

PROCEEDINGS OF THE NUTRITION SOCIETY

ONE-HUNDREDTH SCIENTIFIC MEETING
PHYSIOLOGY LECTURE THEATRE, THE LONDON HOSPITAL
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DIET AND ANAEMIA

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Chairman's opening remarks

By L. S. P. DAVIDSON, *Department of Medicine, University of Edinburgh*

I should like to say how much I appreciate the honour of being asked to preside at this one-hundredth meeting of The Nutrition Society and I should also like to thank the Council for inviting me to be the guest of the Society at dinner to-night.

This meeting is an important landmark in the history of the Society, and one which is being celebrated by a symposium on the anaemias, which should be of the greatest interest to all the members.

In the few minutes allotted to me for introductory remarks I shall give some of my impressions of the anaemias due to nutritional deficiencies as encountered in Edinburgh at the present time and as I saw them 36 years ago, when I became Assistant to Professor Lovell Gulland, Professor of Medicine in the University of Edinburgh, who was then the leading clinical haematologist in Great Britain.

The two main groups of anaemias due to deficiencies of food factors are the hypochromic anaemias, due to deficiency of iron, and the megaloblastic anaemias, due to deficiency of vitamin B₁₂ or folic acid.

It is still true to say that the iron-deficiency anaemias are probably a thousand times as common in Great Britain as are the megaloblastic anaemias, and constitute, therefore, an infinitely more important social and economic problem even though they may not be so exciting from the academic point of view. Some 20 years ago in Aberdeen Dr Fullerton and I constructed a graph based on the haemoglobin levels of more than 2000 unselected persons of both sexes and of all ages from infancy upwards. This graph clearly indicated the liability of certain groups of people to iron-deficiency anaemias. The point I would emphasize is that hypochromic anaemia is particularly likely to develop in infants and young children between the ages of 6 and 24 months, and among women of the child-bearing age, and this is as true to-day as it was 20 years ago. The main differences that I notice between 1956 and 1930 are that the iron-deficiency anaemias, although still commonly encountered, are not so prevalent or so severe as in 1930; also, the striking features of acute glossitis, koilonychia and the Plummer-Vinson syndrome are much less frequently

seen and are of lesser degree at the present time than 25 years ago. The four main reasons for this reduction in the incidence and severity of the nutritional hypochromic anaemias are, firstly, the education of the medical profession in regard to the frequency and pathogenesis of the disease and the appropriate measures for its prevention and treatment; secondly, the formation, immediately before World War II, of child-welfare clinics where these measures could be put into operation, and the provision at the present time of haematological centres at which the general practitioner can get blood examinations undertaken; thirdly, the social revolution that has occurred during the last 20 years which has resulted in the redistribution of the nation's wealth, so that the working classes of 1956 can afford a much better diet with a higher iron content than could their predecessors of 1930; and, fourthly, in the last 10 years women and children have received free medical attention under the National Health Service, whereas formerly only the insured worker got free treatment, so the women and children had to pay the doctor and when poor tended to delay consulting him as long as possible.

With the megaloblastic anaemias in Great Britain the position is entirely different. The number of persons suffering from megaloblastic anaemia at the present time is, I think, considerably higher than it was when I was working with Professor Gulland 36 years ago. The reasons for this higher incidence are also obvious. Firstly, the number of people between the ages of 60 and 80 in Great Britain is much higher now than it was 36 years ago, and therefore the number of people who are liable to develop Addisonian pernicious anaemia is correspondingly greater; secondly, the introduction of liver extract and, later, vitamin B₁₂ has enabled patients suffering from Addisonian pernicious anaemia to have a normal expectation of life, and so the total population of patients with pernicious anaemia is greater than ever before; thirdly, because of our increased knowledge of the pathogenesis of the megaloblastic anaemias due to malabsorption, and our increased interest in, and recognition of, the megaloblastic anaemias of pregnancy, many more of these patients are diagnosed and successfully treated to-day than 36 years ago. For all these reasons there is little doubt that there are more patients with megaloblastic anaemia alive in Britain at the present time than ever before.

The description of pernicious anaemia given in many textbooks of medicine is largely based on the clinical features encountered in patients dying of the disease before 1926. At the present time the disease is diagnosed much earlier than formerly, hence the features of clinical jaundice, splenomegaly, acute glossitis and subacute combined degeneration of the cord, which were held to be of such diagnostic importance before liver therapy was introduced, are found in less than 5% of the patients referred to the Blood Clinic in Edinburgh at the present time.

Another point I wish to draw attention to is that the great majority of megaloblastic anaemias in Great Britain are not due to direct dietary deficiency of vitamin B₁₂ or folic acid. They are due to a conditioned or indirect deficiency due in Addisonian pernicious anaemia to malabsorption owing to lack of intrinsic factor, or in the malabsorption syndrome to mechanical, organic or functional defects in the small intestine. We are still at a loss to know why about one pregnant woman out of every

1000 develops megaloblastic anaemia. Often the cause does not seem to be due to direct dietary deficiency of folic acid. A survey of some 855 cases of macrocytic anaemia seen in our clinic in Edinburgh during the period 1940-54 indicates four points of special interest. Firstly, it does not follow that because a patient has a macrocytic anaemia, as judged by a high colour index and a high mean cell volume, he necessarily has a megaloblastic anaemia. Secondly, it is apparent that in Edinburgh if a patient has a macrocytic anaemia it is six chances to one that he has a megaloblastic anaemia. Thirdly, if he has a megaloblastic anaemia it is nearly six chances to one that he is suffering from Addisonian pernicious anaemia. Fourthly, excluding Addisonian pernicious anaemia, the only two forms of megaloblastic anaemia of numerical importance are those due to malabsorption and to pregnancy and the puerperium, unlike the megaloblastic anaemias in Africa and in the Far East, which are frequently due to direct nutritional deficiency.

There are few physicians in Great Britain, in active practice in hospitals at the present time, who can say, like myself, that they were in charge of patients suffering from Addisonian pernicious anaemia for a period of 6 years before liver therapy was discovered. It is only those who personally watched the invariably tragic and fatal course of this disease who can appreciate the amazing results produced to-day by the injection of a few micrograms of cyanocobalamin. It is no exaggeration to say that the research work undertaken during the last 25 years, which has led to the unravelling of so many mysteries of the megaloblastic anaemias and which has produced such potent remedies as cyanocobalamin and folic acid, constitutes one of the greatest triumphs of science over disease.

It is my pleasure to declare the one-hundredth meeting of The Nutrition Society open.

Diet and anaemia: zymotic and other factors

By B. S. PLATT, *Human Nutrition Research Unit, Medical Research Council Laboratories, Holly Hill, London, N.W.3* and G. R. WADSWORTH, *London School of Hygiene and Tropical Medicine*

By anaemia we mean a reduction in the amount of total circulating haemoglobin (Wintrobe, 1954); this definition is preferred to that which defines anaemia in terms of a lowered concentration of haemoglobin in the blood which may be unimpressive owing to changes in plasma volume (Allington & Taylor, 1955; Hope & Verel, 1955). The main part of this paper will be concerned with the role of protein in maintaining normal amounts of haemoglobin in the blood.

In the space available it is not possible to discuss such aspects as the interrelationship of protein and hormone synthesis, the new work on a humoral factor in the regulation of haematopoiesis (Van Dyke, Contopoulos, Williams, Simpson, Lawrence & Evans, 1954), and the similarity of the characteristics of some tropical anaemias variously attributed to infections and infestations and protein malnutrition (Trowell,