Annotation

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Lead Poisoning

In contrast to their transatlantic colleagues, British paediatricians diagnose few cases of lead poisoning. In New York City the number of diagnoses has risen annually during the past two decades and has now reached 700 per annum; in Chicago 1336 lead poisoned children were detected in the two-year period 1967-68 from a childhood population at risk of 150,000 (Sachs et al., 1970). A recent estimate for the United States suggested that 225,000 children might have abnormally high blood lead concentrations though many must remain undetected (Oberle, 1969; Rothschild, 1970). The situation in this country has not yet been determined, though there is no doubt that few cases are diagnosed and that the distribution of positive diagnoses is not uniform. Hospital Activity Analysis suggested that about 200 cases were diagnosed each year in the whole of England and Wales (W. A. Wilson, personal communication, 1968), though extrapolation on a population basis from one area suggested that 2000 per annum would be more realistic (Barltrop and Killala, 1969).

The recognition of poisoned children is made difficult by the absence of any specific features that might suggest the diagnosis; the classical features of industrial lead poisoning such as wrist drop and the ‘Burtonian’ blue line at the gingival margin seldom, if ever, occur in childhood. The remaining features of early symptomatic lead poisoning include anorexia, irritability, and vomiting in association with pallor due to anaemia and pica; in the later stages these features are superseded by impairment of consciousness and convulsions. Typically affected children are 1 to 5 years old, live in old houses with poorly maintained paintwork, and present during the summer months (McLaughlin, 1956).

Numerous lead hazards for children have been described and have included such bizarre sources as lead nipple shields in nursing mothers (Ammaniti and Longobardi, 1962), lead glazed pottery vessels (Klein et al., 1970), the ‘fall-out’ from local refining plants (Chakraborty et al., 1964), and a retained intragastric foreign body composed of metallic lead (Biehusen and Pulaski, 1956). In this country, sporadic cases among children inhaling fumes from burning battery cases have been reported (Turner, Bamford, and Dodge, 1967) and from contaminated water supplies (Crawford and Morris, 1967), but the most common source in urban communities is indoor domestic paint (Chisolm and Harrison, 1956). A history of pica involving flakes of paint or painted plasterwork is frequently associated with lead poisoning (American Academy of Pediatrics, Subcommittee of Accidental Poisoning, 1969), though pica itself is a common activity in childhood involving 20% of children in the 1 to 5 age group and is thus of limited diagnostic value (Barltrop, 1966).

Though regulations now limit the lead content of paint that may be sold for application to indoor surfaces and to toys and nursery furniture, no provisions have yet been made concerning the paint of high lead content already in situ in the homes of many children. In general, the lead content of domestic paint increases with the age of the house due to incomplete removal of old paint at successive redecorations. In some American cities ‘lead-belts’ are recognized which invariably contain the homes of under-privileged members of their society. However, British lead paint appears to have been applied with less discrimination since toxic levels have been found in dwellings built after 1965 and in the homes of social class I families (Barltrop and Killala, 1969).

Lack of an agreed definition of ‘poisoning’ has contributed to the uncertainties surrounding diagnosis. The term might reasonably be taken to imply the occurrence of symptoms or metabolic disturbances attributable to lead in the tissues, but this distinction has not always been observed. Lead is a cumulative poison and the ingestion of paint flakes containing 1–2 mg lead (normal intake 0·2 mg per day) may continue for 5 to 6 months before symptoms occur (Barltrop and Killala, 1967). It is of interest that among 1155 children with blood lead values greater than 50 μg/100 ml studied by Sachs and her colleagues (1970), only 103 (8·8%) had clinical evidence of lead intoxication. Not all children with increased soft tissue lead progress to sympto-
matoxic poisoning, and some isolated increased values must represent an equilibrium between ingested lead and the removal from the soft tissue pool; others represent the slow return to normal after spontaneous cessation of pica. Radiological evidence of exposure may take as much as three months to develop (Cooper, 1947), persists after exposure has ceased, and is not necessarily related to the blood lead concentration.

