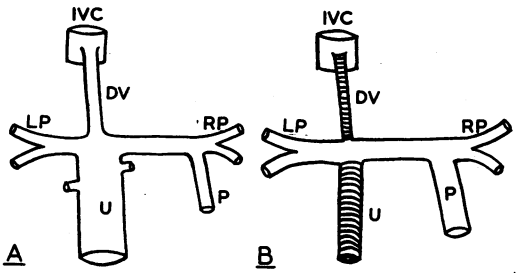


Fig 2 *Portal and umbilical venous system, viewed from below.* A, *before birth.* B, *after birth. U, umbilical vein. P, portal vein. LP, RP, left and right branches. DV, ductus venosus. IVC, inferior vena cava*

When, after birth, umbilical flow ceases, the volume and pressure of blood in the system fall; the direction of flow in that part of the left portal vein between the umbilical vein and the main portal vein reverses, and the ductus, like the umbilical vein, becomes a stagnant channel (Fig 2B), though it does not close anatomically for some time. In a series of 762 cases, 37·5% showed closure in the third week of life, 75·7% in the fourth week, and 97·3% closure by the end of the eighth week (Scammon & Norris 1918). In the presence of infection the speed of closure would probably be altered, but the salient feature is that in healthy infants of the age of those in this series, nearly one-quarter could be expected to have a patent ductus venosus.

*Site of Infection*

If umbilical infection occurs in the neonatal period, there are two vascular channels in which it can remain dormant: the umbilical vein itself and the ductus venosus. If the umbilical vein is involved, the source of infection is evident. Late spread can either be from its extrahepatic portion (Case 2) or intrahepatic in origin with multiple liver abscesses (Case 3). If the ductus venosus alone is involved, however, spread is likely to be hæmatogenous, as the contents of the ductus are discharged directly into the vena cava. The child will then present with a distant infection, either pneumonia (Case 1) or meningitis (Case 4) and the source of infection may not be appreciated unless the ductus is specifically examined. It is unlikely that either of these infections would have been assigned to the correct source if there had not been a known outbreak of umbilical sepsis current at the time.

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# Transient Choreo-athetosis Following Severe Anoxia

**(demonstrated by a film series)**

by J E Cree MB DCH
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*Case History*

On July 26, 1966, N R, a healthy 6-year-old boy, inhaled a plastic bullet which completely obstructed his trachea. The bullet was removed twenty minutes later at bronchoscopy, by which time he was pulseless with widely dilated pupils. He responded to resuscitation, but remained unconscious and had generalized convulsions. He was given intravenous 10% mannitol, intravenous corticosteroids and phenobarbitone.

Two days later he appeared lucid at times but by the following day was again unrousable. Convulsions recommenced and he developed continuous choreo-athetosis of his face and limbs, when awake, only partially controlled by drug therapy. Over the next few days he became semiconscious, emitting high-pitched cries and dysarthric speech. This level of consciousness continued for three weeks, when he began to answer questions appropriately and to regain his memory, though he seemed to be blind. His involuntary movements continued, except when he was asleep, and he then appeared to be hypotonic with normal tendon jerks and flexor plantar responses.

Seven weeks after his anoxia he was able to sit, to hold his head erect and to walk with support. Hearing and sight were tested and found to be normal. An EEG showed no focal abnormality. When discharged twelve weeks after his anoxic episode he appeared an intelligent euphoric child, with occasional involuntary movements, ataxia and a mild left hemiparesis.

In February 1967 he again developed generalized convulsions which were controlled with difficulty.

Two years after the episode he is a happy, well-adjusted child, with normal sight and hearing and an IQ of 100. He has a very mild hemiparesis affecting his left leg; co-ordinated movements, such as writing, he still finds difficult, but he has no other signs of damage to his cortex, basal ganglia or cerebellum.

*Content of Film*

*First sequence*, five weeks after anoxia, shows N R lying on his back, with incessant choreo-athetosis of all limbs and head. He is unable to sit without support, or perform any voluntary movements other than chewing and swallowing. He does not appear to see.

