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gross deformities are not compatible with the concept of developmental arrest. The malformation appears to be due to an increased caudal extension of the cerebellum entirely as a consequence of normal growth in the presence of an early hydrocephalus.


In a postmortem series of 3 small-for-dates infants the brains were examined by naked eye and by light microscope, and compared with controls. In 9 instances disorders of the corticovolitional pattern such as agryria, pachygyria, and microgyria were found. Minor convolutional anomalies occurred in a further 5 infants (and also in 2 of the controls). The principal microscopic lesions observed included failure of neuroblast migration—both in cerebrum and cerebellum—as well as poor stratification of the cerebral cortex with retarded differentiation of ganglion cells. The cerebellar cortex showed impaired regression of the embryonal granular layer, with the outer granular layer embedded in the inner granular layers, and absence or ectopia of Purkinje cells; the latter abnormality, however, is significant only after eight months of gestation.

These anomalies of the brain were correlated with disordered placental morphology. Fetal dysmaturity, in general, was found to be associated with disturbed early embryonal development of chorionic villi, which may be due to maternal hormonal imbalance or, alternatively, to failing immune tolerance between mother and fetus. In addition to qualitative inferiority of the placental villi, there may also be a quantitative defect, due either to smallness of the placenta or to circulatory damage, causing loss of functional villi.

Primary hepatic cancer in childhood. D. Sinniah, P. E. Campbell, and J. H. Colebatch. Department of Pathology, Royal Children's Hospital, Flemington Road, Parkville, Victoria 3052, Australia.

During the period January 1950 to June 1972, there were 2 children with primary malignant liver tumours at the Royal Children's Hospital, Melbourne, representing 0.08/1000 hospital admissions and 2% of all childhood malignancies (excluding leukaemia) seen at this institution. There were 16 hepatoblastomas and 4 hepatocellular carcinomas.

Hepatoblastomas. Age from 4 months to 10 years (75% under 3 years). The commonest presenting sign was abdominal distension and the most useful preoperative investigations were hepatic angiography and liver scan. There have been 4 long-term survivors following partial hepatectomy, chemotherapy (mostly mitomycin C and vincristine), and postoperative radiotherapy. Macroscopically, 11 tumours arose in the right lobe and 4 in the left. There were no cirrhotic livers. Histologically 13/16 had fetal cells, in 6 they were the dominant cell type and in another one the only cell type. 14/16 had embryonal cells, 3 being the dominant cell type. 4/16 had anaplastic cell areas, 1 being the dominant cell type. 2/16 had areas resembling 'adult' hepatocarcinoma. 2/16 consisted predominantly of rhabdomyoblastic cells. In 9/16 osteoid was found. In 3 cases the histological pattern was truly mixed with fetal embryonal and stromal components represented about equally.

Hepatocarcinomas. All were advanced when diagnosed and all 4 children died. Symptomatology was varied and in none was abdominal swelling the initial complaint. Macroscopically these tumours were multiple and variegated. One arose in a cirrhotic liver. Microscopically they appeared 'carcinomatous' with cords and trabeculae of cells separated by sinusoids.

CSF in acute childhood leukaemia: cytocentrifuge studies. D. I. K. Evans. Department of Pathology, The Royal Manchester Children's Hospital, Pendlebury, Manchester M27 1HA.

The cytocentrifuge enables a satisfactory cytopathological preparation to be made when the cells in the CSF are normal in number or only slightly increased. A technique has been developed and the results of its use are described in 114 consecutive samples from 50 children with acute leukaemia, with and without involvement of the CNS. Analysis of the results shows that 30% of the samples with a normal cell count contained leukaemic cells when examined by cytocentrifuge; and only 74% of the samples with a raised count were found to contain leukaemic cells. It also appears that changes in the levels of protein and glucose in the CSF of leukaemic patients are not directly related to the presence of leukaemic cells, but are the result of changes in the cell count from whatever cause.

Screening for abnormal haemoglobins in the immigrant community. D. N. Raine and J. M. Pepper. Department of Clinical Chemistry, The Children's Hospital, Birmingham B16 8ET.

Following the advice that those at risk for sickle cell disease should be examined before any procedure that might precipitate a sickle cell crisis is undertaken, a number of centres have introduced a variety of means whereby these patients can be tested. Experience with the Sclerometer method of screening neonates for amino acids, in which blood is collected in capillary tubes, led to the consideration of a similar approach to the haemoglobinopathies.

The system involves direct testing by starch gel electrophoresis of blood collected via school clinics. The method allows 100 specimens to be processed daily. Data obtained by other methods in other laboratories is also used. A punched card file and magnetic tapes store is maintained in which identification is recorded by surname in words and phonetic code, forename, day and month of birth, and National Health Service number (more than 70% of NHS numbers are being obtained). The method of analysis, centre of testing, date of specimen, and result are also recorded. Fortran programs (IBM 1440 computer) allow the card to be decoded and the data printed, a new subject to be matched against the data bank, the Soundex code to be