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Interrelationships between lead in blood, lead in urine, and ALA in urine during lead work

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Selander, S., and Cramér, K. (1970). *Brit. J. industr. Med.*, **27**, 28-39. **Interrelationships between lead in blood, lead in urine and ALA in urine during lead work.** One hundred and seventy-seven workers from a storage battery factory were examined for lead in blood and lead and δ-aminolevulinic acid (ALA) in urine. The workers were selected at random from those who had been employed for more than one month; most had been employed for several years at the same job. Thirty-six workers were from departments with no lead exposure. In three departments with high exposure a rotating system with three weeks' exposure and three weeks' non-exposed work was applied. As the aim of the study was to establish the relationships between the three parameters during constant exposure, the values from these men were treated separately.

The relationship between lead in blood and urinary ALA was best described by a curvilinear function: $ALA = 10^{0.0157\ Pb_b - 1.0985}$, while the regression lines for ALA on lead in urine, and lead in urine on lead in blood were straight.

Workers from the departments with the rotating system showed lower values for urinary lead and ALA, compared with non-rotating workers with the same level of lead in blood. All these workers were examined during their second or third week of lead work, *i.e.*, with an accumulating lead body burden. This system may be beneficial, especially in departments where prophylactic measures are difficult to install, or for notoriously careless workers.

Those who showed comparatively high ALA and urinary lead values in relation to their blood lead level were found to be workers with repeated incidents of metabolic lead influence, in whom the ALA values had seldom been normal.

The mean values from different factory departments were of the same order as would be expected from previous studies in storage battery plants.

The results are discussed in relation to present concepts of lead absorption and poisoning.

Determinations of lead in blood, lead in urine, and δ-aminolevulinic acid (ALA) in urine are considered to be three of the most reliable tests used in the control of lead-exposed workers. Several investigations concerning the relationship between these parameters and their merits as measures of lead absorption and lead poisoning have been published. Discrepancies between the results can often be explained by differences with regard to the materials and the plan of investigation, and the results are often given in such a way that it is difficult to make direct comparisons. These laboratory analyses are also all somewhat elaborate and some of the published results may therefore be questioned. However, in well-planned investigations performed under standardized conditions a definite correlation between the three parameters has always been shown

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(Haeger-Aronsen, 1960; Cramér and Selander, 1965; Selander, Cramér, and Hallberg, 1966; de Bruin and Hoolboom, 1967; Stopps, 1968; Williams, King, and Walford, 1969).

For the routine control of a large lead industry it is not possible to achieve such standardized conditions. The employment periods of the workers vary, and it is very difficult to obtain genuine 24-hour urine samples or repeated collections of blood samples for several days. These are only some of the factors that influence the results. Nevertheless, it is of practical importance to clarify the relationships between the biochemical tests during such routine conditions and to compare the results with those obtained during more standardized conditions. This has been the principal aim of the present study.

Material and methods

The investigation was carried out in a storage battery factory with a total of 444 employees and included 177 workers, in 16 different departments (Appendix Table), in which work is largely independent of one another. In one department there was no lead exposure, except for the general atmosphere in the factory area. The selection of the workers included in the study was random, with the exception that they must have been employed for at least one month. The lead workers had been, in general, exposed for several years (Cramér and Dahlberg, 1966) and only a few for less than half a year.

Three departments, *i.e.*, tube filling, ball-mills, and paste mixing, differed fundamentally from the others, because a rotating system was applied so that after a three-week period the worker was transferred for the following three weeks to a department without lead work. These workers were always examined during the second or third week of lead work, *i.e.*, during an increasing accumulated lead exposure. The results from these three departments have been treated in part separately. With this exception no change of working place was allowed for the month preceding the study. The weekly working time was the same for all workers, that is 45 hours. The blood and urine samples were collected in the same way as for the ordinary routine control, *i.e.*, at the end of the week (Thursday or Friday) and always at the same time of day, between 9.30 and 11.30 in the morning. To avoid contamination of the blood samples the workers undressed the upper part of the body and washed before the samples were drawn. An infallible avoidance of contamination of the urine samples is more difficult to achieve during routine control conditions. The workers were, however, carefully instructed to wash their hands thoroughly before the samples were delivered in small-necked plastic bottles.

