British Journal of Industrial Medicine 1982;39:283–289

Occupational lead exposure of storage battery workers in Korea

B K LEE

From the Department of Preventive Medicine, Catholic Medical College, Chongno-Ku, Seoul, Korea

ABSTRACT Two hundred and thirty-four lead workers employed in a storage battery factory in Korea were examined for lead in blood (PbB) and urine (PbU), δ-aminolaevulinic acid in urine (ALAU), coproporphyrin in urine (CPU), and haemoglobin. The dose-response relationship between PbB and ALAU suggested that a PbB below 50–60 μg/dl is a proper practical limit of biological monitoring for lead workers. The inter-relationship between PbB and ALAU or PbU was better explained by a segmental straight function than by a curvilinear function. Inclusion of data from workers whose PbB was below 30–40 μg/dl, if they comprise a relatively large proportion of the whole, seems to have a role as a dummy effect on the overall regression function causing the curvilinear trend. At a given blood lead concentration, the ALAU of lead workers increased with an increase in the duration of exposure. This could be explained by the chronic effect of lead on haem precursors. Semi-quantitative measurement of CPU still played an important part in the screening of lead workers due to its simplicity, showing high sensitivity (97·8%) in detecting lead workers with PbB of 60 μg/dl or over.

The effects of lead on human health have been investigated intensively for the past 50 years, and the pay-off in terms of human health has been dramatic. Through application of engineering control measures and proper medical supervision the level of human exposure in lead-using industries has been reduced considerably in recent years, and the incidence of clinical lead poisoning in the populations at risk has been drastically reduced.[1 2] Consequently, the effects of lead on health have become progressively less frequent and serious. But the application of newer, more advanced diagnostic methods[3–5] and the changing concept of unwanted effects of lead on man continue to show previously unknown effects occurring at relatively low levels of exposure.[6] So a great deal of attention is focused nowadays on the untoward effects said to occur when tissue levels are not sufficiently high to produce frank clinical intoxication.[7]

Blood lead (PbB) is considered to reflect the magnitude of the current and recently acquired biologically active fraction of the body burden of lead,[1] and the concentration of lead in the blood is widely used as an index of total exposure and an index of biological permissible limits for lead workers.[8–10]

Many studies suggest that PbB of around 60 μg/dl is more reliable for biological monitoring of lead workers than 80 μg/dl of PbB, which is relevant to air concentration of lead of about 150 μg/m³.[9 11] On the other hand, Williams[12] opined that there appeared to be no evidence or good reason for reducing current limit levels.

Laboratory tests used in the control of lead exposure fall into one of two categories, lead absorption and lead intoxication.[13] Several studies have been made correlating the different tests with each other; the results of these studies, however, are not always in full agreement.[14 15]

Subjects and methods

SUBJECTS

Two hundred and thirty-four male lead workers employed in a storage battery factory in Korea were investigated. Table 1 shows their age, work duration, job, and air concentration of lead.

METHODS

Air sampling for lead concentration of air was car-

Dissertation submitted for the MSc in Occupational Medicine at the London School of Hygiene and Tropical Medicine (University of London).

Received 10 July 1981
Accepted 27 October 1981

Table 1 *Age, work duration, and lead concentration of air by workplace*

Workplace	*No of workers*	*Age (years)*	*Work duration (years)*	*Concentration of lead in air(mg/m³)*
Assembly	102	26·8 ± 6·2*	3·3 ± 3·0	0·38 ± 0·21
Casting	47	29·3 ± 5·8	5·3 ± 3·8	0·20 ± 0·05
Pasting	53	29·7 ± 6·3	5·1 ± 4·3	0·17 ± 0·04
Refining	15	31·1 ± 9·3	5·2 ± 4·3	0·07 ± 0·03
Lead powder	17	28·7 ± 6·2	5·5 ± 4·1	0·21 ± 0·08
Total	234	28·4 ± 6·5	4·4 ± 3·8	

*Mean ± standard deviation.

ried out five times in each workplace with a Hi-volume air sampler; the samples were analysed by a dithizone method.[16]

The following biochemical tests were carried out on all workers. PbB[17] and urine lead (PbU)[16] were measured by a dithizone method; urinary δ-aminolaevulinic acid (ALAU) was measured by the method of Tomokuni and Ogata[18]; semi-quantitative measurement of urinary coproporphyrin (CPU) was carried out by the method described by de Kretser and Waldron[19]; haemoglobin were checked by the cyanmethemoglobin method. All the urine analyses were made on spot samples, and all urine samples were corrected to a specific gravity of 1·016.

