

INTRAUTERINE BLOOD TRANSFER BETWEEN UNIOVULAR TWINS

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The non-haemolytic anaemias of the newborn have recently attracted the attention of many investigators. Of particular interest has been the anaemia due to occult blood loss of the foetus into the maternal circulation (Wiener, 1948; Chown, 1954; Gunson, 1957.

Another type of intrauterine occult blood loss has been recognized in the last few years as being due to transfusion of blood from one uniovular twin to the other through anastomotic vessels in the placenta (Herlitz, 1942; Bosma, 1954; Klingberg, Jones, Allan and Dempsey, 1955; Bergstedt, 1957; Betke, Deibel and Schlicht, 1958; Sacks, 1959). The present paper describes a similar case and our attempts to rectify it.

Case Report

The mother, a multigravida, 29 years of age, gave birth eight weeks before term to twin girls, the first weighing 1,250 g. and the second 1,500 g. Both babies breathed and cried immediately after delivery. A striking difference in colour was noticed; the first twin (A) appeared flushed and red and the second (B) was pale without any other signs of anoxia or shock. Venous blood was examined six hours after birth and a marked difference in the Hb and R.B.C. content was found (twin A: Hb 27.8 g. per 100 ml., R.B.C. 9,000,000 per c.mm.; twin B: Hb 8.2 g. per 100 ml., R.B.C. 2,600,000 per c.mm.). As it became evident that there had been intrauterine transfusion of blood from twin B to twin A, we decided to correct this unequal share of Hb. Eight hours after birth polyvinyl exchange-transfusion catheters were introduced into the umbilical vein of the babies. The venous pressure was found at the same level of 6-8 cm. in both twins. Blood was withdrawn in amounts of 5 ml. from the plethoric twin and was immediately given to twin B. No anticoagulant was used, but each catheter, while not used, was filled with a solution of normal saline containing heparine (1,000 units to one litre). In all, 25 ml. of blood was removed from twin A and transfused into twin B. Both twins tolerated the procedure well and we could record no change in the venous pressure or cardiac rate. As this

first transfer of blood was not sufficient to bring the haemoglobin values near enough to normal the procedure was repeated 31 hours after birth when a further 25 ml. of blood was transferred from twin A to twin B. Full haematological details are recorded in Table 1.

The clinical condition of both babies continued to be good until the fifth day when twin A developed broncho-pneumonia and died two days later in spite of treatment with antibiotics. At necropsy the diagnosis of broncho-pneumonia was confirmed. The other twin continued to thrive and develop normally.

As soon as the diagnosis of intrauterine transfer of blood was made the placenta was examined. This was a single-ovum, monochorial, diamniotic placenta, weighing 680 g. One of the cords had a marginal insertion and its vessels spread over the larger part of the placenta. The insertion of the other cord was membranous and three of its vessels were found ruptured. By that time it was impossible to find out to which of the twins each cord belonged. A large yellow infarct measuring 5 x 6 cm. was seen in the periphery and between the two parts of the placenta. No difference in colour or fullness of the vessels between the two parts could be seen. The rupture of the cord vessels prevented us from performing a full 'milk test'. Nevertheless, neither by naked eye nor by injection of fluid through the other cord could we see a major anastomosis.

TABLE 1
BLOOD DATA OF THE TWINS

	Twin A	Twin B
Birth weight in grams	1,250	1,500
Colour at birth	Dark red	Very pale
Blood type	A, D	A, D
Venous blood at 6 hours		
{ Hb g./100 ml.	27.8	8.27
{ Haematocrit (%)	82	24
{ Hb g./100 ml.	23.6	7.6
{ Haematocrit (%)	74	22
Blood from umbilical vein at 8 hours		
{ R.B.C.s per mm. ³	7,900,000	2,600,000
{ Reticulocytes (%)	6.5	18
{ Erythroblasts per 100 W.B.C.	4	67
{ Foetal Hb (%)	87.3	83.2
Venous blood at 22 hours		
{ Hb g./100 ml.	21.9	10.7
{ Haematocrit (%)	72	34
Venous blood 3rd day		
{ Hb g./100 ml.	—	16.5
Venous blood 5th day		
{ Hb g./100 ml.	18.67	—