Blood lead and urinary lead were analysed by atomic absorption spectrophotometry (Selander and Cramér, 1968a, b). ALA in urine was measured by the method of Mauzerall and Granick (1956). For the urine analyses no corrections were made for specific gravity or creatinine concentration (Cramér and Selander, 1967). All determinations were performed in duplicate and the mean figures are given in the Appendix Table.

Results

Individual values

The individual results within each department are shown in the Appendix Table and are also given in Figs. 1 to 3, with the exception of those from the departments with the rotating system.

Figure 1 illustrates the relationship between lead in blood and ALA in urine. The dotted lines mark the upper normal limits, 0·6 mg/100 ml urine for ALA and 40 μg/100 ml blood for lead (Lane *et al.*, 1968). The latter limit can be a matter of some dispute but it is generally accepted. In the present study it may be regarded as a border line between two populations of workers, one with a lead exposure which did not cause a metabolic dysfunction, *i.e.*, a pathological ALA value, and the other with an exposure high enough to give pathological ALA values. A single straight regression line ($ALA = 0{\cdot}024\ Pb_b - 0{\cdot}479$) for the whole population of workers did not appear satisfactory, and on visual inspection the scatter of the points suggested that it might be possible to find a satisfactory common curvilinear regression line for the whole material. After logarithmic transformation of the ALA values a straight regression line was obtained with the equation: $\log ALA = 0{\cdot}0157\ Pb_b - 1{\cdot}0985$ ($S_b = 0{\cdot}0012$; $S_{yx} = 0{\cdot}2557$; $r = 0{\cdot}74$) and thus a curvilinear regression line, $ALA = 10^{0{\cdot}0157\ Pb_b - 1{\cdot}0985}$, could be drawn as seen in Figure 1.

In Fig. 2 are plotted the values for lead in blood and lead in urine. Here it was not possible to discern two populations as in the case of ALA in urine versus lead in blood. In fact, several of the workers with a blood lead level below 40 μg/100 ml showed a considerably elevated lead excretion in the urine, if 3·2 μg/100 ml was considered as the upper limit of normal (Haeger-Aronsen, 1960). The relationship between the two variables could be described by the straight regression line: $Pb_u = 0{\cdot}265\ Pb_b - 3{\cdot}305$ ($S_b = 0{\cdot}024$; $S_{yx} = 5{\cdot}437$; $r = 0{\cdot}66$).

Figure 3 shows the relationship between ALA and lead in urine. Although both these variables are dependent on the lead in blood, it seems justifiable to consider lead in urine as the independent variable because the lead excretion is a more direct effect of the blood lead level, while the ALA excretion reflects the metabolic effect on the haem synthesis caused by lead.

With lead in urine as the independent variable the relationship was described by the equation: $ALA = 0{\cdot}069\ Pb_u + 0{\cdot}013$ ($S_b = 0{\cdot}005$; $S_{yx} = 0{\cdot}450$; $r = 0{\cdot}75$).

The individual values from the departments with the rotating system were not plotted in diagrams but are shown in the Appendix Table. Most of these ALA values seemed to be rather low in relation to the lead levels in the blood. In Fig. 1 the curvilinear regression line intersects the upper normal limit for ALA in

