Table 1. *Three cases of HDN with anomalous laboratory results following successful intrauterine transfusions*

	Case One	Case Two	Case Three
Antibody specificity causing HDN	Rh1	Rh1	Rh1
Highest maternal titer	256	128	512
Other antibodies	Rh2,Rh3	none	Rh2,Jk1
IUT, weeks gestation	27,29	26,28	25,27
Milliliters transfused (RBCs)	70,100	70,90	50,65
Weeks gestation, delivery	33	32	29
Cord sample DAT	Negative	Negative	Negative
Cord sample Rh:1 Type	0	Weak,mixed field D^u	0
Cord hemoglobin (g/dl)	8.3	11.3	8.1
Normoblasts/100 RBCs	10	19	18
Highest reticulocyte count (%)	2.0	5.8	2.0
No. transfusions postpartum	4	6	9
Last transfusion (weeks)	5	3	6
Correct phenotype (weeks)	7	—	8

cells were of donor phenotype. Kleihauer stain showed 11 F-cells per 2000 adult cells, and blood films contained few erythroblasts. An enzyme-linked antiglobulin test indicated 0.29 percent fetal cells.[5] Reticulocyte count was 2 percent initially, and subsequently declined. Transfusions were carried out on postpartum days 1, 7, and 14. Discharge (day 35) reticulocyte count was 0.2 percent, and on day 49, it was 2.5 percent with hemoglobin concentration of 6 g per dl. Transfusion was repeated at another hospital. At 51 days, the correct phenotype was identified as A; Rh1,-2,3,4,5 (DcE/dce). Mixed field agglutination was observed for A and Rh3 (E) cells. Details are tabulated with cases 2 and 3.

Cases 2 and 3. See Table 1.

Severe fetal hemolysis followed by IUT markedly dilutes neonatal red cells with donor cells. Furthermore, erythropoiesis is depressed in premature infants. Thus, initial cell typing and DAT may be deceptive. Since intrauterine hemolysis may occur in diseases other than alloimmune HDN, definitive diagnosis must await regeneration of cells possessing the pathogenic antigen and repeated testing.

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Acute hemolytic transfusion reaction due to ABO incompatible plasma in a plateletapheresis concentrate

To the Editor:

Acute hemolytic transfusion reactions following the administration of ABO-incompatible plasma in platelet concentrates have been reported,[1-3] but their potential severity is often not appreciated. We observed the occurrence of such a reaction following the infusion of a type O plateletapheresis concentrate into a type A recipient. The patient was a 15-year-old white girl (blood type A, Rh negative) with acute lymphoblastic leukemia, who had received a bone marrow transplant from a histocompatible, ABO-compatible sister. Transfusion support was administered in the form of group A red cells and group A single-donor platelet concentrates obtained by plateletapheresis. On the 46th posttransplant day, she received a 200 ml of group O single-donor platelet concentrate from another sister. During the transfusion, the patient experienced chills and back pain. Her blood pressure dropped from 150/80 torr pretransfusion to 110/40 torr immediately posttransfusion, her pulse rate increased from 96 to 180 per minute, and her temperature rose from 98.6 ° to 103.4 ° F. Bleeding from the gums and intravenous sites was noted, and disseminated intravascular coagulation was documented by laboratory studies. Hemolysis was evident on observation of the patient's posttransfusion blood samples. A direct antiglobulin test was positive, and anti-A was eluted from the patient's cells. Saline anti-A titer performed on the sister's serum was 8192. The titer of dithiothreitol-treated serum was 4096. No hemolysin titers were performed. The patient became anuric, and within 18 hours of transfusion the creatinine rose from 0.9 to 3.0 mg per dl, the serum urea from 29 to 56 mg per dl, and the bilirubin from 0.7 to 8.9 mg per dl. The patient required hemodialysis, but renal function improved gradually. She was discharged on the 121st posttransplant day, at which time the creatinine clearance was 83 ml per minute. At the time of this report, 200 days posttransplant, she remains in remission with mild pancytopenia and does not require transfusion support.

