SESSION II

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OPENING ADDRESS

Current status of childhood lead poisoning

LEAD poisoning is now essentially a disease of childhood. It must be seen in both historical perspective and in the context of adult poisoning. Unlike the adult problem, childhood poisoning has been recognized comparatively recently, and apart from the isolated but massive exposures of entire populations in previous centuries, it is only during the last 30 years that it has been diagnosed with any frequency. Thus, at the beginning of this century there were about 1000 cases of adult poisoning from occupational sources alone each year with seventy deaths, whereas at the present time there are about seventy cases per annum reported and the last adult death was 18 years ago. Since lead poisoning is not a notifiable disease in childhood actual figures are difficult to obtain. However, in the United Kingdom it is thought that there are about 100 admissions of children to hospital each year with a diagnosis of lead poisoning and about two deaths per annum. There is every reason to suppose that these figures, which contrast markedly with those in the U.S.A., underestimate the true position. It is difficult to understand why this entirely preventable disease persists in childhood in 1974 when the adult form has virtually been eliminated. I propose therefore to consider the diagnostic problems involved and to examine the measures currently available for prevention, and to suggest improvements in our current approach.

The clinical recognition of childhood plumbism is not easy. Children do not manifest many of the classical features of the adult disease—they do not suffer peripheral neuropathy, they do not show blue lines at the gingival margin, they may not be anaemic, they do not frequently complain of colic, and they may not even be constipated. The signs and symptoms are thus few and non-specific. Although the classical grades of response to intoxication occur,

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nevertheless the child may first present to the physician in the terminal stage of encephalopathy.

Knowledge of the children most at risk and of the predisposing factors would do much to improve clinical recognition at an early stage. Typically the symptoms become evident in the summer months, the peak incidence is at the age of 2 years with a range of 1-5, the child inhabits an old, poorly maintained home and shows the phenomenon of 'pica', the propensity for the ingestion of non-food substances, if applied to the population at large, would encompass a very large number of children and reliance must therefore be placed on laboratory tests in order to confirm or refute the diagnosis.

Laboratory tests fall into two major groups: (a) primary tests—detection of lead in the tissues; (b) secondary tests—detection of the effects of lead in the tissues.

The latter have enjoyed considerable application during the last few years, especially those concerned with disturbances of porphyrin metabolism. During the last 20 years these have included the determination of urine coproporphyrin, urine aminolaevulinic acid and, more recently, free erythrocyte protoporphyrin. However, they all suffer disadvantages—they are non specific, they are not sufficiently well correlated with blood lead concentration, and they all require confirmation by one of the primary procedures. Their application to diagnosis (as opposed to screening) tends to delay clinical reaction.

Recently, following the elegant studies of HERNBERG and NIKKANEN in Finland, the determination of the erythrocyte enzyme, ALAD, has attracted attention. However, in the hands of many laboratories it would appear that this may not give reproducible results. It is also non-specific and has the disadvantage that significant inhibition begins at a very low threshold concentration of blood lead—indeed there may be no threshold for inhibition. While this has interesting philosophical implications

for the 'normal' population, it inevitably limits the diagnostic value of the test.

Primary tests have now virtually resolved to the determination of blood lead. The last few years have seen remarkable changes in the sophistication of the techniques available for this analysis. Unfortunately, this sophistication has not been matched by corresponding improvements in the standards of accuracy and precision. The problems have been highlighted by quality control schemes, in this country organized by Whitehead in Birmingham, and in the EEC countries by Lauwerys in Brussels. One example involving a single blood specimen resulted in values being reported from the participating laboratories which varied from 1–130 $\mu\text{g}/100\text{ ml}$. Appraisal of the results in the United Kingdom would suggest that there are at most about ten laboratories capable of performing this determination to anything approaching the required standards. It is difficult to escape the conclusion that some cases escape diagnosis and that others are unnecessarily treated.

Even if good laboratory services were available to the clinician, he is still faced with the problem of interpreting the data provided. What do particular blood lead concentrations signify? Part of this problem is semantic, since 'poisoning' is not clearly defined and has been taken to mean the child with manifest signs and symptoms, the asymptomatic child with an abnormal blood lead, and even children with evidence of ALAD inhibition. Some clinicians will not treat with chelating agents until the blood lead approaches 80 $\mu\text{g}/100\text{ ml}$, others regard a blood lead of 30 μg as an indication for admission to hospital. Even the accepted upper limit of normal in this country ranges from 36 $\mu\text{g}/100\text{ ml}$ in one centre in the south to 45 $\mu\text{g}/100\text{ ml}$ in one in the north.

Management of lead poisoning does not cease with diagnosis and treatment of the individual case. It is, or should be, mandatory that the child be removed from his home until such time as the source of lead has been identified and removed. There can be few other poisonings in which this practice is so persistently neglected. The problem is not great since the source of lead is almost invariably lead paint in the home but what is lacking is an effective machinery for this to be done. In one recent case in London, the public health inspectors visited the home of a poisoned child and left without taking a single sample for analysis. Not only did the house remain a risk for the child in question and his siblings but all the similar adjacent properties were entirely overlooked. This is not the fault of the inspectors

but an inevitable consequence of poor training, lack of scientific support, and indifferent supervision.

It has been argued that it would be preferable to detect children at risk before significant absorption of lead has occurred. This has been recognized in the United States in some cities—Baltimore, New York, Chicago—by the institution of massive screening programmes and there would appear to be little doubt that this, coupled with public education and effective follow-up procedure, can result in a profound diminution in the exposure of children to lead. To my knowledge, however, not a single programme of this type exists in the United Kingdom.

Alternatively, it might be thought desirable to limit the source of lead for children. However, no regulations exist which can enforce the removal of lead paint from homes inhabited by children and there is no legislation in existence or even contemplated restricting the sale of lead paint. By contrast, the United States already limits the lead content of paint to 0.5% and this is likely to be reduced to 0.06% by the end of this year. It is difficult to understand why so much attention has been given to less significant sources such as lead in gasoline, the abolition of which would not alter the incidence of lead poisoning at all, and at the same time would result in substantial increases in the importation of crude oil.

The required measures are all obvious and must include the provision of effective centres for the analysis of lead in blood and other material, the institution of blood lead screening programmes, and measures to remove lead paint from children's dwellings. However, these need to be supplemented by improved knowledge of the epidemiology of the disease. My department, under the auspices of the BPA, has now initiated the first study of this type in which information is being sought prospectively from all British paediatricians during the next year. It is hoped that this will also give some indication of geographical variation in the apparent prevalence of the problem and a basis for achieving more uniform diagnostic standards and criteria.

There remains a need, however, for the factors predisposing to lead poisoning in certain population groups to be identified. Are particular racial or cultural groups more sensitive, and if so, why? What is the role of the nutritional status of the individual in determining risk? And is the chemical and physical form of the source relevant to its potential toxicity? It is exciting to see the answers to some of these problems beginning to emerge in the work being reported in this Symposium today.