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 $\frac{3}{4}$ 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 brainstem.

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 ($0.28 \mu g \pm 0.11$); the cells of the obese children were significantly larger ($0.59 \mu g \pm 0.21$). 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 $\mu$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


British Paediatric Association

ELIZABETH PRINGLE introduced by DR. MARY WILMERS (London). ‘A Study of 112 Children with Coeliac Disease from 1950–69.’ (To be published elsewhere.)

E. ROSSIPAL (Graz). ‘Precipitins to Aqueous Extracts of Flour in Coeliac Disease.’ In certain instances it may be difficult to differentiate between coeliac disease and the ‘coeliac syndrome’. An essential diagnostic criterion in coeliac disease is the disappearance of symptoms on a gluten-free diet, and a relapse caused by reintroduction of gluten into the diet. Normally a long period of observation is necessary to confirm the diagnosis in this way. We have been able to develop a sensitive method of detecting precipitins to aqueous and alcoholic extracts of flour from wheat, barley, oats, and rye in the serum of patients with coeliac disease in 15 patients. The diagnosis of coeliac disease was confirmed by disappearance of the symptoms on a gluten-free diet and by a relapse after reintroduction of gluten into the diet. All of these patients had precipitating antibodies to aqueous extracts of wheat and barley; 13 also showed precipitins to extracts of oats, and 10 to extracts of rye. It was proved that the precipitating antibodies to the aqueous extracts of flour belonged to the IgG group. Sera of 60 children suffering from parenteral diarrhoea, *Esch. coli* enteritis, disaccharide intolerance, and mucoviscidosis, and pooled serum of 100 healthy adult blood donors all served as controls. In none of these controls could precipitins to aqueous extracts of the cereals be detected. The determination of these antibodies seems to be of value as a diagnostic tool in coeliac disease and could help to unravel the immunological mystery of this disease.

R. A. RISDON introduced by PROFESSOR A. E. CLAIREAUX (London). ‘Renal Dysplasia—A Clinico-pathological Study of 76 Cases.’ (To be published.)

J. MARTIN and P. P. RICKHAM (Liverpool). ‘Pulmonary Metastases in Wilms’ Tumour—Treatment and Prognosis.’ (Archives of Disease in Childhood 45, 805.)


J. A. DUDGEON (London). ‘Prevention of Congenital Rubella Defects. The Need for Long-term Surveillance.’ Several attenuated rubella vaccines have now been developed. Two have already been licensed abroad and one is likely to be licensed in Great Britain early in 1970.

At the autumn meeting of the BPA we presented data of our comparative trials with three of the attenuated vaccines, and outlined a programme for immunization against rubella in schoolgirls aged 10–14 years in the same year tuberculin testing and BCG vaccination are carried out.

The results of vaccine trials now being undertaken on approximately 1,000 schoolgirls will be presented. If rubella vaccine is used on a sufficiently wide scale in the next few years, there should be a decline in the incidence of rubella defects. These can now be fairly readily identified in the first year of life on clinical grounds, and laboratory tests, such as virus isolation, serology, and immunoglobulin levels can provide confirmatory evidence. Defects such as perceptive deafness not recognizable at birth will in many instances be picked up at a school medical examination. A plan for long-term surveillance will be outlined in which it is hoped paediatricians, Medical Officers of Health, and School Medical Officers will co-operate.

L. HOHENAUER (Innsbruck). ‘Calcium and Phosphorus Homeostasis during the First Days of Life.’ (To be published.)

G. J. I. SNODGRASS introduced by DR. L. STIMMLER (London). ‘Interrelation of Plasma Magnesium, Calcium, and Phosphate for 4 milk Regimens.’ (To be published.)

O. THALHAMMER (Wien). ‘Histidinaemia Detected by Newborn Screening using Guthrie’s Method.’ In 36,462 routine Guthrie tests on newborn infants, 3 cases of histidinaemia have been discovered. Initial tests on day 4, 5, and 6 were positive corresponding to 16, 6, and 6 mg./100 ml. In the same series 62 so-called false positive initial tests were in the same range. There was an excellent concordance between the results of inhibition assay and column chromatography. Histidine blood levels were constantly high in all three infants and histidase activity in stratum corneum was zero. Excretion of histidine in the urine was constantly high, but in spite of this, the ferric chloride test was positive in only two of the three infants. Excretion tests urinary histidine output was 27%–47% of loading dose and formiminoglutaric acid was absent in all samples during 24 hours after loading. Blood levels did not return to starting values less than 24 hours after loading. Histidine-low diet brought histidine blood levels and urine excretion to normal within 1 to 2 weeks.

A Seminar on The Paediatrics of Immigration was held on Saturday morning, 25 April, in the Royal Hotel, Scarborough, with Dr. J. W. Farquhar as Chairman, and introduced by Professor T. E. Oppé. The following speakers took part: Dr. Catriona Hood (London), by invitation, ‘Social and Cultural Factors in the Health of Children of Immigrants.’ Dr. G. S. Prince (London), by invitation, ‘Emotional Deprivation and Pseudo-autism.’ Dr. M. W. Arthurton (Bradford)‘Immigrant Children and the Day-to-Day Work of a Paediatrician.’ Professor C. E. Stroud (London)‘Some Important Organic Problems.’

WINDERMERE LECTURE. The Lecture was delivered on 23 April by Professor C. Henry Kempe (University of Colorado Medical Center)‘Paediatric Implications of the Battered Child Syndrome.’ (To be published in Archives in Disease in Childhood, 1971.)

Members and guests of the Association attended a
Adipose cell size and number in obese children.

C G Brook and J K Lloyd

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