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% celloidin. 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.

**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.

**Paediatric Pathology Society** 147
Collapse of the trachea may be suspected when the newborn child makes a series of respiratory movements without effect. Adequate treatment is, of course, to induce air flow into the trachea by pulling the tongue forwards, by mouth-to-mouth breathing, or by intubation.

A Case of Congenital Lymphoid Hyperplasia. W. L. DONOHOE (Toronto).

Polycystic Disease of the Liver and Kidneys in Childhood. B. G. OCKENDEN and H. BLYTH (The Hospital for Sick Children, Great Ormond Street, London). A combined genetic and morbid anatomical study has been made of children with cystic malformation of both renal tubules and hepatic bile-ducts (polycystic disease of liver and kidney). 27 families have been studied in which the diagnosis has been confirmed histologically. Dominant inheritance is likely in 1 family and 6 others fall into this group on histological grounds. In 20 families an autosomal recessive pattern of inheritance is indicated. On clinico-pathological grounds the latter fall into four contiguous subgroups. Within individual families the type of disorder appears to breed true, therefore these subgroups are likely to be due to different recessive genes.

Relationship in Anencephaly between the Size of the Adrenal Gland and Length of Gestation. K. M. LAURENCE, A. B. N. ANDERSON, and A. C. TURNBULL (Welsh National School of Medicine, Cardiff). Evidence in the human fetus that the pituitary-adrenal axis is concerned in regulating the onset of labour may be obtained indirectly from observations in anencephaly.

A retrospective study was made of the 8 cases of anencephaly delivered at the Cardiff Maternity Hospital between 1959 and 1967, which were not complicated by hydramnios, and where the length of gestation was not in doubt and labour started spontaneously or was induced more than 4 weeks past term. In only one case did labour begin before term and in 5 of the remainder gestation was prolonged. In the 2 with the longest gestations, even to induce labour was difficult. Adrenal weights varied from 2-4 g. in the one with the shortest gestation, by even progression to 0-2 g. in the one with the longest gestation. The fetal (X) zone in the former was of almost normal dimensions and appearance. In the smaller glands there was a progressive diminution in the size of the zone. A histologically normal though small anterior pituitary gland was identified in all but one case; no posterior pituitary gland or hypophysis was found in any case.

A varying anterior pituitary stimulus is implied by the size of the adrenal, and it is suggested that a low pituitary-adrenal activity is associated with prolonged pregnancy and that the fetus to some extent determines the time of onset of labour.

Adrenal Hypoplasia during the Perinatal Period. C. B. F. DAAMEN (Rotterdam). In 88% of our 61 cases of anencephaly (2.9% of our material) there was coexistent hypoplasia of the adrenals, with more pronounced decrease of the fetal cortex.

Hypoplasia of the adrenals may be less pronounced in anencephaly before the 28th week of pregnancy.

In disorders of the whole primitive brain as well as in disorders of the distal part of the primitive brain there exists a preponderance of females. In disorders of the anterior part of the primitive brain there exists a preponderance of males (Hamersma, 1966). In anencephaly postmaturity is frequent (18%). During birth mortality is high. Congenital hypoplasia of the adrenals without developmental disorders in the primitive brain was present in 2-5% of our perinatal mortality cases (2739 cases). In these the histological structure of the adrenals was similar to that found in anencephaly.

There was a preponderance of males and postmaturity. Advanced maternal age was frequent. All our 7 cases of 'isolated' adrenal hypoplasia died on the first day of life, and cyanosis was an obvious clinical feature.

References


Functional Activity of the Fetal and Neonatal Rat Pituitary and Adrenal Cortex. A. SABBERG (Department of Pathology, University of Leiden, Netherlands). Making use of the organ culture technique, three groups of experiments were performed. Anterior pituitaries of 1-week-old rats were cultured for a period of 3 weeks and then transplanted to the sella turcica and under the renal capsule of 6-month-old hypophysectomized rats. At the time of transplantation the cultured explants had lost their specific functional activity, and consisted of completely degranulated cells. Five months after the transplantation the surviving animals were killed. Sections were made of the sites of transplantation. The corticotrophic activity of the transplanted cultures was assessed by measuring the difference in weight between the left adrenal, removed 10 days before the animal was killed and the right adrenal at the time of death. In 4 of the 5 surviving animals vascularized masses of well-differentiated chromophil and chromophobe cells were found in the sella turcica. The implants under the capsule of the kidney consisted entirely of undifferentiated cells. 3 of the 4 animals with viable sella turcica implants showed compensatory hyperthrophy; such a hyperthrophy was not observed in rats with implants under the renal capsule. These experiments suggest that the anterior pituitary can only maintain its activity for a short time if there is no contact with the neural hypophysis and hypothalamic area. The activity is reinitiated if the contact is restored. The adrenal cortex reacts to the reinitiated activity.

Fragments of the adrenal cortex of young rats were cultured in contact with fragments of the anterior pituitary of newborn and fetal rats. The amount of corticosteroids released over a period of 3 days was measured. The results showed that 19-day-old fetal rat pituitaries can produce corticotrophic hormones.
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