shown a shortage of postnatally dividing microneurons in the cerebellar granular layer and other neurones in the deeper layers of the cerebral cortex. The deficit is particularly surprising in view of the comparatively mild growth retardation imposed during the brain's vulnerable period of development. The comparable period of human brain growth extends from about 30 weeks of gestation into the second year of postnatal life. Behavioural correlates in the experimental animal were also discussed.

From mouse to man? R. D. Barnes (*Division of Infant Development, Clinical Research Centre, Northwick Park, Harrow*). The techniques of ovum transplantation and ovum fusion are described since these have been successfully used to investigate various diseases in mice. Ovum transplantation allows the transfer of one fertilized egg to an animal of another strain and is a very useful technique to investigate the role of transplacental infection in any disease. Secondly, ovum fusion derived chimaeras provide an excellent experimental model to investigate the interrelation between two cell populations in the same animal. Furthermore, in this respect ovum fusion allows us to investigate the hypothesis that certain diseases in mice, including autoimmune haemolytic anaemia and leukaemia, occur due to the time-related failure of what might be considered a normal recognition and inhibition control process, and evidence for this is presented.

Finally, the modification and further sophistication of *in utero* grafting techniques might, in the event of proving useful in curing certain defined diseases in mice, suggest that these techniques could subsequently apply to man. However, this must await full investigation in animals, and furthermore, the development of suitable techniques for antenatal diagnosis of affected children.

Coagulopathy in the hypoxic newborn baby. M. A. Chadd (*introduced by O. P. Gray*) (*Department of Child Health, Welsh National School of Medicine*). It is well established that coagulation defects and haemorrhage may result in death in the perinatal period.

Classical haemorrhagic disease as first described in the 1800s by Townsend is now of almost academic interest with the decline in breast feeding and the increased use of vitamin K in neonatal nurseries.

The concept of secondary haemorrhagic disease first proposed by Aballi and de Lamerens 10 years ago is recognized as becoming of increasing importance. It is frequently found in association with hypoxia and is unresponsive to vitamin K.

This study of 75 hypoxic newborn and 75 control infants was undertaken in an attempt to elucidate the nature and incidence of coagulation defects in the newborn and the role of hypoxia in producing such defects.

Brain oedema induced by asphyxia in newborn rats. S. W. D'Souza (*introduced by J. A. Davis*) (*Department of Child Health, University of Manchester*). Using 5-day-old rats an attempt has been made to

induce brain oedema experimentally with asphyxia as the precipitating insult.

Changes in brain water, sodium, and potassium content can be induced by slow prolonged asphyxia at body temperature and to a lesser extent in the cold, but not by acute anoxia. These changes are more marked in the brainstem than in the hemispheres.

Plasma growth hormone response to intravenous glucagon administration. L. Stimmler and G. Snodgrass (*Guy's Hospital, London*). Glucagon administration has been shown to stimulate growth hormone secretion. In a previous study sampling times were delayed, whereas it is known that glucagon produces a very rapid rise in plasma insulin.

Glucagon 15 µg/kg body weight was administered intravenously to a group of 24 children being investigated for abnormally short stature. Blood samples were obtained immediately before and at frequent intervals after glucagon administration. 20 of these children showed a marked rise in growth hormone levels. In 14 of these patients a significant rise had already occurred at 2 minutes after glucagon administration. The time of peak growth hormone response occurred before 20 minutes in 17 of these patients. Insulin and glucose levels were also estimated. The interrelation between these parameters and the growth hormone response was discussed.

Insulin release from human fetal pancreas in vitro. R. D. G. Milner and M. A. Ashworth (*Department of Child Health, University of Manchester*). Pieces of pancreas removed from dead human fetuses delivered by hysterotomy were incubated *in vitro* as described previously for rabbit pancreas. The gestational age of the fetuses studied was between 14 and 24 weeks and their body weights ranged from 50 to 625 g. Insulin released into the incubation medium was measured under basal conditions and in the presence of various substances known to stimulate insulin secretion in adult man and other species. Glucose (3.0 mg/ml) did not stimulate insulin release. Stimuli which are thought to act by raising intracellular levels of cyclic AMP: glucagon (5 µg/ml), theophylline (1 mM), and dibutylryl cyclic AMP (1 mM) stimulated insulin release in the presence of 0.6 or 3.0 mg/ml glucose or in its absence. Ionic stimuli which act late in the stimulus-secretion pathway were also uniformly effective: barium (2.54 mM), ouabain (10⁻⁵ M) and potassium (60 mM). Leucine (5 mM) and arginine (5 mM) were effective in some experiments only. Tolbutamide (400 µg/ml) was ineffective in two experiments.

It was concluded that cells capable of secreting insulin are present in human fetal pancreas from the 14th week of fetal life onwards.

Applications of praecordial accelerometry. D. Pickering (*introduced by B. D. Bower*) (*Department of Paediatrics, Radcliffe Infirmary, Oxford*). A praecordial accelerometer is described which has been