CORRESPONDENCE

We may return unduly long letters to the author for shortening so that we can offer readers as wide a selection as possible. We receive so many letters each week that we have to omit some of them. Letters must be signed personally by all their authors. We cannot acknowledge their receipt unless a stamped addressed envelope or an international reply coupon is enclosed.

Correspondents should present their references in the Vancouver style (see examples in these columns). In particular, the names and initials of all authors must be given unless there are more than six, when only the first three should be given, followed by et al; and the first and last page numbers of articles and chapters should be included. Titles of papers are not, however, included in the correspondence section.

Provision of donors for renal transplantation 1978-80

SIR,—The debate following the recent BBC television programmes on transplantation and brain death has highlighted the paucity of factual information on the pattern of organ donation in this country. This review updates the West Midlands' information from my previous publication.[1]

The West Midlands transplant team based at the Queen Elizabeth Hospital, Birmingham, serves a population of approximately 5·5 million. During the period 1 January 1978-31 December 1980, 156 cadaver donors were operated upon in 31 hospitals in the region. All the donors were patients on ventilators in intensive therapy units in whom brain-stem death had been diagnosed clinically. There are facilities for electroencephalography (EEG) in four of the hospitals but a mobile EEG service is available in any of the hospitals on request by the doctor in charge of the patient. Of the 156 donors, 83 (53%) had suffered a head injury, most commonly following a road accident. Subarachnoid haemorrhage or similar medical conditions accounted for 55 deaths; seven deaths were from primary cerebral tumour; and the remaining 11 were due to a wide variety of causes.

It has been thought that organ donors come mainly from hospitals with neurosurgical facilities. Five of the 31 hospitals in the West Midlands have neurosurgical inpatient facilities. These hospitals provided 51 out of the 156 donors (33%). From the individual patient records there was no evidence that neurosurgeons were involved in 12 of the 51 donors (24%) even in these hospitals. It was also thought that the presence of a dialysis or transplant unit would encourage organ donation. Five of the 31 hospitals have dialysis units and they provided 50 of the 156 donors (32%). Three of the five also have neurosurgical facilities. Hospitals in teaching authorities might also be thought to be more attuned to organ donation. Eight of the 31 hospitals were in the Birmingham AHA(T). They provided 52 of the 156 donors (33%).

This evidence shows that the non-teaching, non-renal, non-neurosurgical hospitals are a very important source of cadaveric organs for transplantation (63 out of 156 donors (40%)). The more active of these provided one donor a quarter over the past three years. There was a relatively smaller proportion of organs from patients with subarachnoid haemorrhage (30%) from these hospitals compared with the hospitals with neurosurgical facilities (45%). There were, however, notable exceptions—

West Midlands RHA cadaveric organ donor activity 1978-80

Type of hospital	No of hospitals	No of donors	%	Head injury	Subarachnoid haemorrhage	Tumour	Other causes of death
Total	31	156	100	83	55	7	11
With neurosurgical facilities	5	51	33	20	23	3	5
With dialysis units	5	50	32	22	20	3	5
AHA(T) hospitals	8	52	33	26	17	6	3
No neurosurgical or renal units	25	105	67	63	33	3	6
Non-AHA(T) and no neurosurgical or renal unit	19	63	40	39	19	0	5

and from one peripheral district general hospital five of the eight donors had suffered a subarachnoid haemorrhage.

When the period 1978-80 is compared with the preceding five years a marked fall in the frequency of donations is seen in some hospitals and an increase in others. Further, there are hospitals where organs from patients who have had a subarachnoid haemorrhage are no longer forthcoming but head injury donors continue, and vice versa. As the overall pattern is one of a slow increase in donations (except during the most recent four months) the changing pattern is more likely to reflect changes in medical and nursing attitudes and management rather than any change among the general public.

I wish to thank the doctors and nurses in the 31 hospitals for the help and co-operation in the past and, hopefully, in the future. This effort has provided kidneys for just less than half the potential recipients of kidney grafts.

A D BARNES

Queen Elizabeth Hospital,
Birmingham B15 2TH

[1] Barnes AD. *Injury* 1979;**11**:10-12.

A matter of life and death

SIR,—May I suggest that in the patient who may be brain dead cerebral activity should be assessed not only for quantity but also for quality? If my brain stem was dead and I was therefore doomed, but had cerebral electroencephalographic activity suggesting consciousness or a functioning thalamus, then this existence could be unbearably agonising. I would wish to be put out of my misery without delay, not by nephrectomy but by morphia.

COLIN W BROWN

Knightwood Hospital,
Glasgow G13 2XG

Evidence for previous hepatitis B virus infection in alcoholic cirrhosis

SIR,—Dr P R Mills and others (7 February, p 437) have reported an increased prevalence of serum antibodies to hepatitis B virus antigens in alcoholic cirrhosis. We too have tested for antibodies to hepatitis B surface antigen (anti-HBsAG) and core antigen (anti-HBc) by radioimmunoassay (Abbott: Ansab and Corab) in patients with alcoholic cirrhosis, alcoholic liver disease without cirrhosis, and a control population of hospital patients with low alcohol intakes. All were Caucasian and HBsAg-negative and the control group was age and sex matched to the patients with alcoholic cirrhosis. Our results are shown in the table, which uses the same method of presentation as that of Dr Mills and his colleagues.

There was no greater prevalence of hepatitis B markers in the cirrhotic patients (14·8%) than in those with non-cirrhotic alcoholic liver disease (14·3%). Although antibodies were found more frequently in the high-alcohol groups than in the low-alcohol control group (4·9%), this was not statistically significant.