*Second sequence*, three months after anoxia: He is walking a few steps, hampered by his involuntary movements and left hemiparesis. He now sees, but actions such as putting a spoon to his mouth are very clumsy.

*Third sequence*, twenty-one months after the anoxia, shows an intelligent, over-eager child with a normal gait, well able to ride his bicycle. Finer movements required in dressing himself are still a little clumsy.

The extent of N R's recovery seems remarkable. Foley (1954) reported a similar occurrence, in a woman of 25 who, following four minutes' apnœa, became stuporous and had athetosis persisting for four months. Polani & Mac Keith (1954) also reported the effects of anoxia in two children aged 3½ and 7½ years, both of whom had athetosis and visual field defects two years after the incident. In none of these reported cases has recovery been so complete as in ours.

Denny-Brown (1965) suggests that such disorders of function may not be due to direct injury to the basal ganglia, but to demyelination of their connexions to the cerebrum. There is evidence (Peters 1960) that the oligodendrocyte is responsible for the formation of myelin in the central nervous system. Is it possible that remyelination has occurred to produce restoration of normal function in this case?

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## Transient Choreo-athetosis Following Hypernatræmia [*Summary*]

by T P Mann FRCP
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A male infant aged 18 months and his baby sister aged 5 months were admitted together in coma with severe gastroenteritis. Both children had severe hypernatræmia, both showed temporary choreo-athetosis (the boy more than the girl) and both made a rapid and full recovery.

[For a full report *see* Mann T P (1969) *Develop. Med. Child Neurol.* (in press)]

## Preliminary Observations on Two Variations of the Placental Transfusion Syndrome

by M J Dillon MRCP DCH[1]
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Many cases of the placental transfusion syndrome have now been reported with particular emphasis on the hæmatological features and on the placental structure. The literature has been well reviewed by Aherne *et al.* (1968). In this paper two further examples of the syndrome will be described, in one of which symptomatic hypoglycæmia and in the other significant hypocalcæmia occurred in the donor twins.

*Case Histories*

**Cases 1 and 2** H twins

Mother primigravida aged 22. Normal pregnancy. Spontaneous onset of labour at 39 weeks. Case 1: plethoric-looking fresh male stillbirth weighing 8 lb 2 oz (3,685 g). Case 2: pale apnœic male weighing 5 lb (2,268 g). Twenty-five minutes' artificial respiration before breathing established. Placenta uniovular. *Second twin (Case 2):* Initial Hb 102%. Respiratory distress with a right pneumothorax developed within the first hour but subsequently resolved. Two apnœic attacks and one convulsion occurred in the first 12 hours. During latter blood sugar 32 mg/100 ml. Cardiac arrest at 15 hours. No colour change on Dextrostix. Dramatic improvement with intravenous 25% dextrose. Further apnœic episodes at 21 and 37 hours associated with blood sugars of 28 and 11 mg/100 ml respectively in spite of continuous intravenous 10% dextrose and parenteral steroids. Good response with intravenous 25% dextrose. Blood sugars by Asatoor & King's method (1954) and therefore true glucose probably lower.

At 60 hours, total plasma protein 3·7, albumin 2·6 g/100 ml. Hypocalcæmia at 84 hours, serum calcium 5·5 mg/100 ml, probably due to hypoproteinæmia. Intravenous calcium gluconate administered. At 90 hours, Hb 76%; 40% normoblasts recorded on nucleated cell count; platelets 11,000/mm³.

The baby deteriorated and died shortly after this. *Post-mortem findings:* Case 1: unaerated lungs and all tissues grossly congested with blood. Case 2: pale baby with pneumonic changes in lungs but no other gross abnormality.

**Cases 3 and 4** W twins

Mother aged 29. Second pregnancy. Uneventful apart from mild hypertension. Spontaneous onset of labour at 38 weeks. Case 3: plethoric male infant weighing 5 lb 7 oz (2,466 g); cord blood Hb 158%; baby did well subsequently; artificially fed. Case 4: breech delivery; pale male infant weighing 5 lb 1 oz (2,296 g); cord blood specimen clotted but capillary Hb next day 80%. Placenta uniovular.

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