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 (μ mol/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.

little effect, and our conclusion remains unaltered. Indeed, the attack rates, adjusted for town as well as age and social class, are identical to one decimal place with those quoted in table IV of our paper.

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Drug abuse: a new problem

SIR,—In their paper on accidental needlestick injuries to children from discarded syringes (29 August, p 526) Dr S S Walsh and colleagues state that "most of these [incidents] occurred after January 1987." Are we to take it that they are presenting evidence of an increased rate of such accidental needlestick injuries among the general public? Is there any evidence from the recovered needles and syringes or from their location that this increased incidence is related to the new injecting equipment exchange scheme established in central Liverpool at the end of 1986, or has any increase in incidents been more gradual, indicating that it is related to a more general and gradual increase in intravenous drug abuse itself?

The answers to these questions are important to the present debate about such exchange schemes, and Dr Walsh and coworkers should be encouraged to provide any subsequent figures that may have become available.

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AUTHORS' REPLY.—Dr Strang raises an important question, which we have also considered. Though most cases of needlestick injury from June 1985 to the end of April 1987 occurred after 1 January 1987 (eight incidents out of 13), we noticed no clustering of cases in dates of presentation or location of discarded needles and syringes, either before or after the introduction of the injecting equipment exchange scheme in Liverpool on 24 October 1986. Exchange needles and syringes are available from some pharmacies in the city as well as from the centre, and they all issue various standard types. Therefore, it was not possible to trace our recovered needles and syringes to any particular source.

The Royal Liverpool Children's Hospital has two branches—an inner city based hospital (Myrtle Street) and a suburban based hospital (Adler Hey). Of the eight patients with needlestick injury, five presented to the former and three to the latter. The children came from widely differing areas of the city and presented at a steady rate of one to three a month from January to April. Interestingly, since writing our report at the end of May we have seen five more patients, who presented at the same rate, emphasising that the problem continues.

Our latest information from colleagues at the exchange centre in Liverpool is that they have a positive balance of the order of 1000-1500 needles and syringes.

From all this information we concluded that the exchange scheme did not play an important part in the increasing incidence of needlestick injuries seen at the hospital. Probably of greater importance was the increased public awareness of the acquired immune deficiency syndrome as a result of media coverage and the government's advertising campaign. In our experience this was the main factor

that had prompted parents to seek medical advice when confronted with the problem.

We, like Dr Strang, obviously support any moves that may help in eliminating this problem.

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Controlled trial of γ linolenic acid in Dukes's C colorectal cancer

SIR,—The controlled trial of γ linolenic acid in Dukes's C colorectal cancer by Drs M B McIlmurray and W Turkie (16 May, p 1260) failed to show any benefit.

Their patients received 270 mg γ linolenic acid daily, which is insufficient for any therapeutic effect of this fatty acid derivative, which has been proved to be cytostatic in vitro. In our study, the abstract of which was quoted by Drs McIlmurray and Turkie, we used up to 36 capsules daily, providing 1.62 g of γ linolenic acid. We have patients with untreatable brain tumours surviving for 39 months and others with various tumours surviving for periods of up to three years.

Because evening primrose oil contains only 9% γ linolenic acid the volume of oil needed to provide adequate daily amounts of the acid becomes prohibitive. Work is currently under way, however, to produce concentrates of γ linolenic acid to enable adequate doses of this metabolite to be given to patients with a high tumour load.

Interestingly, the survival curves in the paper by Drs McIlmurray and Turkie show that γ linolenic acid was ahead of placebo for most of the study. In any further study the use of at least 1.6 g of acid daily should be attempted.

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AUTHOR'S REPLY.—The dose of γ linolenic acid chosen in our study was empirical as it is difficult to translate in vitro data into clinical practice. We thought that it was unreasonable to ask patients to take more than six capsules daily over a period of years. Thus the dose was considerably less than that quoted by Dr Van der Merwe (in an abstract published after our study was started), but the clinical circumstances were entirely different. We were treating patients with no evidence of residual tumour, and the tumour load was small.

The evaluation of higher concentrations of γ linolenic acid in patients with cancer may well be worth while, but we would urge that this be done only in controlled clinical trials.

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Hypothermia in the elderly: scope for prevention

SIR,—Drs Catherine J Otty and M O Roland (15 August, p 419) do well to highlight the complexities of preventing hypothermia. For practical purposes strategies need to be considered for three categories of patient.

Those presenting with hypothermia without underlying illness are fortunately rare, with few doctors working in the community reporting appreciable numbers of cases even in a severe winter.¹ Most of these cases occur after a fall. An

effective strategy here is the increased use of dispersed alarm systems in the community to reduce the time patients lie immobile and exposed.²

It is difficult to conceive of effective preventive strategies for the second and far more common group with hypothermia, those in whom the condition is secondary to underlying illness. A warm environment is no guarantee against this form of hypothermia. It is not uncommon to record low body temperatures in patients developing bronchopneumonia in overheated hospital wards.

Patients in the third category are those who first develop hypothermia, which in turn results in increased rates of stroke and myocardial infarction owing to transient increases in blood pressure and plasma viscosity.³ Unless studied epidemiologically, hypothermia often goes unrecognised as the predisposing event because the patients may have recovered a normal body temperature by the time the vascular episode presents several days later.⁴

There is real potential for preventing this last sequence by ensuring that the elderly live in a warm environment. However, the provision of heating in homes is by itself not necessarily an effective strategy. The Anchor Housing Association found that the provision of unlimited free heating in sheltered housing failed to prevent the increased mortality in occupants over the cold winter months.⁵ This failure was attributed to the elderly turning their heating off, opening windows at night, or going for walks in the cold outdoors.

An attitudinal and behavioural change is also required. At present the authors' findings, mirrored by similar studies in fall prevention by McCabe,⁶ suggest that limitations may be imposed on health maintenance strategies by the refusal of the elderly to change lifelong habits and adopt a more healthy lifestyle.

The final irony in the complexities of preventing hypothermia lies with the theoretical possibility that the more we encourage the elderly to live in warm environments, the less able they may become to acclimatise to cold conditions when encountered. Although mild cold exposure has not so far been shown to bring about an adaptive response in elderly humans, German, studying aged mice, found that intermittent cold exposure did effectively reduce the incidence of hypothermia during cold challenge.⁷

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Pneumothorax in the supine patient

SIR,—We thank Drs Andrew R C Cummin, Michael J Smith, and Alan G Wilson (5 September, p 591) for their guidelines to help clinicians to recognise a pneumothorax in a supine chest radiograph. We believe, however, that there is a second lesson to be learnt from case 1.