Red cell and platelet transfusions and patient's haemoglobin levels.

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has not been routine practice to test ABO-incompatible donors for high-titre anti-A and anti-B. This is unlikely to cause any problem with platelet transfusions in the form of multiple standard platelet concentrates, but haemolysis is much more likely with platelet concentrates prepared by platelepheresis of a single high-risk donor. We report a severe haemolytic reaction in a group A+ patient given a single donor platelet concentrate with high-titre anti-A.

A 30-year-old woman with acute myeloblastic leukaemia was given a platelet concentrate with group A, Rh(D)+, negative. Stored serum from the donor, taken at the time of transfusion, had an anti-A titre of 512 by saline agglutination, and 2048 by the indirect antiglobulin test. The donor was male and had no history of transfusion or recent inoculations or infection that might have boosted his IgG anti-A titre. The greater severity of the later episode was probably related to the greater volume of plasma infused (448 ml vs 255 ml).

You recommend that group O donors on plateletpheresis panels should be tested for high titre anti-A and anti-B. However, precise guidelines have not been set, and there is no generally agreed discriminatory test. It is most important to avoid transfusing high titre immune anti-A to an A+ recipient (as in our case). Group A patients should therefore receive platelets from group A donors wherever possible, and attention should be given to volume of donor plasma in single-donor platelet concentrates.

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Lyme borreliosis and Raynaud's syndrome

SIR,—We believe that some cases of Raynaud's syndrome1—usually those that need unpleasant and expensive investigations and treatment—are caused by Borrelia burgdorferi. These cases might represent a particular type of Lyme borreliosis. We report such a case.

In June, 1989—an unusual time for the appearance of Raynaud's syndrome—a 38-year-old woman with serious symptoms of Raynaud's syndrome was referred to our department. Her disease had begun suddenly, a short time earlier. Her fingertips were cyanosed and painful. She had two pin-head ulcers on the first and second fingers of her left hand. Digital arteries were doppler positive only after warming her hand in water. All laboratory indices were normal, apart from the presence of serum antibodies to Borrelia burgdorferi (titre 3200) detected by the passive haemagglutination method (PHA; 'Lymag', Diagast, France). An absorbent (Diagast) was used to remove cross-reacting antibodies from the sample before titration. PHA titres above 20 were judged of diagnostic importance (manufacturer's recommendation).

After 8 weeks' treatment with doxycycline 300 mg daily, metisoprinol 3 g daily, and multivitamins, the hand ulcers healed. Finger tip erythema disappeared and an algid attack could be provoked only by very cold water or ice. After treatment the serum antibody titre was 1600. The patient has remained symptom-free up to December, 1989, and the antibody titre has decreased to 400.

A sample from the reaction mixture was subjected to agarose-gel electrophoresis, and primer pairs derived from the Chiron HCV sequence (primers C3751/C3930R and C3766-E/C3915R-B) none of the six sera was positive for HCV RNA (figure, lanes 8–13). Another ten sets of primer pairs from other regions of the Chiron genome also produced unsatisfactory results.

HCV isolates showed extensive sequence diversity and a PCR technique based on conserved sequences may revolutionise assays for detecting HCV RNA and yield valuable information on the biology of HCV.

Detection of serum hepatitis C virus RNA

Serum from six patients was analysed for HCV RNA by PCR and was synthesised to contain a BamHI recognition site at its 5' end. The primers were used to test for HCV RNA in the sera of six chronic non-A, non-B hepatitis patients who were positive for HCV antibody (Ortho Diagnostic Systems enzyme immunoassay). After one round of amplification, the PCR-amplified sample was reamplified with a second pair of primers internal to the original pair (PCR-PCR).

In all six samples an HCV cDNA band of expected size (167 bp) was produced on ethidium bromide fluorescence (figure, lanes 1–6). On the other hand, on PCR-PCR with two successive sets of primer pairs derived from the Chiron HCV sequence (primers C3751/C3930R and C3766-E/C3915R-B) none of the six sera was positive for HCV RNA (figure, lanes 8–13). The nucleotide sequences of HCV genome; primer JK3966R, from the reverse (R) strand (5'-ATCCGCTGATGAAGTTCCACATGTGCTTC-3') begins at position 3951 and was synthesised to contain an EcoRI recognition cleavage site at its 5' end; and primer JK3951R-B (5'-ATGGAATTCTTCCACATGTGCTTCGCCCAG-3') begins at position 3794 and was

Serum from six patients was analysed for HCV RNA by PCR-PCR. Serum was extracted from 800 μl of serum, cDNA was synthesised with reverse transcriptase, and amplified by PCR-PCR using two sets of primer pairs—namelyJK3779/JK3966FR and JK3794-E/JK3951R-B in lanes 1–6 and primers C3751/C3820R and C3766E/C3915R-B in lanes 8–13. A sample from the reaction mixture was subjected to agarose-gel electrophoresis, and nucleic acid was visualised under ultraviolet light after staining with ethidium bromide. Lane 7 = molecular weight standard 4X174 (HaeIII digests).

HCV RNA in serum of patients positive for antibody to HCV.

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