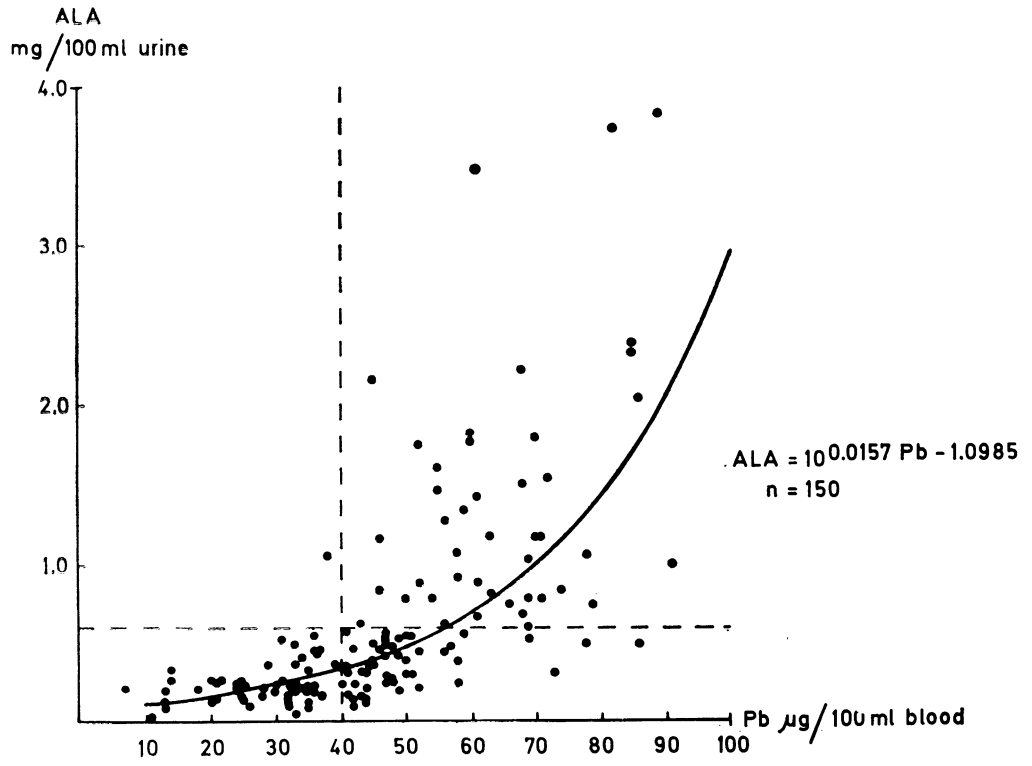

FIG. 1. Values for ALA in urine plotted against those for lead in blood. The dotted lines mark the upper normal limits.

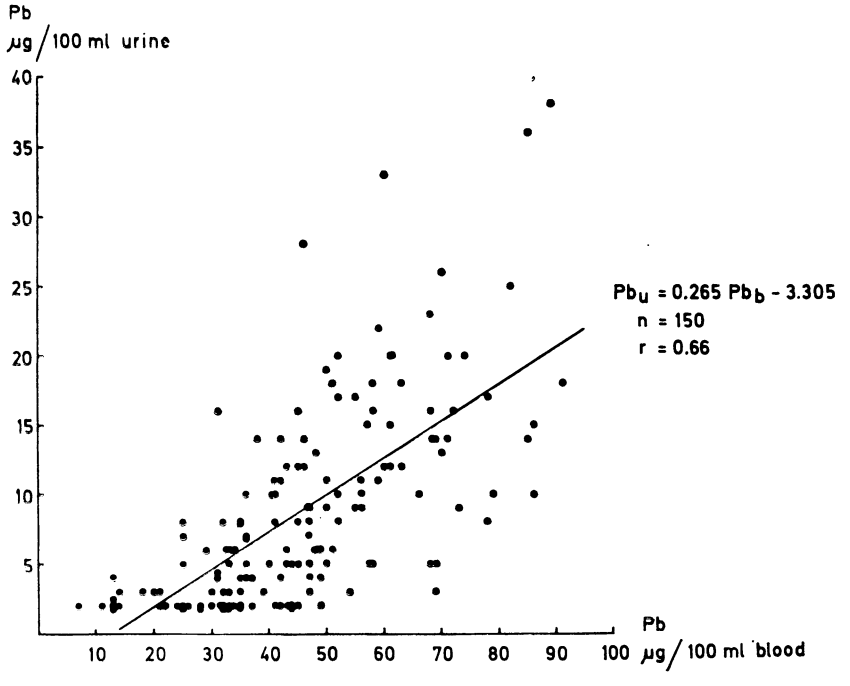

FIG. 2. Values for lead in urine plotted against those for lead in blood.