Results

COMPARISON OF BIOCHEMICAL DATA BETWEEN WORKPLACES AND BY WORK DURATION

Tables 2 and 3 show the mean value of PbB, PbU, ALAU, and Hb by workplace and by work duration. There was no statistical difference of PbB between workplaces, but the proportion of workers whose PbB were over 60 μg/dl was different, the highest values being found in the assembly department, which also had the highest air concentration of lead (table 4).

The mean level of PbB, PbU, and ALAU seemed to be increased as the duration of exposure increased; there was a statistically significant difference between the data from men with under two years' and over six years' exposure ($p<0·05$). No differences were shown in mean level of Hb by work duration.

DOSE-RESPONSE EFFECTS OF LEAD ON THE HAEMATOPOIETIC SYSTEM (table 5)

The response level for each of the biochemical indices was selected as follows:

ALAU—Five and 10 mg/l were selected as the cut-off points. The former was regarded as the upper limit in non-occupationally exposed subjects and the latter as the acceptable limit in lead workers.[9]

Hb—Twelve and 13 mg/dl were selected. The latter was the lower limit of healthy lead workers in

Table 2 *Biochemical data by workplace*

Workplace	*PbB (μg/dl)*	*PbU (μg/l)*	*ALAU (mg/l)*	*Hb (mg/dl)*
Assembly	55·9 ± 21·3*	134 ± 103	8·6 ± 8·0	13·1 ± 1·6
Casting	51·7 ± 17·2	107 ± 64	6·9 ± 5·5	13·5 ± 1·1
Pasting	54·0 ± 16·4	115 ± 68	8·1 ± 6·2	12·9 ± 1·4
Refining	45·8 ± 20·0	90 ± 51	7·2 ± 6·6	12·6 ± 1·9
Lead powder	53·8 ± 16·3	93 ± 49	8·8 ± 5·8	13·2 ± 1·2
Total	53·8 ± 19·0	119 ± 84	8·2 ± 6·9	13·1 ± 1·5

*Mean ± standard deviation.

Table 3 *Biochemical data by work duration*

Work duration (years)	*No of workers*	*PbB (μg/dl)*	*PbU (μg/l)*	*ALAU (mg/l)*	*Hb (mg/dl)*
<2	41	45·6 ± 20·0*	101 ± 79	5·0 ± 3·6	13·4 ± 1·7
2–4	81	47·4 ± 18·2	95 ± 74	6·7 ± 4·9	13·2 ± 1·4
4–6	49	56·7 ± 18·1	135 ± 105	9·5 ± 8·0	13·0 ± 1·4
>6	63	65·2 ± 13·6	148 ± 68	11·1 ± 8·5	12·9 ± 1·4
Total	234	53·8 ± 19·0	119 ± 84	8·2 ± 6·9	13·1 ± 1·5

*Mean ± standard deviation.

Table 4 *Number of workers by level of blood lead and workplace. (Percentages shown in parentheses)*

Workplace	Blood lead (μg/dl)				Total
	≤40·0	40·1–60·0	60·1–80·0	≥80·1	
Assembly	25 (24·5)	30 (29·4)	35 (34·3)	12 (11·8)	102 (100·0)
Casting	12 (25·5)	20 (42·6)	12 (25·5)	3 (6·4)	47 (100·0)
Pasting	11 (20·8)	23 (43·4)	15 (28·3)	4 (7·5)	53 (100·0)
Refining	6 (40·0)	6 (40·0)	2 (13·3)	1 (6·7)	15 (100·0)
Lead powder	3 (17·6)	8 (47·1)	6 (35·3)	—	17 (100·0)
Total	57 (24·4)	87 (37·2)	70 (29·9)	20 (8·5)	234 (100·0)

Table 5 *Number of workers whose biochemical values were over the criteria in subjects grouped by level of PbB. (Percentages shown in parentheses)*

