

MARGARET CHADD introduced by PROFESSOR O. P. GRAY (Cardiff). 'Disseminated Intravascular Coagulation and Coagulation Defects in the Newborn.' Haemorrhage into vital organs is a common necropsy finding in perinatal deaths. In a 6-year retrospective study at the Cardiff Maternity Hospital, we have found significant haemorrhage in 88 out of 192 consecutive necropsies studied. Coagulation defects have been found to be associated with some haemorrhages.

The cause of the coagulation abnormalities remains obscure, one possible factor leading to a consumption coagulopathy is disseminated intravascular coagulation.

Evidence is presented to demonstrate the role of disseminated intravascular coagulation in perinatal coagulation defects.

Using special stains, fibrin deposits have been found in various organs (liver, lungs, suprarenal, etc.) of 13 out of 40 consecutive necropsies. These deposits have been seen only in infants who have had severe acidosis, anoxia, and in  $\frac{2}{3}$  of the cases a rectal temperature of 34 °C. or less. Half the babies required artificial ventilation during life: 40% had very low levels of prothrombin, consistent with a consumption coagulopathy.

Functional tests of disseminated intravascular coagulopathy confirm that the condition occurs in the newborn.

The results of these tests namely platelet counts, thrombin clotting times, kaolin cephalin clotting times, and levels of fibrin degradation products will be presented. The fibrin degradation products indicate that the abnormalities can be attributed to disseminated intravascular coagulation.

A three-day study of the coagulation status of 124 consecutive admissions to the Glossop Special Care Unit has shown that acidosis and anoxia appear to be major contributory factors in the initiation of disseminated intravascular coagulation in the perinatal period.

M. J. MACCULLOCH introduced by DR. MARGARET GRIFFITHS (Birmingham). 'In Search of a Cause of Infantile Autism.' The paper presents a brief review of current concepts of infantile autism and suggests that insufficient attention has been paid to the bizarre motor movements which in part characterize this condition or group of conditions. It is suggested that some dysfunction of afferent neurone pathways would explain the psychiatric and psychological findings in the literature. A single site where auditory, vestibular, and somatic afferent pathways are in juxtaposition is in the dorsal part of the brain-stem, in the area immediately ventral to the floor of the fourth ventricle. A lesion in this area will be expected to affect the modulation of heart rate. Experimental data are presented to show that heart rate variation taken by remote radio-telemetry in 19 autistic children is significantly higher than in a group of 10 normal children and 9 non-autistic subnormal children. Data are also presented on these groups, which indicate that ocular strabismus is significantly more common in the autistic group.

In the theoretical discussion it is pointed out that oculo-motor nuclei connections are specially related to

the cardio-regulatory centre. Some neuro-physiological findings in the cat indicate that the nucleus of the tractus solitarius regulates parasympathetic outflow, as well as reticular formation activity and cortical arousal.

The data presented together with the findings of differing disciplines support a cohesive theory which states that children with the infantile autism syndrome are suffering from neuronal damage in the dorsal brain-stem.

J. M. TANNER, H. GOLDSTEIN, and R. H. WHITEHOUSE (London). 'Standards for Children's Height at Age 2 to 9 years allowing for height of Parents. *Archives of Disease in Childhood*, 45, 755.

C. G. D. BROOK introduced by DR. JUNE K. LLOYD (London). 'Adipose Cell Size and Number in Obese Children.' Measurements of the size and number of adipose cells have been made in 34 obese children and in 40 children of normal weight. Cell size has been determined by the weight of lipid per cell (Hirsch and Gallian, 1968), and the total number of adipose cells in the body has been calculated after estimation of the adipose tissue mass. The latter has been derived from skinfold measurements (Durnin and Rahaman, 1967) in all subjects and in addition by estimations of total body water in the obese children.

In normal children the number of adipose cells increases with age; the mean cell number for adults is  $27 \times 10^9 \pm 9 \times 10^9$  (1 SD). The majority of obese children had an increased number of cells and some of the youngest already had the normal adult complement. The cell lipid content did not vary with age in normal children ( $0.28 \mu\text{g.} \pm 0.11$ ); the cells of the obese children were significantly larger ( $0.59 \mu\text{g.} \pm 0.21$ ). The adipose tissue triglyceride of obese children contained an increased percentage of oleic acid ( $50.4\% \pm 2.7$ , normal  $46.6\% \pm 2.9$ ) and palmitoleic acid ( $9.8\% \pm 1.9$ , normal  $6.5\% \pm 1.9$ ), probably due to increased lipogenesis from carbohydrate.

In 15 grossly obese children oral glucose loads resulted in very high levels of plasma insulin (up to  $250 \mu\text{U./ml.}$  at 30 or 60 minutes), indicating relative insensitivity of the large adipose cell to insulin. After a period of weight loss, there was a reduction in cell size in the majority of children, but no change in cell number; the degree of hyperinsulinaemia after oral glucose was reduced in those children in whom cell size was shown to be reduced, but unchanged in the others.

These observations show that obese children have an increase both in the size and number of their adipose cells. The tendency for childhood obesity to relapse may be related to the persistence of the large number of adipose cells despite treatment.

#### REFERENCES

- Durnin, J. V. G. A., and Rahaman, M. M. (1967). The assessment of the amount of fat in the human body from measurement of skinfold thickness. *British Journal of Nutrition*, 21, 681.  
 Hirsch, J., and Gallian, E. (1968). Method for the determination of adipose cell size in man and animals. *Journal of Lipid Research*, 9, 110.