imported factor VIII.

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SIR,—At the risk of appearing immodest, I would like to respond to Dr John Cash's claim that several distinguished directors of haemophilia centres support an alleged lack of government commitment to self sufficiency in blood and blood products (12 September, p 617). As Dr Cash should know, delegations including directors of blood transfusion and haemophilia centres met representatives of the Department of Health and Social Security on many occasions to press the need for such self sufficiency until argument and pragmatism resulted in the development of the new Blood Products Laboratory at Elstree.

The concentrates of factor VIII and IX now produced at Elstree compare more than favourably with those produced abroad in the public or commercial sectors. They are secure from contamination with the human immunodeficiency virus and are currently undergoing clinical assessment with regard to freedom from transmission of serum hepatitis and other virus diseases. Use of these concentrates in England and Wales is restricted almost entirely by the lack of supply, which, hopefully, will be corrected when the manufacturing facility is in full use. At the same time it must be recognised that technology is moving on. The development of sophisticated purification methods such as immunoaffinity separation on monoclonal antibodies of both plasma derived and recombinant factor VIII heralds the appearance of new generations of coagulation factor concentrates. If these prove to be safer and more effective than those produced by the United Kingdom fractionators and are available and licensed then individual doctors may be under pressure to prescribe them in the best interests of their patients. Hopefully, therefore, the government will recognise the need for substantial investment in research and development at our plasma fractionation facilities. It would be counterproductive to fall behind in technological developments.

The operational reservation of some directors of haemophilia centres sensed by Dr Cash may reflect a fear about government commitment to financing adequate research and does not imply that they are unsupportive of national self sufficiency. Directors of haemophilia centres in the United Kingdom with whom I have discussed the subject fully support and wish to promote efficient national self sufficiency in blood and blood products.

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SIR,—We are currently reviewing all elective admissions to our unit for 1986, looking at our blood crossmatching and transfusion practices. A review of roughly half of our data, albeit from the narrow confines of a paediatric orthopaedic practice, seems to support the comment made by Dr John Cash (12 September, p 617) that "the conduct of affairs at the clinical interface of blood transfusion throughout the UK leaves much to be desired."

Our preliminary data show that of 210 children admitted 47 (22%) had their blood crossmatched for an average of 1.8 units each but only 8 (4%) were actually transfused, with an average of 1.4

units. We do not regard a cross match to transfusion ratio of 7.6:1 as making good use of a scarce resource. It also shows that 26% of the blood crossmatched was for removal of metalwork and 17% for soft tissue surgery of the legs but none whatsoever was used. On the other hand, 20% of blood crossmatched was for femoral osteotomies, of which 24% was used, and 13% for scoliosis surgery, of which 55% was used. We hope that a full analysis of our data will provide us with better guidelines for crossmatching and help to identify which procedures really need to have blood available. A safe and more efficient use of the blood transfusion service should ensue.

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Does nifedipine ameliorate cyclosporin A nephrotoxicity?

SIR,—The interesting report by Dr J Feehally and colleagues (1 August, p 310) has prompted us to conduct a similar survey among our renal transplant patients. Our findings, however, are different from those reported by Dr Feehally and coworkers.

We surveyed 104 patients, of whom 61 were treated with cyclosporin and low dose prednisolone (10 mg/day) and 43 with azathioprine and prednisolone (10 mg/day). At the time of the study all the patients had had transplants for at least three months and all had stable graft function, as determined by clinical and laboratory assessments. Hypertension was found in only 75% of the patients treated with cyclosporin. This was lower than the figure of 85% quoted by Dr Feehally and colleagues. In contrast, a higher percentage of our patients treated with azathioprine had hypertension (67% v 54%).

