Discussion

CHAIRMAN: It is interesting that Dr White has shown with concentrations of 50 μg/100 ml of blood there is a 20% depression in globin synthesis. It provokes questions on the mechanisms of action and on the wider issues of what are the levels of blood lead which are tolerable and what might be harmful to various members of the community.

People do not seem to fit into in vitro and animal studies; they seem to make their own adjustments in these things and this may apply to other environmental pollutants such as cadmium and mercury. It may be that man has taken an unconscionable time in surviving and in doing so has managed to overcome all sorts of pollutants from prehistoric monsters to the drug explosion as well as the pollutants of the Industrial Revolution. There is no doubt that he has his own mechanisms for overcoming all these things in various ways.

DR M. K. WILLIAMS: Sub-clinical lead poisoning is an interesting term when it is used to refer to specific possible hazards, such as effects on fetuses and pregnant women, but it has also been used to express a fear that the whole adult and paediatric population of the country is subject to some risk which is, as yet, undefined. From classical times to the modern day there has been very little disagreement between specialists who have observed clinical lead poisoning; they have all described colic, anaemia, encephalopathy and neurological effects and we know that as the dose is reduced, so these effects tend to disappear. To suggest that when the dose is reduced, some other unmentioned effect will begin to appear, would seem to be so inherently unlikely that it might well be discounted. This of course does not mean that we should not continue to look for long-term effects such as coronary artery disease, but I think it should be borne in mind that these are long-term effects which require properly controlled epidemiological studies to determine whether they exist. It is like saying that every cigarette smoker is suffering sub-clinical poisoning because some of them develop cancer of the lung.

Professor Goldberg mentioned renal effects in rural workers but I wonder if there were suitable controls so that these effects could really be attributed to lead. Ten years ago, in the United States, Tepper was unable to find renal effects in children or in adults who had suffered lead poisoning as children.

If I might say so, I think Professor Goldberg approached this as a clinician who sees sick people in hospital and then tries to find the cause for it. I am an industrial medical officer with control of large numbers of lead workers in whom we are unable to find any of the effects he described.

It was stated that there was general agreement that the lead content of petrol should be reduced. I do not accept that there is a general agreement. The cost benefit of this has not been shown to be a reasonable way of spending our gross national product. If it costs £200 million a year to remove lead in petrol, this could be spent very much better in other ways on the health of the country.

Reference might have also been made to the El Paso smelter study on children, where children were exposed to vast amounts of lead, presumably from birth, and recent studies have been unable to detect any effect.

CHAIRMAN: I defined sub-clinical lead poisoning as the possible, yet unproved effects on community health of an increased exposure to lead, insufficient to cause frank lead poisoning. We are in a stage in clinical medicine where there are a whole number of areas of sub-clinical risks, and sub-clinical lead intoxication is one of these.

In our study of industrial lead poisoning we have also shown that there was no evidence of renal dysfunction (Gibson, MacKenzie and Goldberg, 1968).* Tepper's work agrees with that, but there is no question that lead can cause effects on the kidney. The 'nephropathia sarturnia' has been known for about 100 years and we know that this is true. How can we tell that lead was a toxic factor in the small group of rural studies carried out by us? It happened that these patients in fact did improve during follow-up when they were not exposed to this high level of lead. We are also carrying out a bigger study in rural Scotland, in association with the College of General Practitioners, to see whether the incidence of renal disease may be associated with excessive lead in the water supplies.

Now as far as lead in petrol is concerned, I think that contamination should be at least restrained.

DR WILLIAMS: There are much more important things to do with the money that it would cost.

DR D. MALCOLM: I agree with Dr Williams that we do not have sufficient evidence as yet for lead in petrol to show that it really needs to be reduced.

One of the confusions in the whole of the lead field is the quoting of blood leads. I would not pay much attention to any study where adequate interlaboratory controls and control samples had not been included. I think that this quoting of blood lead levels has resulted in a great deal of confusion because many papers are written where the standards of analysis are inadequate.

The study on haemoglobins is interesting. It would be worth looking at lead workers because I have had the impression that there is a tendency in well-controlled lead workers for haemoglobins to rise early on. With sustained blood lead levels over 100 μg/100 ml, the haemoglobin levels may come down slowly and when you get sustained blood levels of over 120 μg/100 ml, the haemoglobin may come down fairly quickly, say within three months. However, in my own work and in Dr

Williams’s studies, we found no correlation in our own groups between lead levels and haemoglobin.

