Correspondence

Blood donors with a history of jaundice

Sir,—The leading article from Dr P M Jones (25 September, p 934) reopens the question of whether blood donors with a stated history of jaundice are safe for transfusion. In an earlier study from the west of Scotland1 we found that a history of jaundice was no more common among carriers of hepatitis B virus than with hepatitis B virus. In addition, it is possible that, as with hepatitis A virus, clinical jaundice may be an indicator of elimination of virus rather than carriage.

Viral hepatitis markers in blood donors with a history of jaundice

<table>
<thead>
<tr>
<th>Age at time of jaundice</th>
<th>Hepatitis A virus</th>
<th>Hepatitis B virus</th>
<th>Anti-HEV</th>
<th>Neither A nor B</th>
</tr>
</thead>
<tbody>
<tr>
<td>3-12</td>
<td>101</td>
<td>99</td>
<td>4</td>
<td>2 (2-0%)</td>
</tr>
<tr>
<td>13 and over</td>
<td>72</td>
<td>55</td>
<td>15</td>
<td>20 (26-0%)</td>
</tr>
<tr>
<td>Total</td>
<td>173</td>
<td>154</td>
<td>17</td>
<td>22 (17-9%)</td>
</tr>
</tbody>
</table>

Serum was tested by radioimmunoassay (Abbott Diagnostic) for hepatitis A virus immunoglobulin, anti-HBc, and anti-HBc.

The risk of post-transfusion hepatitis of 10% is an American estimate2 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 absence of hepatitis A and 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 donors3 in the west of Scotland—Alter's American figure is hardly relevant to the UK.4 We have now studied a group of donors according to the age at which the jaundice occurred. All the cases 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.

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