PbB (μg/dl)	No of workers	ALAU (mg/l)		Hb (mg/dl)		CPU positive*
		5·0	10·0	13·0	12·0	
≤30·0	30	3 (10·0)	—	11 (36·7)	1 (3·3)	2 (6·7)
30·1–40·0	27	5 (18·7)	—	7 (25·9)	2 (7·4)	6 (22·2)
40·1–50·0	38	12 (31·6)	3 (7·9)	9 (23·7)	5 (13·2)	11 (28·9)
50·1–60·0	49	30 (61·2)	13 (26·5)	15 (28·6)	9 (18·4)	28 (57·3)
60·1–70·0	50	42 (84·0)	24 (48·0)	22 (44·0)	10 (20·0)	48 (96·0)
70·1–80·0	20	19 (95·0)	13 (65·0)	14 (70·0)	9 (45·0)	20 (100·0)
80·1–90·0	16	15 (93·8)	14 (87·5)	12 (75·0)	6 (37·5)	16 (100·0)
≥90·1	4	4 (100·0)	4 (100·0)	4 (100·0)	3 (75·0)	4 (100·0)
Total	234	130	71	93	45	135

*Reading from ≥ ±.

Korea and the former the critical limit of mild lead poisoning.[20]

CPU (semi-quantitative)—A positive response (±) was considered as the response level.

INTERRELATION AMONG BIOCHEMICAL DATA

Figures 1 and 2 show the changes in the mean value of PbU and ALAU with an increase in PbB. Table 6 shows the correlation coefficients and the regression equations for various biochemical tests by workplace and work duration.

Since a visual inspection of the scatter diagrams of some of the pairings in table 6 suggested that their regression lines were likely to be curvilinear or segmental, the workers were divided into two groups by PbB concentration of under 40 and over 40 μg/dl; two regression lines were obtained from each group and their slopes compared to assess their equality (table 7).

Discussion

There is little precise information about the relation between the concentration of lead in air and the blood lead concentration in subjects who are occupationally exposed.[8] Not surprisingly, only a weak correlation or no correlation would be derived from comparing these two parameters if the complicated mode of absorption of lead, individual variability, physical characteristics of the dust itself, and differences in physical activity are taken into account.[21] In this study there was no statistically significant difference between the mean blood lead concentrations by job, although the air concentrations of lead varied at each workplace; there was a significant difference in the proportion of workers with PbB of over 60 μg/dl by job. Baker *et al*[11] reported that 60–80% of the workers at three plants in the United States in which the air concentration of lead was over 200 μg/m³ showed a PbB above 60 μg/dl and in 30–70% of workers the PbB was above 80 μg/dl. The present study, which seemed to have the same working conditions as Baker's showed a relatively small proportion of men with a PbB above 60 μg/dl.

The dose-response relationship of lead on ALAU has been well presented in the WHO criteria document for lead[8] based on calculations made by Zielhuis.[9] These calculations dealt only with male workers. The present study showed results similar to those of Zielhuis[9] except for a slightly low PbB threshold level for an ALAU of 5 mg/l. If an ALAU of over 10 mg/l is considered as an unacceptable limit for lead workers the present study suggested that a PbB of 50–60 μg/dl would be the proper practical limit for biological monitoring so far as response of ALAU is concerned.

Although determination of coproporphyrin in urine is less sensitive to the effects of lead exposure than determination of ALAU, semi-quantitative testing of coproporphyrin still plays an important