Hemolytic trasnfusion reactions subsequent to infusion of ABO-incompatible plasma in platelet concentrates, although rare, may, as illustrated by this case and others,[1-3] cause significant morbidity. The hemolytic reaction was more severe in our patient, who received 200 ml of high-

titered single-donor plasma, than in the patients previously reported, who received 50 to 100 ml of high-titered plasma as part of a pooled platelet concentrate. The risk of a severe hemolytic reaction may be greater in platelet concentrates obtained by apheresis because the high-titered plasma is not diluted by pooling.

Due to the need for highly selected apheresis support (e.g., HLA-matched, cytomegalic virus antibody-negative products), it is not always feasible to select only ABO-compatible donors. One method to reduce the risks associated with transfusion of incompatible plasma is the screening of "dangerous donors" for high titers of isoagglutinins. Although saline titers, hemolysin titers, and "immune" titers have all been used, unfortunately there is no agreement as to what constitutes a "dangerous" titer and no one assay method that appears superior for routine use.[4] Plasma removal is an alternative method that may be used to avoid complications from infusion of incompatible plasma.[5] Any screening or plasma removal method would create increased laboratory work and add to the cost of transfusion. Cost-to-benefit analysis may not favor establishing a screening procedure at this time, but if the use of plateletapheresis concentrates continues to increase, such testing may be necessary. Physicians should be aware of potential problems associated with the use of ABO-incompatible platelet concentrates.

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Rapid temperature check for heat blocks

To the Editor:

Many blood banks use multi-well heat blocks for their 37° C incubation. The temperature of a heat block usually is checked by keeping a thermometer in one of the wells and the temperatures of the other wells are assumed to be the same. However, temperature variations do occur within one heat block. In our laboratory, the temperature of individual wells in a 76-well heat block (set at 37° C) ranged from 35° C to 38.5° C. It would be time-consuming to test the temperature of each well with a thermometer. We modified a method[1] that allows a quick temperature check to be run on several wells simultaneously.

Cholesteric liquid crystals (Djinnii Industries, Inc., Dayton, OH) were used for the rapid temperature check. These crystals are long chains of organic molecules that exhibit the flow properties of a liquid and the optical properties of a crystal. When a cholesteric liquid crystal compound is heated, the crystals begin to melt. The compound enters an intermediate state called the "mesophase" or liquid crystal phase. While in the "mesophase," the compound is capable of scattering light into its color components. The color of the compound changes, as the temperature increases, from red, to green, to blue, and finally disappearing as the compound becomes a true liquid. A black background is necessary for the colors to be seen. Upon cooling, the order of color change is reversed. The same crystals can be used repeatedly. According to the product information brochure, the crystals show "no indication of fatigue after one million cycles."

Using a paper punch, cholesteric liquid crystals that had been coated onto a black plastic sheet were cut into small dots. The dots were placed in 12 × 75 test tubes, one dot per tube. Each test tube was then placed in the 37° C heat block. After 60 seconds, each test tube was taken out of the block and the color observed. The color must be noted within 5 seconds because as the dot cools its color reverts to black.

The color changes were reproducible and accurate when compared with thermometer readings. The colors and temperatures (°C) corresponded as follows: black, 36; red, 36.5; green, 37; blue, 37.5; and black, 38.

The temperature check using cholesteric liquid crystals is quick, accurate, and allows all wells of a 76-well 37° C heat block to be tested within 5 minutes.

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Lectin in Cannabis

To the Editor:

In a recent survey of plants for lectins, Hardman et al.[1] reported a failure to find a previously described lectin in *Cannabis sativa*. At least two reasons may explain this finding. First, extraction of seeds without prior removal of fats, oils, and possible inhibiting materials may lead to low yields of lectins, and second, there are large regional variations in plants. The presence of a lectin in some seeds of *Cannabis* recently has been confirmed by this laboratory,[2] although not in all batches tested. In addition, the same or similar lectin was found in the pollen of the plant. In the case of marihuana, the concentration of the lectin was found to be greater in the pollen than in the seed, and this indicates a possible new source of lectins.

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