I am grateful to Dr. T. M. Barratt for permission to publish this case report.

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joint capsules, ligaments, and dura mater in all 27 cases. In some of the 27 there was extensive bruising and destruction of the spinal cord, haemorrhage into the media cases. In bruising and destruction of the spinal trauma were: upper thoracic more than 10% of all newborn infants at necropsy, the common sites of spinal injury being cervical and upper thoracic spine. Some factors that could have contributed to spinal injury in addition to birth trauma were: prematurity, intrauterine malposition, dystocia, and precipitate delivery.

Severe birth trauma to vital centres in the upper cervical cord and brain stem may lead to death shortly after birth. Infants who survive with spinal injury may have permanent neurological abnormalities due to damage to the spinal cord or vertebral arteries. The present case illustrates that spinal cord injury due to birth trauma can produce a paraplegia. Though there are recent reports of spinal cord injury due to birth trauma (Melchior and Tygstrup, 1963; Jones, 1970; Shulman et al., 1971), such injury in the newborn asphyxiated infant may be overlooked, attention being primarily directed to cerebral lesions. Thus, some cases of paraplegia and quadriplegia attributed to cerebral palsy may be suffering from the after-effects of spinal cord damage.

Summary

A neurologically abnormal infant who died at the age of 8 weeks was found to have spinal cord atrophy involving about 2.5 cm in the midcervical region. He was asphyxiated during birth and was delivered by breech extraction. Spinal cord injury was probably related to trauma associated with breech extraction. Asphyxiated babies are usually hypotonic and therefore may be particularly liable to sustain spinal injury.

We thank Dr. P. D. Moss (Blackburn Royal Infirmary) for allowing us to study this case and publish some of his clinical findings; Dr. C. K. Heffernan (Blackburn Royal Infirmary) for allowing us to publish his necropsy findings; and Dr. F. N. Bamford (St. Mary’s Hospital) for helpful advice.

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Congenital erythroid hypoplastic anaemia in mother and daughter

Pure red cell anaemia, congenital erythroid hypoplastic anaemia, the syndrome of Diamond and Blackfan (1938) was first described briefly by Josephs (1936). Despite the many reports and reviews since then, there are only 9 familial occurrences of well-documented overt disease recorded, all in sibs (Burgert, Kennedy, and Pease, 1954; Diamond, Allen, and Magill, 1961; Förare, 1963; Seligmann et al., 1963; Mott, Apley, and Raper, 1969). Nevertheless, the two separate and unusual families reported by Förare (1963) and Mott et al. (1969), where step-sibs, progeny of the same father by different mothers, suffered the anaemia, suggest that congenital erythroid hypoplastic anaemia can be transmitted in a mendelian-dominant fashion. This report documents definite vertical transmission of the disease from mother to daughter.

Case reports

Mother. Born of unrelated parents on 7 November 1945, after a term normal pregnancy. Birthweight 2270 g, blood group B, Rhesus negative. She presented at 21 months with pallor and listlessness, Hb 4.7 g/100 ml, normal red cell morphology, white cell count 9400/mm³, and normal differential count for her age. She had a urinary infection and was treated with alkali and oral iron. Hb rose to 10.2 g/100 ml over 2 months. At 3 years severe anaemia recurred, Hb 4.9 g/100 ml, white cell count 3700/mm³, reticulocytes 8%, the marrow showing selective erythroid hypoplasia. Investigations excluded haemolysis, mucoviscidosis, and malabsorption, and Hb rose with iron, liver extract, and folic acid to 10.9 g/100 ml over 6 months. Convalescence was
Spinal cord damage in a newborn infant.

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