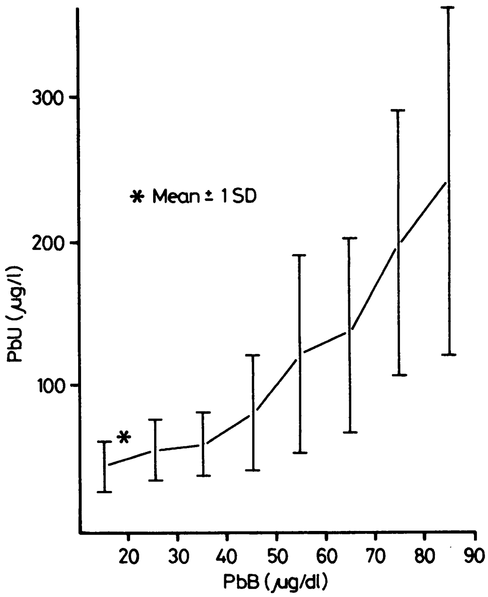

Fig 1 *Relation of PbU to PbB in groups divided by PbB concentration.*

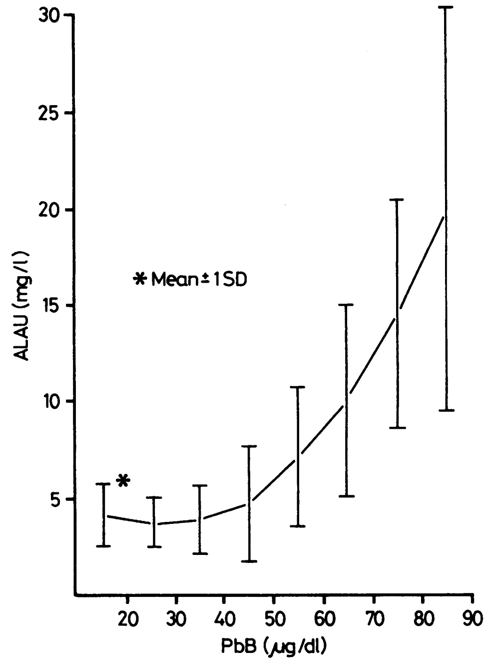

Fig 2 *Relation of ALAU to PbB in groups divided by PbB concentration.*

part when screening lead workers in some countries because of its simplicity.

The response level of CPU by increase of PbB was proportionally increased showing a similar pattern to that of ALAU. At a PbB of 60 μg/dl and above, 96% of workers responded positively. If a PbB of 60 μg/dl is considered as the critical point of unacceptable lead absorption from a practical point of view the semi-quantitative CPU test can detect nearly all workers with a PbB of over 60 μg/dl with high sensitivity (97·8%).

Anaemia is a characteristic toxic effect of lead in man, but the PbB threshold level for this effect is still not certain. Williams[22] reported that anaemia did not occur in industrial workers with a PbB below 110 μg/dl. On the other hand, Tola *et al*[23] and Baker *et al*[11] reported that the effect of lead on Hb was observed from a PbB concentration of about 50 μg/dl. Anaemia is a late sign in lead poisoning and is seldom severe unless there is concomitant iron deficiency.[7] It is difficult to interpret the anaemia of workers in the lead industry only on the basis of

Table 6 *Regression analysis of various pairings by workplace and work duration*

	*PbB v PbU**		*PbB v ALAU**		*PbB v Hb**	
	r	*Regression equation*	*r*	*Regression equation*	*r*	*Regression equation*
Work place						
I	0·65	y = 3·15x − 40·9	0·62	y = 0·23x − 4·1	−0·22	y = −0·017x + 14·1
II	0·65	y = 2·43x − 18·3	0·62	y = 0·20x − 3·3	−0·33	y = −0·021x + 14·6
III	0·60	y = 2·46x − 17·6	0·73	y = 0·28x − 6·9	−0·31	y = −0·027x + 14·3
IV	0·78	y = 2·00x − 1·8	0·70	y = 0·24x − 3·7	−0·46	y = −0·044x + 14·6
V	0·79	y = 2·44x − 38·2	0·75	y = 0·28x − 6·0	−0·61	y = −0·047x + 14·8
Work duration (years)						
<2	0·55	y = 2·18x + 1·6	0·56	y = 0·10x + 0·4	−0·35	y = −0·029x + 14·7
2–4	0·67	y = 2·73x − 34·1	0·70	y = 0·19x − 2·2	−0·18	y = −0·014x + 14·5
4–6	0·72	y = 4·15x − 100·5	0·69	y = 0·31x − 7·9	−0·34	y = −0·027x + 14·5
6>	0·45	y = 2.25x + 1·6	0·62	y = 0·38x − 13·8	−0·21	y = −0·023x + 14·4
Total	0·65	y = 2·85x − 34·2	0·65	y = 0·24x − 4·5	−0·28	y = −0·022x + 14·3

*y axis.
NS: Statistically non-significant.
I Assembly, II Casting, III Pasting, IV Refining, V Lead powder.

their lead exposure, unless other causes can be discounted.

There were no significant differences of response of Hb below 13 mg/dl between workers of PbB 30–70 μg/dl, but there was an increasing number of workers with Hb below 12 mg/dl as the PbB increased. Bearing in mind that lead can shorten red cell life and affect haem synthesis, this study suggested that the effect of lead on Hb could be identified from a PbB of 70–80 μg/dl or even of a somewhat lower concentration.

Zielhuis[14] made an intensive review of publications concerning the inter-relationship of biochemical response to the absorption of inorganic lead. According to his review, different observers found a wide variation in the relationships between various of the parameters relevant to lead poisoning.

