Moderately raised blood lead levels in children

BY R. Lansdown

Department of Psychological Medicine, The Hospital for Sick Children, Great Ormond Street, London WC1N 3JH, U.K.

There is no doubt that high blood lead levels are associated with mental subnormality and hyperactivity. Several recent studies in Britain and America have investigated the relation between moderate levels, i.e. between 20 and 40 µg/100 ml and behavioural and cognitive phenomena. Epidemiological studies have generally failed to point to a clearcut relation between such levels and overactivity or decrements in performance on standard intelligence and educational tests. Published studies with the use of chelation techniques have suffered from methodological weaknesses. It is known that socio-economic factors are powerfully related to measured intelligence and behaviour and, on the evidence available, it is to them that attention should primarily be given if preventative measures are being considered.

There remains the possibility that more refined test measures would detect impaired functioning in children with moderately raised lead levels, and that there is an interaction effect between lead and host resistance.

The subject of this paper carries a high emotional charge. ‘Lead poisoning’ is a loaded phrase and it is easy to arouse emotions with descriptions like ‘one of the oldest and stealthiest poisons known to man’ (Ottaway & Terry 1976).

The effects of damaging levels of lead ingestion, notably behaviour disturbance and learning difficulty, are also high arousal subjects. Those of us who have been concerned with educational failure frequently find ourselves under pressure to provide a single, testable answer to the question ‘Why can’t Johnny read?’ and the history of education is full of attempts to meet that pressure. Lead, for some commentators, appears in part to meet it.

No one writing in the last few years has doubted that high levels of lead in the body can be harmful. As Millar et al. (1970) noted, the health hazard has been discussed since the seventeenth century. But, as the report of the Department of the Environment Working Group on Heavy Metals made clear, lead is naturally present in all components of our environment and in man himself (Department of the Environment 1974). In a study published in 1967, quoted in the Working Group’s report, the average blood lead level for U.K. inhabitants was 23 µg/100 ml, which was exactly the same figure as that obtained from a sample in Central Brazil and only 1 µg/100 ml higher than the figure gained from mountain dwellers in New Guinea. The D.O.E. report concludes that ‘evidence of harmful effects in adults is rarely seen at blood levels below 80 µg/100 ml; indeed, cases of poisoning usually involve levels considerably in excess of this’.
Symptoms in children are different from those in adults and the disease is generally more serious, though signs may appear at lower blood lead concentrations. The report underlines this conclusion in its next paragraph, which cites levels up to 40 μg/100 ml as normal; from 40 to 80 μg as acceptable, from 80 to 120 μg/100 ml as excessive and more than 120 μg/100 ml as dangerous.

These figures, though, are described as both arbitrary and not applicable to children. As with adults, there is no doubt that a high level of lead, say 120 μg/100 ml, is dangerous and can give rise to behavioural and learning difficulties. There is, however, a grey area, of around 25–60 μg/100 ml, where there is considerable uncertainty about the effects of lead on children. This moderately raised level is the subject of my paper today. I propose first to report some studies which have argued that a moderate level results in disturbance of functioning, and then others, including an epidemiological study, which found no such thing. This will be followed by an interpretation of later work which, in my view, supports the null hypothesis, and I shall conclude with a mention of a recent work which may stand my argument on its head.

The work of David and his colleagues in America has consistently pointed to the adverse effects of very moderately raised lead levels (David et al. 1972, 1976). For example in the 1972 study they reported on 91 children who had been referred to the Out Patient Clinic of a Brooklyn hospital. Of these, 54 were placed in a ‘hyperactive’ group and 37 in the control group. The former had blood lead levels of 26 μg/100 ml compared with 22 μg/100 ml in the controls. There were also differences in urine lead levels in the same direction. Now, what can be concluded from these figures? Both the hyperactive and the control group have blood lead levels very close to the already quoted average for adults. With no evidence to the contrary it would appear that David’s two samples came from a population exposed to roughly the same amount of lead and one must, therefore, look for other possible causes for the increased hyperactivity of the 54 children. It is possible that disturbance preceded lead ingestion, leading to inappropriate feeding habits. The general possibility of such an antedating is acknowledged by Landrigan et al. (1975) and remains a plausible explanation.

