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Spinal cord damage in a newborn infant

The obstetrician may sometimes have to decide between rapid delivery, with the risk to the baby of traumatic injury, and delay with its risk of severe birth asphyxia. Strain imposed on the neck of the baby during delivery may in certain cases damage the brain stem and spinal cord (Yates, 1959; Towbin, 1969). The mechanical effects on the spine of manipulation of the head and trunk are important especially during breech delivery, but so is the state of the baby during delivery in that if asphyxiated it will usually be hypotonic and therefore be unable to resist stretch. It is the purpose of this paper to draw attention to the likelihood of spinal injury to the asphyxiated baby during delivery.

Case report

After a 41-week, normal pregnancy, a primigravid mother was admitted to hospital with breech presentation. Labour was induced by anterior rupture of the membranes. Fetal heart rate during labour was 120-150/min. About 3 hours after the onset of labour the cord prolapsed. As the cord was nonpulsatile and the liquor was meconium stained, the mother was delivered by breech extraction, forceps being applied to the after-coming head. A male infant was born, limp, cyanosed,

and apnoeic weighing 2.7 kg. He was resuscitated by intubation and intermittent positive pressure ventilation. Later the baby was found to be neurologically abnormal and to have a chronically distended bladder. The neurological abnormalities were hypotonia, absence of Moro reflex, sluggish limb movements (especially lower limb movements), and a patulous anus. X-ray of the spine revealed no bony defect.

Aged one week the baby weighed 2.7 kg and his head circumference was 35.0 cm. He had a high-pitched cry but sucked normally and turned towards diffuse light. Spontaneous movements were present in the upper but not in the lower limbs, and the trunk and lower limbs were conspicuously hypotonic. The traction, grasp, crossed extensor, asymmetric tonic neck, and Moro reflexes were absent. His tendon and abdominal reflexes were sluggish, his cremasteric reflexes normal, and his plantar responses extensor. Urine was passed in dribbles and the bladder was distended. Clonic fits occurred in the following week and were controlled with phenobarbitone.

At 3 weeks the infant had some flexor tone in his upper limbs, fed well, and often gazed steadily at the person feeding him. A cystogram showed a slightly trabeculated bladder and a urinary infection with *Esch. coli* was treated with trimethoprim and sulphamethoxazole (Septrin).

When about 4 weeks old he was discharged from hospital. 4 weeks later he was readmitted with severe hypothermia and died the same day.

Necropsy findings were spinal cord atrophy involving about 2.5 cm in the midcervical region with thickened adherent dura mater, a small subdural haematoma in the right temporo-parietal area, and moderate haemorrhagic cystitis.

Discussion

The spinal cord, blood vessels, and dura mater are protected by the vertebral column, ligaments, and muscles, and can normally withstand the stresses imposed during labour and delivery. When muscle tone is inadequate, the ligaments may permit the vertebral column to be unduly stretched and flexed with elongation of the spinal cord, blood vessels, and dura mater. These structures could therefore be compressed and torn without associated bony injury. In several early reports of spinal injury in the newborn infant (Burr, 1920; Crothers, 1923; Ford, 1925; Crothers and Putnam, 1927) emphasis was given to the mechanical effects of breech delivery but not to the state of the baby during delivery. It is possible that a baby asphyxiated *in utero* runs a higher risk of sustaining a spinal injury because it is hypotonic.

Yates (1959) reported spinal injuries in 27 of 60 unselected perinatal deaths. The affected infants were born by breech delivery, normal delivery, or caesarean section. There were extradural and subdural haemorrhages and haemorrhages into

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 of the vertebral arteries, and occlusion of a vertebral artery by thrombus. Such damage to vertebral arteries could impair circulation to the brain stem and cerebellum. Towbin (1969) found spinal cord and brain stem injury in 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