Most studies agreed that a significant positive correlation between PbB and PbU or ALAU could be found in occupationally exposed lead workers, but they did not show agreement on the linearity of the regression line.[14]

ALA excretion in urine has long been used as a measure of a biological effect of lead, and correlates with an increase in PbB.[8] Many studies show a straight single regression trend between PbB and ALAU, but a few recent studies of this relationship in industrially exposed subjects indicate a curvilinear relationship.[14] Selander and Cramer[24] achieved a satisfactory result with log-transformation of ALAU, showing a high correlation coefficient ($r = 0{\cdot}74$) between this relationship. Haeger-Aronsen[25] also agreed with the curvilinear function of Selander and Cramer, showing a very similar result. On the other hand, King *et al*[26] suggested a second-degree regression model instead of a primary regression model.

To obtain the model that best fitted various pairings, a stepwise statistical approach was undertaken. Visual inspection of scatter diagrams and figs 1 and 2 suggested a curvilinear function or two segmental straight lines divided at a PbB of around 40 μg/dl. Statistical tests of the slopes between the regression line of the groups with PbB below and above 40 μg/dl (table 7) showed significant differences only between PbB and PbU and PbB and ALAU. Assuming that these two pairings are not well-explained in a single straight model, second-degree regression and segmental model functions (figs 3, 4) were calculated and compared by analysis of variance to determine which best fitted the scatter diagram. In both the pairing of PbB and PbU and PbB and ALAU a segmental model appeared to fit the results better than the quadratic model. These results do not support King's study[26] that a second-degree regression function is better fitted for the relation between PbB and PbU.

Selander and Cramer also expressed a segmental model of relationship between PbB and ALAU divided at a PbB of 40 μg/dl, which was the upper limit of normal in an unexposed population, but they concluded a curvilinear function from this segmental data.[24]

ALAU is a good indicator of lead intoxication and usually responds from a PbB of 30–40 μg/dl; some studies show no relationship between PbB and ALAU in subjects whose PbB was under 40 μg/dl.[8,9] Inclusion of data from workers with a PbB under 40 μg/dl would tend to make the relationship between PbB and ALAU appear curvilinear.

To test the straight trend of PbB with ALAU in workers whose PbB were above 40 μg/dl, an F test was carried out between a simple straight line and a second-degree regression line; this showed a smaller

*PbU v ALAU**		*PbU v Hb**		*ALAU v Hb**	
r	*Regression equation*	*r*	*Regression equation*	*r*	*Regression equation*
0·58	y = 0·045x + 2·8	−0·23	y = −0·0035x + 13·6	−0·35	y = −0·071x + 13·7
0·48	y = 0·042x + 2·5	NS		−0·47	y = −0·071x + 14·2
0·59	y = 0·055x + 1·8	−0·33	y = −0·0069x + 13·7	−0·57	y = −0·130x + 13·9
0·72	y = 0·095x − 1·3	−0·53	y = −0·0196x + 14·4	−0·76	y = −0·213x + 14·2
0·81	y = 0·097x − 1·4	−0·63	y = −0·0159x + 14·7	−0·63	y = −0·135x + 14·4
0·65	y = 0·029x + 2·1	−0·46	y = −0·0095x + 14·3	−0·59	y = −0·270x + 14·7
0·65	y = 0·042x + 2·6	−0·20	y = −0·0036x + 13·6	−0·48	y = −0·138x + 14·2
0·69	y = 0·053x + 2·3	−0·32	y = −0·0044x + 13·6	−0·48	y = −0·085x + 13·8
0·34	y = 0·043x + 4·8	NS		−0·40	y = −0·069x + 13·7
0·58	y = 0·048x + 2·5	−0·25	y = −0·0044x + 13·6	−0·45	y = −0·095x + 13·9

Table 7 *Regression analysis of various pairings in groups with PbB below and above 40 μg/dl*

	Correlation coefficient (r)	Regression equation	Difference between slopes*
PbB v PbU	40 ≥ 0·22 40 < 0·55	y = 0·63x + 35·7 y = 3·41x − 71·3	p < 0·05
PbB v ALAU	40 ≥ 0·01 (NS) 40 < 0·62	y = 0·01x + 3·4 y = 0·33x − 10·5	p < 0·05
PbB v Hb	40 ≥ 0·13 (NS) 40 < 0·32	y = 0·020x + 12·9 y = −0·035x + 15·2	NS
PbU v ALAU	40 ≥ 0·30 40 < 0·50	y = 0·020x + 2·4 y = 0·042x + 3·8	NS
PbU v Hb	40 ≥ −0·01 (NS) 40 < −0·23	y = −0·0003x + 13·5 y = −0·0042x + 13·6	NS
ALAU v Hb	40 ≥ 0·37 40 < 0·46	y = −0·29x + 14·5 y = −0·10x + 13·9	NS

$$*Z = \frac{b_1 - b_2}{\sqrt{SE(b_1) + SE(b_2)}}.$$

NS = Not significant.