TABLE 2

DATA FROM REPORTED CASES OF INTRAUTERINE BLOOD TRANSFER

Author	Sex of Twins	Order of Birth		Birth Weight (g.)		Initial Hb (g./100 ml.)		Icterus		Treatment		Outcome		
		A	B	A	B	A	B	A	B	A	B	A	B	
Herlitz (1942)	1	M	II	I	2,580	1,580	160%	50%	None	None	None	Oral iron	A+W	A+W
Herlitz (1942)	2	F	I	II	3,030	2,220	200%	65%	None	None	None	Oral iron	A+W	A+W
Bosma (1954)	..	?	?	?	?	?	31	4	Marked	?	None	?	Kernikterus	?
Klingberg <i>et al.</i> (1955)	..	F	I	II	2,690	1,770	25.2	3.7	Moderate (S.B.12.3)	None	Removal of 100 ml.	Exch. transf.	A+W	A+W
Bergstedt (1957)	..	F	I	II	3,030	1,810	23.6	13.6	Marked (S.B.23.5)	Slight	None	Oral iron	A+W	A+W
Betke <i>et al.</i> (1958)	..	M	I	II	2,600	2,130	24.6	9.5	Marked (S.B.22.4)	Slight (S.B. 7.1)	None	Transfusion iron, etc.	A+W	A+W
Sacks (1959)	1	M	II	I	2,084	1,431	24	5.5	Marked	None	Bleeding attempted	Transfusion	Kernikterus	Cerebral palsy
Sacks (1959)	2	M	II	I	1,843	1,424	30	18	—	None	Removal of 60 ml.	Transfusion later	Died atelectasis	A+W
Sacks (1959)	3	F	I	II	2,523	2,041	33	12.8	Moderate (S.B.11.5)	None	Removal of 75 ml.	None	A+W	A+W
Sacks (1959)	4	F	II	I	1,163	1,021	27	15	—	—	—	—	Died at 16 hrs	Died at 26 hrs
Present case	..	F	I	II	1,250	1,500	27.8	8.27	Slight	None	Removal of 50 ml.	Transfusion	Died at 7 days	A+W

A = Polycythaemic twin. B = Anaemic twin.

Discussion

In the literature available to us we found 10 other pairs of twins exhibiting the same condition. Table 2 presents the relevant data from all the cases.

The present pair is the only one in which the polycythaemic twin was the smaller of the two. The unequal distribution of blood cannot be due, therefore, to the same cause as the unequal birth weight.

It is well known that there are numerous anastomoses of all types between the placental circulations of the identical twins (Schatz, 1898). The mere demonstration by the 'milk test' of the presence of these anastomoses constituting what is called 'third circulation' (Schatz, 1898) or 'parabiotic circulation' (Klingberg *et al.*, 1955) cannot explain the unequal distribution of blood. This could arise either during labour or exist for some time before. In our case the weight of evidence is in favour of the second alternative. There were no signs of oligoemia in the anaemic twin who, furthermore, showed marked erythropoietic activity. It seems, therefore, that the blood loss must have taken place sufficiently before labour to allow for the homeostatic mechanisms to reconstitute the blood volume and to stimulate the erythropoietic tissues. For the same reason blood loss through the ruptured vessels can be ruled out as the cause of the anaemia. Moreover, this explanation cannot account for the polycythaemia of the other twin.

Seip (1956) attributed the slightly higher haemoglobin values of the second-born observed in many

pairs of twins to an additional amount of blood received from the placenta due to more complete uterine contractions. However, this explanation does not account for the great differences observed in the reported cases, in more than half of which, the polycythaemic twin was born first.

The mechanism of the unequal distribution of blood before the onset of labour is not clear. Klingberg *et al.* (1955) observed a difference in the colour and fullness of the vessels between the two parts of the placenta and believed that the more distended vessels belonged to the cord of the anaemic twin. They postulated some sort of obstruction in the venous return to the anaemic twin causing back pressure and a shunt of blood to the polycythaemic infant. We believe this to be a likely explanation but in our case we could not confirm this observation because of the rupture of the cord vessels.

Both twins are at risk but for a different reason. The anaemic infant begins its life with a low Hb content and previous authors (Klingberg *et al.*, 1955; Bergstedt, 1957; Sacks, 1959) gave blood transfusions for the correction of the anaemia. In the polycythaemic twin severe jaundice with its possible consequences is the main risk and in two cases (Bosma, 1954; Sacks, 1959) kernikterus occurred, while in two others (Bergstedt, 1957; Betke *et al.*, 1958) the serum bilirubin was above 20 mg. per 100 ml. It seems that the higher risk of kernikterus in polycythaemic newborn infants is due to the increased load of red cell breakdown

products presented to the liver cells for excretion. Klingberg *et al.* (1955) and Sacks (1959), with this risk in mind, removed blood from the polycythaemic twin.

In our case we avoided the danger facing each twin by returning to the anaemic infant the blood which was shunted to the polycythaemic before birth. Technically this procedure proved easy and it seems the best way to deal with such a situation.

Summary

A pair of uniovular twins one of which was anaemic and the other polycythaemic at birth are described. This difference was due to intrauterine transfusion of blood from one twin to the other.

The fault was corrected by removing blood from the polycythaemic twin and transfusing it to the anaemic.

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Addendum

Another pair of uniovular male twins exhibiting the same condition was recently sent to us. Twin A weighing 2310 g. had Hb 23·3 g. and 7% reticulocytes. Twin B weighing 1670 g. had Hb 12·8 g. and 12% reticulocytes. When the infants were 14 hours old 40 ml. blood was taken from Twin A and given to twin B using the same technique as above; 24 hours later twin A had Hb 19·4 g. and of twin B 14·4 g. Twin A had slight jaundice never reaching dangerous levels.