Evidence against the simple equation of raised lead levels equals adverse effects comes from Kotok (1972) in which 24 children with raised lead levels were compared with two comparison groups. Both the experimental and comparison groups included children with delays in fine motor and language areas and there was no evidence for a relation between raised lead levels and the results of the developmental test used. There was, however, a correlation between the degree of environmental disruption experienced by the children and their developmental scores. Kotok also quotes unpublished work by H. Costoff and S. Provence which yielded similar results, finding a correlation between development and the quality of mother–child relationships and none between development and lead levels.

There was, then, no clearcut evidence on what I have already referred to as this grey area when in 1972 my colleagues and I became aware of the population
living in the vicinity of a smelting works. The factors causing contamination had been present for about 30 years, so we could assume that all children born and still living in the area had been exposed to a higher than normal level of environmental lead all their lives. The intake of lead was derived in part by inhalation of airborne emissions, in part by inhalation of dust in houses and on roads and in part by the ingestion of dusts.

The area is not one of London's most fashionable quarters. A description of it in 1972 by a student of London history saw it as '... a desperate place... every indicator of urban health is pointing resolutely downwards... shops are woefully inadequate... factories are closing and jobs fleeing... the housing is mostly unattractive and unpopular - 97% of it being in public ownership. At one stage there were 400 council flats untaken and the council admit that the "housing officer is obliged to go further down the waiting list than is normal" to fill them... it is hard to see why any young people should stay'. I have quoted at some length from this article because much of the interpretation of our figures rests on a knowledge of the area as a social entity as well as just a place where children gained high lead levels.

Here we had a densely populated area in which we felt we could examine the results of moderately raised lead levels epidemiologically, i.e. we did not take a selected sample with all the bias that such a technique can give rise to; we were able to have access to a total population. This methodological point is stressed since it is comparatively rare in research of this kind. The population to be studied was identified by a house-to-house search for children living within 500 m of the works. A total of 476 under the age of 16 years were found, of whom 119 were under the age of 5 years.

Lead levels were determined in duplicate in 10 µl samples of whole blood by atomic absorption spectrophotometry (Delves 1970). Recent work has shown that the micro-method we used may overestimate the lead in blood by 5 µg/100 ml. Since much of our statistical analysis relied on correlation techniques the uncertainty of our measures is less important than it might otherwise have been.

Our first question was whether there was evidence of raised lead levels and whether such levels seemed to be related to the works in question. The mean lead level of the children in the main study, i.e. those between 6 and 16 years (n = 125) was 33 µg/100 ml with a standard deviation of 9. Exactly 20% had levels of 40 or over. The figure for pre-school children with a 40+ level was a similar 21%. More striking was the relation between lead levels and proximity of the children's home to the works. Taking mothers, pre-school and school age children separately there was a statistically significant relation, with the trend of nearer to the works meaning higher lead levels. (See Lansdown et al. (1974) for details.)

The next question was to examine the effect of such exposure on children's functioning. An examination of the literature suggested that we should look at several areas, including:
(a) intelligence, if possible using tests which would differentiate between verbal skills and those involving visuo-motor abilities;

(b) general behavioural characteristics, with particular reference to overactivity;

(c) reading ability.

Because we were fortunate enough to be able to carry out a population study we did not need a control group but, since we were planning to employ correlational techniques we did need tests which fulfilled the requirements of parametric statistics, and we therefore chose an established, well standardized test to look at intelligence, namely the Wechsler Scales for Children and Adults. Because of the standardization characteristics of these scales we could not test all of the pre-school children and therefore chose the age group from 6 to 16 years.

The reading test used was the Burt Graded Word Test. This is simple to administer and has a highly significant correlation with other standardized tests (Lansdown 1973). All testing was carried out by supervised psychology graduates.

Information of the previous addresses of families, the length of stay in any one address, the social class of the father (based on occupation) and length of education of both parents was obtained by health visitors who made home visits to each family.

Ratings of behaviour were gained in school, by using a scale filled in by teachers (Rutter 1967). This scale is of known reliability and validity and has been widely used both in primary school (Rutter 1973) and in secondary schools (M. J. Rutter, personal communication). It contains three items relevant to overactivity and enabled us to extract an admittedly crude measure of this characteristic.

The results of our tests can be stated fairly simply: they were negative. To take intelligence first: The mean I.Q. was 101 with a standard deviation of 11. In order to look more closely at these figures we correlated them with blood lead levels and found a correlation of 0.05, i.e. there was no significant relation.

