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 should be typed with double spacing between lines and must be signed personally by all their authors, who should include their degrees. 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.

Blood donors with a history of jaundice

SIR,—The leading article from Dr P M Jones (25 September, p 834) reopens the question of whether blood from donors with a stated history of jaundice is safe for transfusion. In an earlier study from the west of Scotland[1] we found that these donors were much more likely to have had an infection with hepatitis A virus than with hepatitis B virus. In addition, we found that a history of jaundice was no more common among carriers of hepatitis B surface antigen and hence was of little use as a marker of hepatitis B infectivity.

A history of jaundice is obtained from 2·8% of blood donors[2] in the west of Scotland—Alter's American figure is hardly relevant to the UK.[3] We have now studied a group of donors according to the age at which the jaundice occurred. Almost all the episodes of jaundice occurring before the age of 13 years were due to hepatitis A infection (table), but about 20% of those with jaundice in adolescence or later had no markers for hepatitis A or B. Other viruses can cause jaundice—for example, Epstein-Barr virus, cytomegalovirus, Coxsackie virus, adenovirus—and many other agents can cause liver problems. We cannot, therefore, equate unexplained jaundice with infection by the elusive non-A, non-B viruses. Indeed, it is uncertain whether sporadic non-A, non-B hepatitis is caused by the same agent as the form of the disease transmitted by transfusion, and it is not known how often a carrier state follows sporadic infection. Furthermore, it is possible that, as with hepatitis B, clinical jaundice may be an indicator of elimination of virus rather than carriage.

Viral hepatitis markers in blood donors with a history of jaundice

Age at time of jaundice (yr)	No	Hepatitis A virus immunoglobulin	Anti-HBs or anti-HBc or both	Neither A nor B
3-12	101	99	4	2 (2·0%)
13 and over	72	55	8	15 (20·8%)
Total	173	154	12	17 (9·8%)

Sera were tested by radioimmunoassay (Abbott Diagnostics) for hepatitis A virus immunoglobulin, anti-HBc, and anti-HBs.

The risk of post-transfusion hepatitis of 10% is an American estimate[4] and cannot be extrapolated to European transfusion services. In the last three years this region has transfused nearly 400 000 donations of blood and their derivatives. Only 12 cases of overt post-transfusion hepatitis possibly attributable to non-A, non-B agents have been notified. Of these, four were haemophiliacs who had been receiving imported blood products in addition to Scottish large-pool factor concentrate. None of the donors involved in the eight cases associated with red-cell transfusion had given a history of jaundice, and these cases could not have been prevented by the policy proposed by Dr Jones.

As the sensitivity and specificity of serological tests for non-A, non-B carriers have yet to be proved we could find ourselves excluding 2·8% of donors because of a history of jaundice, perhaps 2% because of serological findings, and a further 3% on the strength of alanine aminotransferase concentrations. The use of alanine aminotransferase concentrations has not been validated for UK volunteer donors.[5]

The present British policy appears to be correct, and any change could cause a serious loss of blood products when some regions are still struggling to make 80% of the blood plasma they collect available for factor VIII production. We endorse Dr Jones's encouragement to doctors to report all cases of post-transfusion jaundice.

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[1] Follett EAC, Barr A, Crawford RJ, Mitchell R. *Lancet* 1980;i:246-9.
[2] Crawford RJ, Barr A, Macvarish I, Dow BC, Mitchell R, Follett EAC. *Lancet* 1979;ii:155.
[3] Alter HJ. *Vox Sang* 1981;41:112-3.
[4] Vyas GN, Blum HE. *N Engl J Med* 1982;307:628-9.
[5] Mitchell R. *Vox Sang* (in press).