Our results from patients in London and West Yorkshire have failed to demonstrate an increase in hepatitis B markers in alcoholic cirrhosis, and are similar to those of Omata *et al*,[1] who found them in only 10 out of 93 patients. Perhaps relevant to the understanding of the relationship between hepatitis B infection and the pathogenesis of cirrhosis in the alcoholic is the study of Tabor *et al*,[2] who found that alcohol infusions produced no significant alteration in the course of experimentally induced hepatitis B infection in chimpanzees.

D M CHALMERS
A W BULLEN

Department of Medicine,
St James's Hospital,
Leeds LS9 7TF

A J LEVI

Northwick Park Hospital and Clinical Research Centre,
Harrow, Middx HA1 3UJ

[1] Omata M, Afroudakis A, Liew C-T, Ashcarai M, Peters RL. *Gastroenterology* 1978;**75**:1003-9.
[2] Tabor E, Gerety RJ, Barker LF, Howard CR, Zuckerman AJ. *J Med Virol* 1978;**2**:295-303.

A 20-year prospective study of cirrhosis

SIR,—The latest "episode" in the series of studies of cirrhosis in Birmingham by Dr J B Saunders and colleagues (24 January, p 263) was of great interest. With their extensive research methods it was surprising that only 108 patients were traced in the six-year period 1971-6 (an average annual incidence of 6·5 per 100 000 adults).

In a population of similar size (397 605) in Tayside,[1] a retrospective study of all histologically proved cirrhosis cases from 1975 to 1979 inclusive revealed 217 patients (197 with cirrhosis alone and 20 in association with primary liver cell cancer).[2] Given the same proportion of patients above the age of 15, our overall incidence figure is 14·6 per 100 000 adults—more than twice that in Birmingham. This may indeed reflect the more severe problem of alcoholism in Scotland.[3]

In the period 1975-9 no dramatic changes in the epidemiology have been observed. We were not able to show any rise in incidence of cirrhosis, and the aetiological pattern had remained stable. The contributions of each type of cirrhosis in percentage terms was as follows: alcoholic cirrhosis 55%, cryptogenic cirrhosis 12%, primary biliary cirrhosis 11%, chronic active hepatitis 9%, haemochromatosis 4%, secondary biliary cirrhosis 2%, other types 3%, records untraceable 4%.

So far as age and sex distributions are concerned, the mean age of onset of cirrhosis was 55 years in men and 59 years in women. In alcoholic cirrhosis the mean age of onset was identical at 55 years in both sexes. We could not demonstrate any change in the male:female ratio for alcoholic cirrhosis during the five-year period of the study (2·3:1), which suggests that the incidence of this condition in females in this region is stable at present.

Our study is not of sufficient duration to make any statement on survival, etc, but we have no reason to believe that it is different from the Birmingham experience as it seems difficult for many Scottish patients to re-adjust to an alcohol-free existence after diagnosis has been established.

W STUART HISLOP

University Department of Medicine,
Ninewells Hospital and Medical School,
Dundee DD1 9SY

[1] Hislop WS. *Br Med J* 1980;**281**:1069.
[2] Hislop WS, Masterton N, Bouchier IAD, Hopwood D. *Gut* 1980;**21**:909.
[3] Sclare AB. *British Journal on Alcohol and Alcoholism* 1978;**13**:86-91.

Management of alcohol withdrawal symptoms

SIR,—I should like to comment on several points in your leading article (14 February, p 502) "Management of alcohol withdrawal symptoms" drawing on my experiences with the Edinburgh alcoholic detoxification project, which, inter alia, studied 337 admissions for detoxification.[1] This project, "which sought to provide a medical and rehabilitative alternative to penal management of habitual drunken offenders," was first located in a regional poisoning treatment centre and found so many difficulties and problems in caring for such patients in a general medical ward that the facility was transferred to a psychiatric hospital. Your editorial recognises that some patients can be disruptive and our experience showed that sympathetic handling is much easier when the nursing staff concerned are used to the care of patients exhibiting disturbed behaviour.

The psychiatric hospital staff were no less efficient in their management of either the withdrawal syndrome or the many pathological conditions associated with chronic alcoholism. Among the 337 admissions, only once was gastric lavage required (on a patient who had also taken a drug overdose) and intravenous fluids were never used. Potassium supplements and vitamin preparations were never necessary. Readers should note a misprint in your article: the dose for chlormethiazole is given in grams instead of milligrams. We used decreasing doses of chlormethiazole starting at 1500 mg every six hours.

Although for many patients withdrawal symptoms will start "six to eight hours after the last drink" it should be remembered that symptoms start when the blood alcohol level drops below what is "normal" for the individual because of his tolerance. In the Edinburgh project blood alcohol concentrations ranged from 240 to 420 mg/100 ml and many patients were seen having withdrawal symptoms while still drinking.

Hepatitis B antibodies in patients with alcoholic liver disease and controls

Group	Diagnosis	No of patients	No of women	Mean age (years)	No of patients with: Anti-HBc alone	Anti-HBs alone	Both anti-HBc and anti-HBs	Total No of patients with hepatitis B virus antibodies
1	Alcoholic cirrhosis	61	17	53	4	2	3	9
2	Non-cirrhotic alcoholic liver disease, age matched for 1	49	16	53	2	3	2	7
3	Hospital controls, matched for 1	61	17	52	2	1	0	3