Olive Scott and F. J. Macartney. Killingbeck Hospital, Leeds. 'The sick sinus syndrome'. This syndrome is probably a commoner cause of syncopal dizzy attacks, convulsions, or sudden death in childhood than previously recognized. The symptoms may be induced by exercise. The first manifestation of the condition is usually sinus bradycardia. This may progress to sinus arrest (when depolarization of the sinus node fails to occur) or to sinoatrial block (when the sinus node discharge is not propagated to the surrounding atrium). This results in cardiac arrest unless another pacemaker takes over. This new pacemaker is usually in the atroventricular node and the rhythm produced by it may be abnormally rapid or slow. If it is inappropriately slow, giddiness and syncope result.

Clinical examination between attacks is normal except for bradycardia. An electrocardiogram may show abnormalities of the 'p' waves which may change their shape or direction during the recording (suggesting a new focus for the heart beat). If cardiac arrest for 2 or 3 beats is recorded then the sick sinus syndrome is more certain.

Since recognizing our first case one year ago, 3 other cases have been diagnosed. Details of our 4 patients were given and the problems of management by medical means or the introduction of a cardiac pacemaker (which may be life-saving) were discussed.

J. R. Graham Pole. Royal Hospital for Sick Children, Glasgow. 'Use of a continuous flow cell separator in paediatrics'. The cell separator was designed to treat leukaemic patients by leucopheresis. Similar techniques can be used to collect normal leucocytes in health and abnormal plasma constituents in disease states.

1) Leukaemic cells were collected from 3 patients with white counts greater than 100,000/mm³. 2 had evidence of leucostasis and CNS involvement. Up to $2 \times 10^{11}$ cells were collected into liquid nitrogen, and have retained their viability indefinitely. (2) 11 granulocyte transfusions were given to patients with profound neutropenia; 3 from chronic myeloid leukaemic donors by standard leucopheresis, and 8 from normal parental donors by continuous flow. A mean of $2.07 \times 10^{10}$ leucocytes were collected from normal donors, comparing well with chronic myeloid leukaemic donors, and producing mean polymorph increments of 830/mm³ in recipients. Adding Dextran-110 markedly increased the leucocyte yield. Patients with proved or strongly suspected septicemia had lower increments than those with profound neutropenia but less evidence of sepsis, suggesting rapid utilization of infused polymorphs. The cells were shown to be viable and phagocytic up to 24 hours after harvesting, but the total tended to fall with increasing collection/infusion interval. (3) Replacement of missing enzymes and excretion of accumulated material in inborn metabolic errors was achieved with infused leucocytes in mucopolysaccharidosis and metachromatic leucodystrophy, showing transfer of a missing factor to defective cells. (4) Exchange plasmapheresis in 3 patients with severe rhesus isoimmunization markedly reduced the plasma anti-D antibody early in pregnancy, in one case from 55-7 μg/ml to 4-2 μg/ml over 28 plasmaphereses.

Further application of these techniques are under trial.

S. Packer, R. J. Rowlett, and J. T. Harries. The Hospital for Sick Children, Great Ormond Street, London. 'Reappraisal of a past diagnosis of "coeliac disease"'. In true coeliac disease intolerance to dietary gluten is permanent, 1 in 10 first-degree relatives are affected, and there may be an increased risk of neoplasia. The implications to the child and its family are major and the application of strict diagnostic criteria by means of intestinal biopsy is essential.

The results of a study designed to reassess a past diagnosis of 'coeliac disease' in 44 patients aged 4–16 years were presented. The patients fell into 1 of 3 groups. Group 1 had received a gluten-free diet (GFD) for 1–13 (mean 6) years with no past biopsy (19), or past biopsy considered uncharacteristic on review (5); group 2 had received a normal diet (ND) for 1–6 (mean 2½) years after a GFD for 1–5½ (mean 2½) years with no past biopsy (12), or uncharacteristic biopsy on review (2); group 3 ND for 2½–9½ (mean 6) years after GFD for 2–5½ (mean 3½) years with past biopsy characteristic (6).

Biopsies were performed in group 1 before and after gluten challenge (<10 g wheat protein/day for 3 months). Challenge induced mucosal abnormalities in 18 (75%), and of these only 7 (39%) developed symptoms. 5 (38%) patients in group 2 were proven to have coeliac disease. 3 (50%) patients in group 3 had normal biopsies (? transient gluten intolerance), and the remaining 3 patients had flat biopsies despite normal growth and RBC folate levels.

These results illustrate the importance of applying strict diagnostic criteria to patients with suspected coeliac disease, and the necessity of reassessing a past diagnosis of 'coeliac disease'.

M. C. O. Bax and T. K. Whitmore. Guy's Hospital, and Department of Education and Science, London. 'Health and welfare of London schoolchildren'. All entrants to 5 infant schools in the London Borough of Deptford during the school year 1971–72 were examined at a special school entrant medical. Comparisons were made with a special group of 100 children examined at the same time on the Isle of Wight. Specially trained school nurses administered a parental questionnaire which collected data about behavioural and social aspects of the child's life as well as medical and developmental data. Most of the London children had in fact been born in or near the borough where they were now living, but in contrast their parents were born in many countries of the world. The data indicate that in terms of physical growth, by 5 years these new Londoners were comparable to the Isle of Wight children, but in many other aspects of their development they lagged far behind. There are important implications for the development of child health and community paediatric services.
Proceedings: Reappraisal of a past diagnosis of "coeliac disease".
S Packer, R J Rowlatt and J T Harries

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