Behavioural ratings gave results which were, to some people, astonishingly high. Overall 22.8% of the children were rated by their teachers as disturbed. This is a high figure only to anyone not acquainted with London schools in general and socially deprived areas in particular. I recently spoke at a conference of teachers in Somerset and heard a London teacher introduced as ‘straight from the battlefield’. Rutter, in a study of children 10 years old in another London borough, used the same scale and gained a total of 23.2% rated as disturbed (Rutter 1973).

The children seen as disturbed were considered as a function of their lead levels and again there was no significant relation. The same was found when overactivity was considered, and an examination of reading scores yielded similar results.

There was, however, an anomalous result. When we broke down our figures by lead levels, we found that children with higher levels had, generally, higher intelligence scores and a lower rate of disturbance. This we found difficult to comprehend until we plotted scores with geographical mobility in mind. It was pointed
out to us that much of the new housing in the district was on the extreme edge of the area that we surveyed. This was the housing that the local council found so difficult to fill because the district is so unfashionable.

An examination of test results of children according to where they had spent the first two years of life shed some light on our apparently anomalous finding, since there emerged a new equation: the nearer the lead works meant the higher the lead level, which meant the more stable the family, which meant the higher intelligence. When we isolated children who had not spent the first two years of their lives in the area this pattern became clear. Anecdotal evidence from residents and health visitors bore out the comments of the Housing Officer quoted above, i.e. the newcomers were those who might be expected to produce less intelligent, more disturbed children for reasons quite other than lead ingestion. There was, however, one slight hint that our findings were not as simple as that. The pattern of overactivity in children showed the same general picture, with the ‘newcomers’ being more overactive, but there was a trend among the others which suggested that the nearer the works one was born the higher would be one’s overactivity. This failed to reach statistical significance but it should be noted.

Several other reports have been published since this one. Landrigan et al. (1975) examined 46 children with lead levels from 40 to 68 µg/100 ml (mean 48) and compared them with 78 controls. All children lived within 6.6 km of a lead emitting smelter. They conclude that ‘chronic lead absorption ... may produce subclinical impairment in neuropsychologic function’. I question, though, the grounds for going beyond the tentative ‘may’ in this conclusion. There were no significant differences between the two groups on 18 out of the 21 measures taken, including hyperactivity and full scale intelligence quotient. Of the three tests yielding different scores one can be explained as a statistical artefact and the other two have internal contradictions, being similar in content to two which were not different.

In a large-scale study, Hebel et al. (1976) examined the 11-plus scores of 851 Birmingham children living since birth in a lead polluted area and compared them with 1642 children from two similar but unpolluted areas. They found that children living in lead polluted communities were academically more successful than those living in unpolluted communities. Their conclusion was that the quality of education in the schools in the lead area was higher than in the other two and that this factor was paramount in explaining their results.

There have been other related studies, e.g. on water borne lead (Beattie et al. 1975), but my own conclusion so far remains that epidemiological evidence suggests that the effects of moderately raised lead levels in children have been exaggerated. It is, of course, possible that tests used so far have been too coarse to pick up the fine differences that do exist. It is possible, as P. Bryce-Smith has suggested (personal communication), that there is an interaction between lead and some other factor in the child. Both points need evidence to substantiate them. Failing
such evidence, I suggest that we should look primarily to social and educational measures if we wish significantly to improve children's learning and behaviour.

There remains one final piece of work, that of the de-leading chelation procedure used by David and his colleagues, reported last year (David et al. 1976). To summarize: David took a group of hyperactive children, found that some had lead levels of 25 \( \mu \text{g}/100 \text{ ml} \) or more and de-leaded them by using chelation medication. In those children for whom there was no other known cause for hyperactivity there was a dramatic improvement in behaviour. Unfortunately the number of children involved was small, only 13 in the whole study and there was no report of the use of a placebo. David concludes by admitting that many children have raised lead levels and are asymptomatic and looks to host resistance as a major factor in what he describes as a complex interplay. I understand that a report of a placebo trial is in preparation (D. Bryce-Smith, personal communication).

I said at the beginning of this paper that this final work, on chelation, might stand my own conclusions on their head. This is not strictly true, of course, because the emphasis on interaction effects is quite different from the earlier quoted work which sought to show that a moderately raised lead level is, ipso facto, dangerous. Given that human behaviour and learning are multifactorial in their determinants, it seems likely that an approach allowing for this characteristic will possibly be fruitful in future.

References (Lansdown)


Department of the Environment 1974 Lead in the environment and its significance to man. (Pollution Paper no. 2.) London: H.M.S.O.


Moderately raised blood lead levels in children

