The current status of lead poisoning in children

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Introduction

In 1964, Moncrieff *et al.* recorded a normal range for the level of lead in blood as $24\text{ }\mu\text{g}\% \pm 4.9\text{ }\mu\text{g}\%$, with an upper limit of normal of $36\text{ }\mu\text{g}\%$. There has been discussion ever since as to the level of blood lead which denotes poisoning. A blood lead level of $80\text{ }\mu\text{g}\%$ was designated as a toxic level for children, requiring urgent and immediate treatment (U.S. Surgeon General, 1971). Barltrop (1971) presumed that $60\text{ }\mu\text{g}\%$ was an acceptable level below which symptoms did not occur. The Department of Public Health of Massachusetts (1973) took $50\text{ }\mu\text{g}\%$ as their upper limit of acceptability. More recently, Betts, Astley and Raine (1973) have shown a significant correlation between haemoglobin levels and blood lead concentrations in the range $37\text{--}60\text{ }\mu\text{g}\%$; levels which were previously accepted as harmless.

Incidence of lead intoxication in Great Britain

There is very little information on which to base an accurate estimate of lead intoxication in this country. Table 1 shows the incidence of deaths from acute encephalopathy (Registrar General), and the

TABLE 1. Lead poisoning in children

Year	1966	1967	1968	1969	1970	1971	1972
Deaths (Registrar General)	2	4	0	2	3	2	—
Hospital admissions (In-patient analysis)	—	—	220	76	133	100	183

frequency of the diagnosis of lead poisoning from the hospital in-patient analysis of about 200 cases per annum. These figures are notoriously inaccurate and Barltrop (1971) suggested that a more accurate incidence would be 2000 cases per annum. If the upper limit of normal blood lead is taken as $40\text{ }\mu\text{g}\%$ then a reasonable estimate of the frequency of lead intoxication can be made from the frequency with which elevated levels occur in previous series of normal and control children. Table 2 shows the results of previously reported series of normal children. The final column gives the numbers in each series found to have a blood lead above $40\text{ }\mu\text{g}\%$.

TABLE 2. Blood lead levels—British series

Reference	Number	Mean Pb level	$\pm$ S.D.	Elevated blood lead
Moncrieff <i>et al.</i> (1964)	77	24.4	± 4.9	2
Gibson <i>et al.</i> (1967)	20	29.6	—	3
Gordon <i>et al.</i> (1967)	73	30.9	$\pm 20.9^*$	16*
Bicknell <i>et al.</i> (1968)	14	12.0	± 5.2	0
Betts <i>et al.</i> (1973)	18	22.2	± 4.7	0
Delves <i>et al.</i> (1973)	44	11.7	—	0

* There is considerable doubt about the validity of these results.

TABLE 3. D.H.S.S. lead works survey (controls)

	Number	Mean Pb level	Elevated blood lead
Bristol (pre school)	679	13.6	2
(school)	140	14.4	0
Welwyn Garden City	93	27.5	10
Tower Hamlets	80	30.0	11
Matlock	53	22.0	1
Buxton	93	25.0	3

Table 3 gives details of healthy children gathered by the Department of Health as controls for Lead Works Surveys (Martin 1974): the final column again showing the number of elevated estimations.

If the results presented by Gordon *et al.* (1967) are included, a total of 48 out of 1,384 children have elevated blood lead levels (3.4%). The results of the series in question show a high mean value and a very high standard deviation and it is thought that they should be excluded. If this is done the total becomes 32 children with an elevated level out of 1311, an incidence of 2.4%. This figure compares with a recent survey of screening programmes in the United States of America of a total of 344,657 children which revealed that between 9% and 30% had a blood lead level equal to or greater than $40\text{ }\mu\text{g}\%$ (Lin-Fu, 1973). There are upwards of four million children in the U.K. between the ages of 1 and 5 years, which are the ages at greatest risk from lead poisoning (American Academy of Pediatrics, 1969). This means that there are approximately

100,000 children in the U.K. with elevated blood lead levels who are awaiting identification. This figure is of epidemic proportions but is probably more accurate than previous estimates. Obviously any large screening programme would need to offer immediate treatment of cases with levels in the toxic range. It would also attempt to identify and remove the source of lead.

Factors affecting lead intoxication in children

There is little doubt that children are more sensitive than adults to the toxic effects of lead (Bartrop, 1969; Lin-Fu, 1973). Kostial, Simonović and Pisonić (1971) showed that the uptake of lead from the intestine of suckling rats was fifty times that of the adult rat. Balance studies on eight healthy children aged 3 months to 8½ years showed an apparent absorption of 53% of the lead in the diet, 18% of which was retained (Alexander, Delves and Clayton, 1973).

From their experiments on rats, Six and Goyer (1972) concluded that an iron-deficient diet potentiated the effect of lead on this animal. Iron deficiency is common in the children at greatest risk from lead poisoning and may thus be a contributory factor.

These same workers (Six and Goyer, 1970) also showed a similar potentiation for the toxic effects of lead by a calcium-deficient diet. In particular, calcium deficiency increased the proportion of lead

in the soft tissues. Again these results are probably applicable to children. Residents in soft water areas have a higher tissue lead than those in hard water areas (Crawford and Clayton, 1973). The death of a child with typical lead encephalopathy was reported at a blood lead level of only 111 µg%. Figure 1 shows the X-ray of the child's bones which were compatible with severe rickets (Alexander and Delves, 1972).

As blood levels between 37 and 60 µg% have been correlated with pathological effects in the blood, it is logical to assume that lead also has an effect at these levels upon the brain and its development. David, Clark and Voeller (1972) showed evidence of a greater lead burden in over-active children as compared with controls. De la Burde and Choate (1972) reported reduced fine motor control in children with pica and evidence of mild lead intoxication. It could be argued that children who have one handicap are more likely to have associated abnormalities and have higher lead levels because of possible bizarre habits and diets. Kotok (1972) and more recently Lansdown *et al.* (1974) have been unable to show any relationship between lead ingestion or blood lead levels on the one hand and any measure of mental functioning on the other. All these studies have been limited by the testing procedures and the validity of controls. Nevertheless, if pre-school children are being affected by these lower levels of

FIG. 1. X-ray of lower limb bones showing severe rickets.

lead—albeit subtly or temporarily—then these children require identification and, if necessary, treatment.

Management

The most important part of the management of a child with lead intoxication is to separate the child from the source of lead. A combination of dimercaprol and E.D.T.A. given parenterally, as described by Chisolm (1968), is required for the urgent treatment of lead poisoning. Oral d-penicillamine is now well established as the treatment of choice either as a supplement to this chelation therapy (Chisolm, 1968) or alone for chronic lead intoxication (Vitale *et al.*, 1973). Apart from mild eosinophilia, no side effects were observed in the treatment of eight asymptomatic children over a two-month period (Vitale *et al.*, 1973).

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