Though our population of patients was smaller than that of Dr Feehally and coworkers, the graft durations and age range were similar. The patients receiving cyclosporin were divided into three groups: normotensive patients, hypertensive patients not receiving nifedipine, and hypertensive patients receiving nifedipine (table). The mean doses of cyclosporin in the three groups were 4.4, 5.0, and 4.7 mg/kg/day respectively, and the whole blood cyclosporin concentrations were 422, 425, and 402 ng/ml respectively. No significant difference was found, with Student's *t* test, in the mean serum creatinine concentration in the group treated with nifedipine compared with the normotensive group or the group who did not receive nifedipine, showing that nifedipine did not seem to ameliorate cyclosporin nephrotoxicity. One main difference in our patients was the lower dose of cyclosporin (mean 4.7 mg/kg/day) required to maintain adequate immunosuppression.

One possible explanation for the findings reported by Dr Feehally and colleagues is that cyclosporin nephrotoxicity is cumulative and

related to time and that the duration of grafts in the group of hypertensive patients treated with nifedipine was significantly shorter than that in the normotensive patients or the hypertensive patients who did not receive nifedipine. We conclude that more prospective control studies are needed before nifedipine may conclusively be shown to ameliorate cyclosporin A nephrotoxicity.

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Type A behaviour and ischaemic heart disease

SIR,—Dr J E J Gallacher (22 August, p 501) asks whether there is evidence that type A behaviour (as measured by the Bortner score) differs between towns in the British regional heart study and what effect any such differences might have on our conclusion that type A behaviour is unrelated to the risk of coronary heart disease in British men.

Differences in Bortner scores between towns (table) are in the expected direction—that is, towns with a high percentage of manual workers (such as Grimsby) have the lowest mean Bortner scores.

Mean Bortner scores by town, unadjusted and adjusted for social class and age and ordered by ascending mean scores

Town	Mean (SEM)	% Manual workers	Adjusted mean
Grimsby	88.96 (1.15)	81	91.19
Falkirk	91.00 (1.15)	74	92.14
Burnley	91.39 (1.17)	69	92.57
Merthyr Tydfil	91.82 (1.23)	72	93.20
Scunthorpe	92.03 (1.08)	77	94.10
Gloucester	92.51 (1.17)	70	93.44
Hartlepool	92.64 (1.12)	73	93.93
Newcastle under Lyme	92.75 (1.15)	68	93.27
Carlisle	92.85 (1.02)	58	92.98
Ayr	93.68 (1.14)	63	93.64
Dunfermline	93.81 (1.07)	61	94.06
Exeter	94.20 (1.09)	45	92.94
Dewsbury	95.35 (1.11)	64	95.45
Wigan	95.47 (1.07)	65	96.44
Ipswich	95.89 (1.06)	42	94.25
Bedford	95.96 (1.15)	46	94.70
Maidstone	96.16 (1.14)	55	95.41
Darlington	98.47 (1.01)	41	97.01
Guildford	99.61 (1.08)	23	95.90

The differences between towns are smaller than the differences between the social classes (see table I in our paper, 11 July, p 86), and when the town means are adjusted for differences in social class and age structure differences between towns decrease considerably.

Therefore it is not surprising that in calculating the crude attack rates by quintiles of the Bortner score allowing for differences between towns has

Characteristics of the patients treated with cyclosporin A. Values are means (SEM)

	Age (years)	Graft duration (months)	Serum creatinine concentration (μmol/l)	Systolic blood pressure (mm Hg)	Diastolic blood pressure (mm Hg)
Normotensive (n=15)	51.3 (3.9)	25.1 (2.5)	135.0* (8.4)	145.6 (4.6)	81.6 (1.6)
Hypertensive, not receiving nifedipine (n=13)	43.6 (4.5)	25.3 (3.2)	153.8† (19.0)	144.0 (3.9)	82.4 (1.9)
Hypertensive, receiving nifedipine (n=33)	44.5 (2.8)	22.8 (20.0)	162.0 (11.0)	143.2 (2.9)	82.4 (1.1)

*Not significant compared with other groups.

†Not significant compared with other groups.