Society committee appointed in 1885 to report on the origin of spina bifida gave as a cause the primary defect in the formation of mesoblast from which the structures closing in the vertebral furrow are developed.

Bland-Sutton (1888) advanced a theory in which he regarded the spinal cord as a highly differentiated section of the primitive alimentary tract. Its central canal was originally part and parcel of the system, but is now functionless and, like obsolete canals in general, is often the seat of cystic dilatation. The conclusion arrived at by Good is that it is the arrested development of the mesodermal tissues from which the spinal meninges are produced, and that it is due to a mechanical cause which prevents the neural plate becoming converted into a neural tube; there is no evidence as to the nature of this mechanical agent.

A few cases are on record of spina bifida occurring as a family defect. Butler-Smythe (1889) reports 3 cases of spina bifida with hydrocephalus occurring in the same family. Pybus (1921) describes a family in which 4 children had congenital defects of the spine. The eldest child died at 18 months with spina bifida associated with hydrocephalus; the second, 10 years old, had spina bifida occulta, indicated by a scar and depression in the upper part of the thoracic region of the spine, a radiograph of which showed a defect of the underlying vertebrae; the third had a meningocele in the lumbosacral region, treated successfully by operation; the fourth child had an exaggerated coccygeal dimple.

The only case on record of spina bifida in uniovular twins is from Norway (Eskelund and Bartels, 1941). There are no cases on record of spina bifida in binovular twins.

The dizygotic origin of the twins in the present case is confirmed by the two separate placentae and chorions and the absence of striking similarity in the general appearance of the two infants, which is in conformity with the foetal-membrane method and similarity method of diagnosis of zygotic origin of twins (Newman, Freeman, and Holzinger, 1937). This case tends to support the theory that the cause of spina bifida is primarily a defect in the development of the mesoblastic tissues from which the spinal meninges are produced, as this is the only factor that is likely to affect equally the two separate ova, rather than the mechanical agent or the external influences mentioned in the several theories advanced.

South Norwood.

A. FRY, M.R.C.S., L.R.C.P., D.M.R.

REFERENCES

- Bland-Sutton, J. (1888). *Trans. path. Soc.*, 39, 432.
 Boyd, W. (1938). *Textbook of Pathology*, p. 965, Philadelphia.
 Butler-Smythe, A. C. (1889). *Lancet*, 1, 272.
 Eskelund, V., and Bartels, E. D. (1941). *Nord. Med.*, 11, 2075.
 Good, J. P. (1915). *An Enquiry into Causation of Spina Bifida*, University of Birmingham.
 Gray's Anatomy (1938), p. 35, London.
 Koch, W. (1881). *Beiträge zur Lehre von Spina Bifida*, Kassel.
 Newman, H. H., Freeman, F. N., and Holzinger, K. J. (1937). *Twins: A Study of Heredity and Environment*, p. 32, Chicago.
 Pybus, F. C. (1921). *Lancet*, 2, 599.
 Wertheim, C. O. (1857). *M Schr. Geburtsh. Gynäk.*, 9, 127.

The Rh Factor in Haemolytic Anaemia of the Newborn

Boorman, Dodd, and Mollison (*Journal*, 1942, 2, 569) suggest that only group O Rh-negative blood should be used for transfusion in cases of haemolytic anaemia of the newborn, with or without severe jaundice. This suggestion is based on the assumption, for which there is already some evidence, that blood of this group will prove to be immune to the action of the immune bodies responsible for the haemolytic process. Unfortunately, at present few laboratories have supplies of potent anti-Rh serum with which to establish a panel of suitable donors. I should like to make an alternative suggestion on the basis of the following two cases.

CASE I

A female infant aged 5 weeks was admitted to the Children's Hospital, Birmingham, on Aug. 2, 1939. She was the second child and was delivered at term; the first was said to have been very pale for two months, but was now healthy. Jaundice was observed within a few hours of birth, but was never more than a lemon colour and had faded by the seventh day. Pallor soon became marked and it persisted till admission. On Aug. 2, 4, and 8 transfusions of maternal blood—45, 45, and 50 c.cm. respectively—were given, with substantial improvement in the blood picture and the general condition. A sudden drop in colour index, mean cell volume, and mean diameter was noticed at this time, and the change in shape of the Price-Jones curve was such as only to admit of the conclusion that the infant's cells had been completely replaced by those of the mother. An aregenerative phase ensued from Aug. 5 to Aug. 15, during which no change occurred save for a steady though relatively slow drop in the red cell count. The onset of a fresh reticulocytosis, however, was accompanied by a dramatic change in the Price-Jones curve indicating the steady replacement of the donor's

cells by those of the infant. The child died shortly afterwards of a severe intercurrent infection.