**Dr. J. White:** It would be interesting to look at the haemoglobin and measure their rates of chain synthesis in some of these workers, for it is our only indication that the cell lacks haemoglobin. Now some of the enzymes on the mitochondria to be shown by Dr. Moore have impaired function, but what the cell needs is haemoglobin. If workers were studied at the beginning of their employment and then, say, after a few months, it would be possible to relate increases in their lead level, with changes in alpha:beta ratio. If blood lead increased sharply I would expect a fall in the ratio.

**Chairman:** Blood lead analyses can be utilized but there are means now for quality control. The blood lead reflects a certain state in the body in relation to a particular time, if there are problems in the quality control then they have got to be put right, but I think that the blood lead level, efficiently measured, is very useful.

**Dr. Malcolm:** I am criticizing those studies that are not properly controlled. As a doctor working in industry I find this is, so far, the best method of control, provided it is properly done.

**Chairman:** So far as haemoglobin is concerned, we are reaching a stage when lead poisoning is now very much less severe than it used to be. Anaemia, I should imagine, was very rife in the old days and the haemoglobin level was a very useful test but now, with the fall in the incidence of lead poisoning, it is less and less helpful. We are dealing with more sensitive indices of lead poisoning.

**Professor D. Bryce-Smith:** Psychological assessment of children at El Paso with blood lead levels greater than or equal to 40 \( \mu g/100 \) ml, has shown impairment in a broad range of psychological function. They showed significant reduction in performance IQ and this was also apparent even on re-testing after their blood lead level had subsequently fallen below 40 \( \mu g/100 \) ml. The El Paso experiment does not support any suggestion that lead has no effect at these levels.

**Dr. P. S. I. Barry:** It is necessary to correct Professor Bryce-Smith’s remarks. I was involved with the El Paso study at some earlier stage and have seen the final report. Some psychometric tests were undertaken on the children involved. The blood lead levels in most of the children were over 40 \( \mu g/100 \) ml but there were very few above 80 \( \mu g/100 \) ml. The results of the psychometric tests were all negative with the exception of one test which it was thought was probably influenced by the social disruption of the families following their redistribution in El Paso, after the Smeltertown community had been dispersed. Apart from this one test none of the others showed any evidence of mental aberration or of IQ change from a normal expectancy.

**Dr. J. G. Dathan:** I was rather horrified to hear people talking about sustained blood lead levels of 100 or 125 \( \mu g/100 \) ml. Why should they be allowed to be sustained at that level? I am engaged at present on a lead survey in the pottery industry. We set our standards, I thought, rather too high. We accept as normal up to 35 \( \mu g/100 \) ml, we call them acceptable up to 60 \( \mu g/100 \) ml, and over that level we really begin to get aggressively more concerned about them. We automatically suspend them immediately if we find one of 125 \( \mu g/100 \) ml. We would probably suspend most of those over 100 \( \mu g/100 \) ml and I have certainly suspended them at 85 \( \mu g/100 \) ml onwards if they are tending to rise. Why they should be allowed to go on over 125 \( \mu g/100 \) ml, I do not understand, and how rigid are these figures? I was at a meeting earlier this week when people were quite gaily talking about blood lead levels of 120 \( \mu g/100 \) ml and really thinking nothing much of them, yet in Ontario a workman automatically qualifies for compensation if he has a blood lead level of 60 \( \mu g/100 \) ml. There seems to be considerable broadness of opinion.

**Dr. J. Quarterman:** I would like to ask some of the human clinicians if they think that the symptoms, and the severity of lead poisoning may depend on a whole range of factors other than the level of lead, either in the diet or in the body. At the Rowett Institute, we have in two experiments produced sheep, both groups of which had blood lead levels of 50 \( \mu g/100 \) ml. They were on different diets, of different sexes and different ages. One group of sheep was perfectly normal—we could detect no clinical changes except a small decrease in growth rate. The other group of sheep was dying. They were losing weight, had kidney failure and osteoporosis. So I am beginning seriously to doubt the clinical value of blood lead determinations, but I cannot suggest anything better at the moment. In sheep in our experiments, and in rats to some extent, and in observations on clinically poisoned sheep and other animals throughout the country, there is a wide variety of clinical symptoms, with similar levels of lead intake. So there must be other factors which influence the clinical picture.

**Dr. White:** Probably one species will not be the same as another species, and there is a marked variation in effect.

**Dr. J. Taylor:** With regard to lead workers’ health should we be using blood leads or some other test to find out if a person is sensitive, rather than his blood lead?

