

defined primary deficit of their immunological mechanisms, the other 3 cases were markedly dystrophic infants subjected to protracted chemotherapy. These last 3 cases may also have had an underlying but ill-defined impaired resistance; alternatively, however, the pneumocystis carinii pneumonia could have been the primary disease, which initiated a vicious circle in which both pathological processes were involved.

The main pathology of pneumocystis carinii infection and the parasites were limited to the lungs in all 6 cases, despite the generalized character of the underlying condition which predisposed to the disease.

Neonatal Meningitis and Pneumonia due to Lancefield Group B Streptococci. K. B. ROGERS (The Children's Hospital, Birmingham). 4 newborn infants with pneumonia and 3 with meningitis were all infected with Lancefield Group B streptococci. Three of the infants with pneumonia were stillborn and the fourth only lived 18 hours: all four showed histological evidence of pneumonia in the lungs obtained at necropsy. Lancefield Group B streptococci were grown from vaginal swabs of all the mothers.

One of the 3 infants with meningitis was admitted at the age of 1 day, and a vaginal swab from this baby's mother grew Lancefield Group B streptococci: the other 2 were admitted at the ages of 10 and 11 days. All 3 only survived for a day after the onset of meningitis. Group B streptococci were isolated from the umbilicus of 2 of the 3 babies. Necropsy showed that 2 of the 3 babies had pneumonia as well as meningitis.

Group B streptococci were grown from 7 other babies; 2 were stillborn and the other 5 died a few days after birth. The streptococci were isolated from the throat of a 1-day-old infant on admission to hospital, from the lungs of 5 babies at necropsy, and from a cut-down wound in the seventh baby. Vaginal swabs from 3 of these babies' mothers grew Group B streptococci.

It is suggested that the Lancefield Group B streptococci were commensals in the mother's vagina, and that the infants were infected from this source: if vaginal swabs had been taken during pregnancy the streptococci could have been eliminated before the onset of labour. So many of these babies had been infected during or before labour that swabs taken when the mother was admitted for delivery would diagnose vaginal carriage too late for effective chemotherapy.

Birth Trauma and the Cervical Spine. E. LYNN JONES (Birmingham). The findings of Yates (1959) that distortional trauma to the cervical spine can occur at birth, and result in damage to the cervical portions of the vertebral artery was investigated further in a series of perinatal deaths.

The cases were selected during 1967 from 78 stillbirths and 114 neonatal deaths amongst a total of 320 necropsies performed at the Birmingham Children's Hospital.

The intact cervical spine was removed from 30 fetuses selected at random, fixed in 4% formaldehyde

in saline, and decalcified in 25% formic acid. The specimens were divided in horizontal planes and double-embedded in paraffin-wax and 2% celoidin. 5 μ thick sections cut on a sledge microtome were stained by a wide variety of methods.

Evidence of distortional trauma to the cervical spine was seen in 25 cases. The lesions were considered under 4 main groups: (1) extradural, dural, subdural, and subarachnoid haemorrhage; (2) tears and haemorrhages in nerve roots and spinal ganglia; (3) in 19 cases evidence of haemorrhage around one or both of the vertebral arteries was seen either in the form of a crescentic adventitial haematoma or massive haemorrhage encircling the vessel; (4) spinal cord lesions: these were seen in 2 cases, a precipitate and a breech delivery, and consisted of contusion in one case and bilateral necrosis of the lateral columns in the other.

The vertebral artery haemorrhages found in 19 fetuses may be important causes of perinatal mortality and morbidity. Many recorded cases of cerebral palsy could be explained on the basis of vertebral artery trauma and ischaemic cerebral damage at birth.

REFERENCES

- Yates, P. O. (1959). Birth trauma to the vertebral arteries. *Archives of Disease in Childhood*, 34, 436.

Intrauterine Causes of Neonatal Asphyxia. K. F. KLOOS (Berlin). The gross and histological study of the placenta is essential not only in the investigation of stillbirths, but also of neonatal deaths (and infantile disorders). Disturbance of implantation generally leads to abnormal shape of the placenta or to abnormal nidation: this can occasionally be the cause of fetal death and/or of maternal complications.

Of greater importance—and so far very little appreciated—are disorders of maturation, affecting the placental vascularity and hence the exchange of gases and metabolites. The fetus is especially vulnerable during labour and even a relatively mild degree of hypoxia can lead to damage to the central nervous system, depression of regulatory function, and failure of spontaneous respiration on delivery. This in turn can start a vicious circle ending in metabolic acidosis.

Collapse of the Trachea—a Possible Cause of Asphyxia and Death? S. RANSTROM (Gothenberg). It seems probable that a preparatory dilatation of the trachea with air precedes the start of respiration after delivery, and that collapse of the trachea may be an obstacle to this. Such a collapse may be caused by foreign material in the laryngeal aditus, or by anomalies of the trachea such as hypoplasia with defective thickness or elasticity of the cartilage, a broad membranous part of the tracheal wall, or doubling of the posterior wall, the edges of the cartilage bows lapping over each other. Collapse of the trachea in the newborn may be maintained by respiratory effort, the walls of the trachea being held together more tightly the greater the strength of the inspiratory movement. During expiration there is no material in the respiratory tract capable of dilating the trachea.