Table showing Laboratory Data of Case I

Date	Red Cells (mill.)	Hb %	C.I.	Retics. %	M.C.V.	M.D.	σ
2/8/39	1.23	27	1.08	16.3	92 μ^3	7.92 μ	0.885 μ
5/8/39	2.30	44	0.98	0.4	86 μ^3		
8/8/39	3.52	58	0.83	0	70 μ^3	6.80 μ	0.542 μ
14/8/39	2.54	40	0.80	0.6	71 μ^3	6.83 μ	0.495 μ
17/8/39	3.19	53	0.83	5.9	68 μ^3	7.26 μ	0.974 μ
21/8/39	2.61	45	0.88	14.4	77 μ^3	7.72 μ	1.102 μ
Donor's blood, 10/8/39	..	..	0.86		68 μ^3	6.92 μ	0.495 μ

CASE II

A female infant—fourth pregnancy. First pregnancy: breech delivery, stillbirth. The second child was a full-term female infant who developed a mild form of haemolytic anaemia of the newborn with moderate jaundice. She was treated with liver extract (campolon) only, and is now healthy. The third pregnancy miscarried at 24 weeks.

The mother was delivered at term in the Maternity Hospital, Loveday Street, Birmingham, early on Sept. 2, 1939. The infant's spleen was found to be one fingerbreadth below the costal margin at birth. Jaundice was evident at dawn and rapidly became more pronounced, but it was never severe and faded completely by the fifth day. There was no anaemia at birth, but pallor became progressively more obvious despite frequent injections of campolon. Transfusions of the mother's blood were given on Sept. 10, 12, and 14—45, 50, and 50 c.cm. respectively—with great improvement. Here again the change in colour index, mean cell volume, and Price-Jones curve indicated replacement of the child's cells by the donor's; there was also a short aregenerative phase from Sept. 15 to 20, and thereafter a steady increase in the reticulocyte count, though no significant improvement in the red cell count occurred till Oct. 13. From this date recovery was rapid and uneventful. It is perhaps worth noting that this sudden improvement followed an injection of 1 c.cm. of campolon on Oct. 10. This is the only occasion in my experience of noteworthy improvement following or coinciding with liver therapy in this disease.

Table showing Laboratory Data of Case II

Date	Red Cells (mill.)	Hb %	C.I.	Retics. %	M.C.V.	M.D.	σ
2/9/39	4.90	121	1.21	33.5	108 μ^3	7.99 μ	0.885 μ
8/9/39	1.33	32	1.23	11.3	108 μ^3		
10/9/39	1.01	24	1.20	3.6		8.04 μ	0.745 μ
15/9/39	3.08	58	0.97	0.4	80 μ^3	7.56 μ	0.580 μ
22/9/39	3.06	59	0.96	1.4	79 μ^3	7.50 μ	0.565 μ
3/10/39	2.86	52	0.91	10.4		7.59 μ	0.603 μ
10/10/39	2.31	44	0.96	11.1	75 μ^3		
13/10/39	3.28	60	0.91	21.6			
20/10/39	3.52	62	0.90	22.2	80 μ^3		
17/11/39	5.15	88	0.86	3.5	77 μ^3		
Donor's blood, 12/9/39	..	..	0.98		79 μ^3	7.39 μ	0.550

DISCUSSION

These two cases illustrate quite well the point I wish to make. When the degree of anaemia is such that transfusion is urgently needed it may be preferable to use maternal blood, the survival of which for some time is certain, rather than to risk repeated unsuccessful transfusions from a donor selected at random, and therefore probably Rh +, even though rapid destruction of the few native cells remaining may occur. I do not feel that this last is a valid objection to the procedure, for I take it the object of treatment in future will be to circumvent the haemolytic process in the early days by replacing the child's cells so far as possible by Rh-negative cells. On the other hand one would not willingly inject large doses of antibody if it could be avoided. There are two ways out of this dilemma. In the first place it is to be hoped that the serum of every woman whose child is affected will be examined for anti-Rh agglutinins. Where these are present in sufficient strength it will be possible, perhaps, to find a donor whose cells are compatible both with the serum of the child and with that of the mother, and in any case these women should be regarded as unsuitable as donors. The alternative, where facilities are available, would be to use the washed cells of the mother suspended in fresh plasma or saline (a suggestion I owe to my colleague Dr. H. S. Baar).

It may be argued that it is undesirable to depress bone-marrow function so far as in the cases described. I do not believe this effect to be due to the extra dose of antibody, but attribute it rather to success in raising the blood count substantially above the pre-transfusion level. Aplasia and hypoplasia of the bone marrow are exceedingly rare in icterus gravis and haemolytic anaemia of the newborn, which suggests that the main action of the antibody is exerted on the circulating erythrocytes rather than on the erythron as a whole.

T. W. LLOYD, D.M., M.R.C.P.