Several metabolic disturbances have been attributed to lead intoxication resulting in delta-aminolaevulinicaciduria, coproporphyrinuria, increased erythrocyte protoporphyrin, and a ‘sidero-achrestic’ anaemia in which the entry of iron into the porphyrin ring is blocked. Recently, a more profound disturbance of globin synthesis has been noted in which there is an alteration of the alpha/beta chain ratio (J. M. White, personal communication, 1970). Mitochondrial abnormalities occur in the renal tubular cells in lead poisoning and probably account for the glycosuria, phosphaturia, and aminoaciduria that may be present. Previously, few metabolic disturbances were thought to occur until the ‘asymptomatic increased exposure’ stage was well advanced and the blood lead increased above the upper limit of normal (40 µg/100 ml). There is now evidence that the enzyme ALA dehydratase (E.C.4.2.1.24) is inhibited at blood lead concentrations hitherto regarded as ‘normal’ (Hernberg and Nikkanen, 1970; Millar et al., 1970), so that it may be necessary to revise our concept of poisoning.

Children with severe symptomatic poisoning may progress to encephalopathy and death, and this has occurred even after admission to hospital. Suspected cases should, therefore, be investigated with some urgency so that treatment may be started without delay. There are three groups of diagnostic tests available: the detection of lead in the blood, tests indicating disturbance of metabolism consistent with lead poisoning, and non-specific tests such as the demonstration of anaemia and glycosuria. The semiquantitative determination of urinary coproporphyrin (Benson and Chisolm, 1960) is the most convenient for emergency work. It amounts to little more than shaking acidified urine with ether and examining the extract obtained for fluorescence with an ultraviolet lamp. Blood lead analyses in the past were associated with methodological problems and required relatively large blood samples obtained with specially prepared syringes, but this situation has now changed. Commercially available, disposable syringes, and heparinized tubes are satisfactory and a semi-automated method is available for analysis. Recently a technique for the direct analysis of 10 µl (0.01 ml) of blood by atomic absorption spectroscopy has become available. Initial tests should all be completed within a few hours so that the transmission of specimens by post and the collection of timed specimens of urine as in the sodium calcium edetate provocation test are precluded.

Treatment of pre-encephalopathic lead poisoning comprises removal of the child from the source by admission to hospital, removal of soft-tissue lead by chelating drugs, and the prevention of recurrence. The drug therapy of lead intoxication is limited to sodium calcium edetate, dimercaprol and D-penicillamine which all bind with free lead and enhance its urinary excretion in the bound form. Sodium calcium edetate, 50–75 mg/kg body weight/day i.m. for 5 to 7 days, has been extensively used but it has some disadvantages in that it may not penetrate cell membranes, and the lead which it removes is probably derived from the surface of bone, with only secondary removal from soft tissues (Hammond, Aronson, and Olson, 1967). Children with early or suspected encephalopathy should be treated with chelators and restriction of fluids to basal needs before the results of the laboratory tests are known (Chisolm, 1968). Intravenous infusions of mannitol, 2·0 g/kg body weight or urea, 1·0 g/kg body weight, may be used to induce urine flow and reduce cerebral oedema; dexamethasone, 1·0 mg/kg body weight, has been used in the management of encephalopathy but it is of uncertain value. Combinations of sodium calcium edetate and dimercaprol, 4·0 mg/kg body weight 4-hourly, decreasing to 2·5 mg/kg on the 4th day, have been shown to be the most effective means for the removal of lead from the body and for reducing the blood lead concentration (Chisolm, 1968). Other authors had advocated the use of sodium calcium edetate alone in all but the mildest cases in which D-penicillamine, 20 mg/kg body weight per day, is used. The advantages of prolonged courses of chelation are dubious since the most significant period of treatment is probably the first 48 hours; repeated courses may mobilize decreasing amounts of lead, but these probably represent a very small fraction of the total body burden. Animal studies with radioactive isotopes of lead have shown that some soft tissue lead is excreted in the urine and in the bile (Castellino, Lamanna, and Grieco, 1966), but the remainder is deposited in bone (Bolanowska, Piotrowski, and Trojanowska, 1967) where it is metabolically inactive. Ideally, treated children should not be returned home until the source of lead has been chemically identified and removed, but in this country this is not readily achieved.
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The sequelae of encephalopathy include cerebral damage in about 25% of poisoned children. Though the mortality from encephalopathy can be reduced to about 5% (American Academy of Pediatrics, Subcommittee on Accidental Poisoning, 1969), this is still an unacceptably high figure. An increased liability to chronic nephritis in later life has been reported (Henderson and Inglis, 1957). Reduction in mortality and morbidity is unlikely with existing methods of treatment and will probably be achieved by preventive rather than therapeutic measures. Logically, screening programmes should be designed to detect lead in the domestic environment so that it may be removed before poisoning occurs in exposed children. Perhaps local authorities should be encouraged to play a more positive role in the prevention of lead poisoning than is at present the case.

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REFERENCES