**Chairman:** There are a whole number of tests which one can do but in all of this one should take an eclectic approach and take the things that you can do practically. The blood lead is useful, and the haemoglobin test is useful. A clinical examination is also useful. We know that the response of different workers is different and I have certainly been impressed by the fact that early on in lead exposure this tends to show itself. For example, there are some people who get lead poisoning quickly, after about 3–6 months in industry and these tend to be sifted out of the community, whereas the workers that have been there for 5–20 years are pretty tough people who have been exposed for a long period. There are other factors, including alcoholism.

In these animal studies, of course, it sounds as if you have got osteoporosis and a whole number of other factors as well. What was the relationship of the calcium intake in these particular animals and were there many factors in addition?

**Dr. P. Sayers:** I regard the blood lead as an index of environment and I would group my factories according to mean blood lead values and supervise them accordingly. But if I found a man with a significantly raised blood lead then I would look at him in depth using every possible test in the book, of which I would regard a raised urinary ALA and a low haemoglobin as important. However,
determining the blood lead is not going to cure the problem, it serves as a red light calling for the help of the industrial hygienist.

Mr R. H. Hill: I agree very much with Dr Sayers. The blood lead level can only be used as an index of the environment and must be based on a big enough group. In a study, that we hope to publish, in Newcastle on a group of workers whose blood was sent to three separate laboratories, all well known and of high standing, we found that not only was there a variability within each laboratory of at least ±9 μg/100 ml whole blood but that they differed from each other as to who had a 'high' level: some laboratories reported high blood lead levels for one man and some for another but there was little correspondence between them.

In any discussion we must have reservations on what people have to say about a given individual’s blood lead level. The individual should be reviewed in terms of all the factors available, not one.

Dr Malcolm mentioned blood lead levels of 100 or 120 μg/100 ml. Such levels can be reported in people who have absolutely no evidence whatever, clinically or biochemically, of any other disturbance. For them to be suspended merely for having these levels seems to me to be unjustifiable. We should all learn to distinguish between the valid use of grouped blood lead figures for environmental monitoring as opposed to its inaccurate use in the assessment of one individual’s health.

Dr A. Berlin: We have carried out two inter-comparison programmes on lead measurements within the European Community, and unfortunately both of them gave poor results. In the first programme, for a blood sample having approximately 30 μg/100 ml of blood, the values reported ranged from about 10 to 200 μg/100 ml. In the second programme, which we ran with the same laboratories and additional ones about a year and a half later, things did not really improve much. Furthermore, we tested the intra-laboratory variability by sending identical samples without knowledge of the laboratories. Intra-laboratory variability was very high, much more than what the laboratories reported on duplicate analyses which they ran on the same samples.

We looked at another parameter, ALAD, and on this we had more success. We first started by sending two blood samples and looking at the ratios between ALAD values for different laboratories each using its own methods; obviously one could not look at the absolute values. The ALAD ratios looked very good; in fact that variation was less than 20% between ten laboratories on the same two bloods.

We then developed a standard method which was tested by about twenty laboratories and in this case the results were even better; the variation reduced itself to about 10-15%. We then carried out a population study on about fifty persons at nineteen locations in the European Community and also Switzerland, Finland and Sweden, and looked at the ALAD and the blood leads. The ALAD’s looked reasonable, and significant variations were found between different cities. They did not correspond to the blood lead variations and there was little correspondence between the blood leads found by the local laboratory that did the analysis and various central laboratories which served as a centre. Blood lead levels should be regarded as an index of an effect. On the other hand, one could look at the ALAD and consider it from this latter point of view.

While the lead intake from air might be relatively minor in comparison with other sources, one should be aware that, in the long run, injecting lead into the air redistributes the lead in our local environment, which might lead to an increase of uptake of lead from other sources and not directly from the air. I agree that one should try to restrain it whenever possible, taking into account what effects this might have, even from an economic point of view, but it is really better to try to leave it where it is for the moment.

Dr H. A. Waldron: I would like to allude to the study by McRoberts, 1973,* on a very small group of lead workers in which he appeared to show that the development of symptoms depends upon the partition of lead between the red cells and the plasma. In the group of workers with high blood lead levels who showed no ill effects, he found the partition between red cell and plasma was low. That is to say, the lead was bound on the red cell where it seems to be relatively inert and the development of symptoms depended upon an increase in the plasma lead concentration. This is what one would expect because it is the ionised lead which is likely to have an effect on the tissues and perhaps one should perform plasma lead analyses in addition to whole blood lead analyses in order to look for changes in the partition coefficient.

Discussion

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