sum of squares of the residual in the straight line than in the quadratic line. This suggests that a curvilinear trend does not exist between PbB concentrations in the range 40–100 μg/dl. In a previous study[20] the interrelation between PbB and ALAU was highly correlated (r = 0·91) and gave a straight trend rather than a curved trend. Because this study covered a wide range of PbB from below 40 μg/dl to above 200 μg/dl, the lower portion of PbB (below 40 μg/dl) did not have an important influence on the regression line. The study of Selander and Hallberg, which showed a straight line between these parameters in workers with blood leads in the range 40–130 μg/dl, supports this concept.[27] Figures 3 and 4 show the differences of lines of various approaches. Curvilinear data seem to be underestimated in PbB below 60 μg/dl and overestimated from that level compared with the straight line.

ALAU response is determined not only by the blood lead but also by the duration of exposure.[24] At a given blood lead concentration, the ALAU of workers tended to increase by duration of exposure (table 3). This tendency was confirmed by comparing the slope of regression line of PbB with ALAU (table 6). The slope of the regression was steeper as work duration of workers increased. This could be explained by the chronic effect of lead on haem precursors.

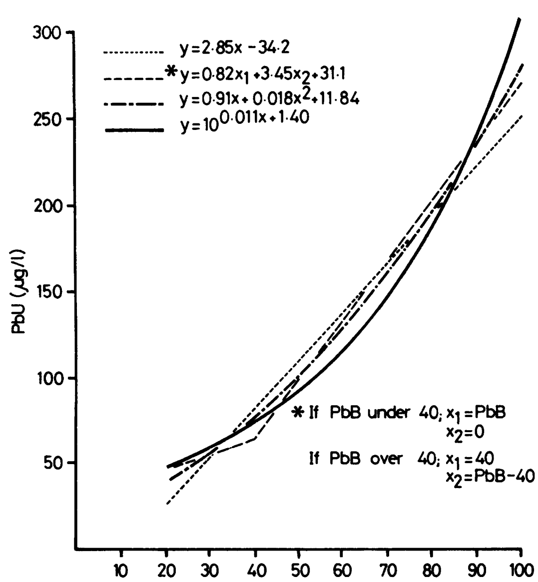

Fig 3 *Regression equation of PbB with PbU in various functions.*

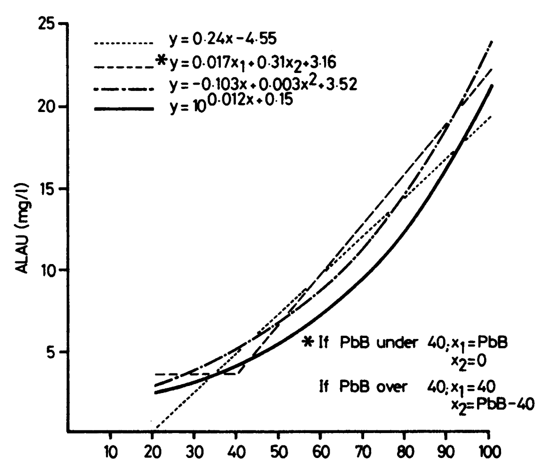

Fig 4 *Regression equation of PbB with ALAU in various functions.*

Waldron[13] reported no correlation between ALAU and Hb with 137 lead workers. On the other hand, Gibson *et al*[28] reported a strong correlation (r = 0·57) between these parameters. In the present study Hb concentration was fairly correlated with ALAU (r = − 0·45). Relative steepness of slope of regression between ALAU and Hb was shown in workers whose work duration was under two years compared with other workers with a period of long service. This could be partly explained by the acute effect of lead on haem synthesis in newly introduced workers.[23]

I am extremely grateful to Dr H A Waldron of London School of Hygiene and Tropical Medicine for his guidance and help in preparing this paper. I am also grateful to Dr D Oakes of the same school for his valuable statistical advice and to Mrs M Whittingham for her excellent computing help.

