HDN DUE TO ANTI-D AND ANTI-C

ANSWERS:

1. Calculate the likelihood of paternal transmission of an Rh positive gene. (Hint: start by listing his possible genotypes in DCE notation.)

Potential paternal genotypes:	*DCe/DcE*	*.89*
	DCe/dcE	*.06*
	dCe/DcE	*.05*
	dCE/Dce	*rare*
	DCE/Dce	*rare*

Likelihood of transmitting a D gene = 95%
Likelihood of transmitting a C gene = 50%

2. What considerations would be appropriate in deciding whether to use of maternal blood? How would you process it?

Maternal RBCs are automatically compatible. Maternal donor must meet all donor criteria , since transplacental transmission of viruses including HIV is not invariable, but transmission rates by transfusion are very high.. CMV has been a concern, but recent evidence suggests that maternal antibody is protective against in-utero disease. RBCs are transfused after washing to remove maternal anti-A and –B, leukocyte filtration to reduce the risk of CMV transmission, and irradiation to prevent GVHD.

3. Why did this mother become immunized in spite of receiving RhIG?

The mother was presumably immunized by exposure to her husband's Rh positive RBCs when they shared needles when using IV drugs.

4. Why did the antibody titer increase so dramatically after the transfusion in week 21.

PUBS or other invasive diagnostic procedures including amniocentesis may cause fetal maternal hemorrhage, further stimulating maternal antibody production.

5. How would you predict when the next in a series of IUT's is required?

One formula used to predict need for subsequent IUT follows:

$$\text{Final Hct on } D_0 \times (\text{Est. fetal wt. on } D_0 / \text{Est. fetal wt. on } D_X) \times (100 - X)/100$$

Solve for X, the # of days between one transfusion (D_0) and the subsequent transfusion (D_X)

6. How do you explain the infant's Rh typing results and DAT at birth?

The neonate appeared Rh and DAT neg because all of his RBCs had been replaced by donor cells.

7. Why might the baby have needed so many exchange transfusions?

Inspection of the hematocrits at the end of the last several intrauterine transfusions reveals that the infant was under-transfused. This happened because the bedside hemoglobinometer used in the labor room had not been calibrated, and was becoming progressively inaccurate. The fetus attempted to compensate with accelerated erythropoiesis. Ideally, intrauterine transfusion should inhibit fetal production of Rh positive RBCs. Instead, this newborn had a large liver and spleen, which would have contained many Rh positive RBC precursors. These RBC precursors were not removable by exchange transfusion, and as they were destroyed by the maternal antibody, the newborn developed hyperbilirubinemia. The relative IN-efficiency of exchange transfusion in removing bilirubin, compared with removing "potential" bilirubin (RBCs

destined for destruction) is also evident.