SIR,—On Nov 30, 1986, surrogate testing—ie, testing for hepatitis B core antibody (anti-HBc) and alanine aminotransferase (ALT)—to prevent post-transfusion non-A, non-B (NANB) hepatitis became a requirement for accreditation to the American Association of Blood Banks. Will the rest of the world now have to follow suit?

The introduction of surrogate testing in the USA is based on data which suggest that about 7% of recipients of volunteer donor blood acquire post-transfusion NANB hepatitis, and evidence, from a small number of cases, that half of those so infected will proceed to chronic NANB hepatitis with the further risk of cirrhosis of the liver.1

The frequency of post-transfusion NANB hepatitis is about 27% for other than voluntary blood donations in the USA.1 However, an Australian study2 found that less than 3% cardiac surgery patients transfused with blood from volunteer donors acquired NANB hepatitis. There is no equivalent British study on the incidence of post-transfusion hepatitis or its long-term sequelae. Collins et al in 1983 reported the incidence of jaundice after cardiac surgery and the 1974 Medical Research Council study3 reported on the frequency of raised liver enzyme values after transfusion—but this was before the introduction of testing of donor blood to exclude other causes of post-transfusion hepatitis.

Between June, 1981, and October, 1983, Harefield Hospital provided the North London Blood Transfusion Centre with freshly frozen serum samples from patients undergoing cardiac surgery who had received 3 or more units of blood. The patients were sampled preoperatively and 2 months postoperatively. Of 186 patients assessed only 6 had raised ALT levels, and only 1 reached a level at which NANB hepatitis might have been diagnosed on American criteria.1 This would give a frequency for post-transfusion hepatitis of less than 1%, assuming that all ALT increases were due to hepatitis and not to other complications of the patient’s treatment.

We collected more than 190 000 units of blood per annum, and reports of post-transfusion hepatitis are received from hospitals and investigated to try and identify the type of hepatitis and its source. Since 1974 the number of cases reported has been 3-9 per annum, most being attributed to hepatitis B virus.4 No association has been reported between cirrhosis and previous blood transfusion, nor do we have evidence in the UK of a high prevalence of post-transfusion NANB hepatitis or its severe clinical sequelae.

One type of surrogate test relies on detecting anti-HBc, this being assumed to identify donors who will have the highest risk of transmitting NANB hepatitis, on the premise that many of those likely to contract hepatitis B are also at risk for NANB hepatitis. The study of Koziol et al in the USA suggests that the incidence of post-transfusion NANB hepatitis might be reduced by up to 40% by excluding donors positive for anti-HBc, although there have been no prospective studies to confirm this.

ALT testing is an even less specific surrogate test because there are many causes for a raised ALT (eg, obesity, drugs, and alcohol). However, by excluding donors with an ALT above 45 IU/l it is predicted that a further 30% of NANB hepatitis might be prevented.

In 1973 we measured ALT activity in 1000 donors. 1.8% had an ALT greater than 40 IU/l (measured at 35°C, which is equivalent to 45 IU/l at 37°C).5 In August, 1986, we screened 2000 donors; 3.6% had an ALT greater than 45 IU/l at 37°C. This is more than double the previous frequency. It seems unlikely that this increase reflects a sharp rise in the number of NANB hepatitis infections; it is more likely that it reflects some other factor such as an increase in alcohol consumption, so that exclusion of these donors might have had no significant impact on the transmission of NANB hepatitis by transfusion.

In 1983-84 we ran a preliminary study to screen donors for anti-HBc by a competitive radioimmunoassay. 1.8% of donors were positive and would have been excluded with the use of this screening test. In 1985, when fewer donors were screened, the prevalence had fallen to only 0.6%. Although both these studies preceded testing for antibodies to human immunodeficiency virus (HIV), the 1985 study followed extensive education to promote self-exclusion of donors at risk of transmitting HIV, which might explain the reduced prevalence of anti-HBc in the second series.

The above data raise the following questions:

(1) Is there any evidence that the incidence of post-transfusion NANB hepatitis in the UK is similar to that in the USA?

(2) Are the epidemiological patterns and clinical sequelae of the NANB virus(es) the same in the UK and the USA?

(3) Will exclusion of donor blood with an ALT greater than 45 IU/l reduce the incidence of post-transfusion NANB hepatitis in the UK?

(4) With the advent of self-deferral and HIV screening in the UK, will detection and exclusion of anti-HBc positive donors significantly reduce the transmission of post-transfusion NANB hepatitis?

We estimate that it would cost our blood transfusion centre at least £254 000 a year to screen donors for the above two surrogate tests, taking no account of the loss to the service of the donors who would be deferred and of the cost of follow-up and counselling of rejected donors.

Before we are forced to accept two screening tests of unproven benefits, which have high revenue implications, we need a national study to assess the incidence of raised ALT and anti-HBc in donors in different parts of the country. Also, and perhaps more importantly, a study is needed to assess the incidence of acute post-transfusion NANB hepatitis and to assess how many of those affected develop evidence of chronicity and serious clinical sequelae.

If the true incidence of post-transfusion NANB hepatitis and its serious clinical sequelae are as much lower than reported from the USA, then screening of donations to reduce the incidence of NANB hepatitis may not be cost effective in the UK.

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