References

1. Hammond PB. Human health implication. In: Boggess WR, Wixson BG, eds. *Lead in the environment.* Tunbridge Wells, Kent: Castle House Publications Ltd, 1979:195–8.
2. Malcolm D. Prevention of long-term sequalae following the absorption of lead. *Arch Environ Health* 1971;**23**:292–8.
3. Joselow MM, Flores J. Application of the zinc protoporphyrin (ZP) tests as a monitor of occupational exposure to lead. *Am Ind Hyg Assoc J* 1977;**38**:63–6.
4. Hanninen H, Hernberg S, Mantere P, Vesanto R, Jalkanen M. Psychological performance of subjects with low exposure to lead. *JOM* 1978;**20**:683–9.
5. Araki S, Honma T. Relationships between lead absorption and peripheral nerve conduction velocities in lead workers. *Scand J Work Environ Health* 1976;**4**:225–31.
6. Guinee VF. Lead poisoning. *Am J Med* 1972;**52**:283–8.
7. Waldron HA. *Metals in the environment.* London: Academic Press, 1980:155–97.
8. World Health Organisation. *Environmental health criteria: 3. Lead.* Geneva: World Health Organisation, 1977.
9. Zielhuis RL. Dose-response relationships for inorganic lead. *Int Arch Occup Environ Health* 1976;**35**:1–18.
10. Zielhuis RL. Second international workshop permissible levels for occupational exposure to inorganic lead. *Int Arch Occup Environ Health* 1977;**39**:59–72.
11. Baker EL, Landrigan PJ, Barbour AG, *et al.* Occupational lead poisoning in the United States: clinical and biochemical findings related to blood lead levels. *Br J Ind Med* 1979;**36**:314–22.
12. Williams MK. Permissible limits for occupational exposure to inorganic lead and the blood lead-air lead relationship. *Int Arch Occup Environ Health* 1978;**41**:151–7.
13. Waldron HA. Correlation between some parameters of lead absorption and lead intoxication. *Br J Ind Med* 1971;**28**:195–9.
14. Zielhuis RL. Interrelationship of biochemical responses to the absorption of inorganic lead. *Arch Environ Health* 1971;**23**:299–311.
15. de Bruin A, Hoolboom H. Early signs of lead exposure. A comparative study of laboratory tests. *Br J Ind Med* 1967;**24**:203–11.
16. Jacobs MB, Herden J. Simplified one colour dithizone method for lead in urine. *Am Ind Hyg Assoc J* 1961;**22**:372–6.
17. Kehoe RA. Industrial lead poisoning. In: Patty FA, ed. *Industrial hygiene and toxicology.* New York: Interscience Publishers Inc, 1963:941.
18. Tomokuni K, Ogata M. Simple method for determination of urinary δ-aminolevulinic acid as an index of lead exposure. *Clin Chem* 1972;**18**:1534–6.
19. de Kretser AJ, Waldron HA. Urinary delta aminolaevulinic acid and porphobilinogen in lead-exposed workers. *Br J Ind Med* 1963;**20**:35–40.
20. Lee BK. Urinary excretion of δ-aminolevulinic acid in lead workers. *Journal of Catholic Medical College* 1973;**24**:485–92.
21. Stankovic MK. Biochemical tests for the appraisal of exposure to lead. *Arch Environ Health* 1971;**23**:265–9.
22. Williams MK. Blood lead and haemoglobin in lead absorption. *Br J Ind Med* 1966;**23**:105–10.
23. Tola S, Hernberg S, Asp S, Nikkanen J. Parameter indicative of absorption and biological effect in new lead exposure: A prospective study. *Br J Ind Med* 1973;**30**:134–41.
24. Selander S, Cramer K. Interrelationships between lead in blood, lead in urine, and ALA in urine during lead work. *Br J Ind Med* 1970;**27**:28–39.
25. Haeger-Aronsen B. An assessment of the laboratory tests used to monitor the exposure of lead workers. *Br J Ind Med* 1971;**28**:52–8.
26. King E, Conchie A, Hiett D, Milligan B. Industrial lead absorption. *Ann Occup Hyg* 1979;**22**:213–39.
27. Selander S, Hallberg L. Studies in lead poisoning. Oral therapy with penicillamine; relationship between lead in blood and other laboratory tests. *Br J Ind Med* 1966;**23**:282–91.
28. Gibson SLM, Mackenzie JC, Goldberg A. The diagnosis of industrial lead poisoning. *Br J Ind Med* 1968;**25**:40–51.