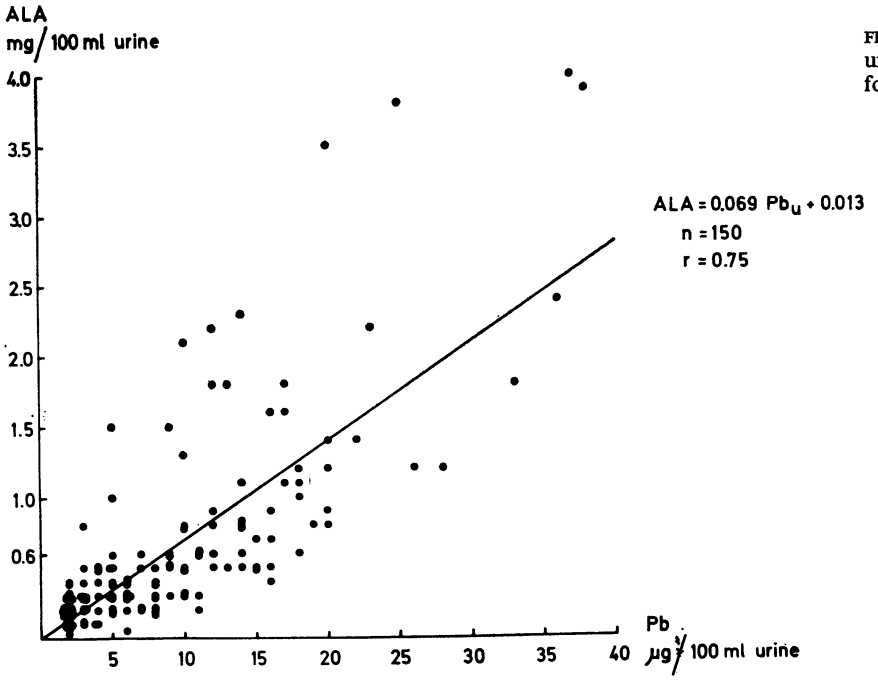

FIG. 3. Values for ALA in urine plotted against those for lead in urine.

urine at the blood lead level of 55 µg/100 ml. For this and higher lead values there were 10 normal and 35 pathological ALA values. Corresponding figures for the departments with the rotating system were 10 normal and only seven pathological ALA values. The difference was statistically significant ($P < 0.01$), applying the two tail Chi square test. The mean value for lead in blood was the same in both groups (68 µg/100 ml). With the rotating system of work, the values for lead in urine also seemed to be lower for comparable levels of blood lead. A similar calculation could not be applied because of the low upper normal limit for lead in urine. However, the mean values for lead in urine were estimated and compared. For the departments with the rotating system the value was 11 µg/100 ml, S.D. = 7·8, and for the other group it was 15 µg/100 ml, S.D. = 7·7. The difference was not statistically significant.

Department mean values

The mean values for the different departments are given in the Appendix Table and are plotted in Figures 4 and 5. The values for the departments with the rotating system are also plotted but are not included in the regression calculations.

As with the individual values, the group values for ALA in urine and lead in blood could be divided into two populations, one with lead levels high enough to give a pathological ALA response (> 0·6 mg/100 ml) and one with lower lead levels, giving mean values for ALA within the normal range. The scatter of the points suggested a curvilinear regression line. Logarithmic transformation of the ALA values yielded a straight regression line with the equation: $\log ALA = 0{\cdot}0231 Pb_b - 1{\cdot}3496$ ($S_b = 0{\cdot}0024$; $S_{yx} = 0{\cdot}0877$; $r = 0{\cdot}94$), and the curvilinear line, $ALA = 10^{0{\cdot}0231\ Pb_b - 1{\cdot}3496}$, could be drawn as in Figure 4.

For lead in urine and lead in blood the relationship, as for the individual values, could be described by a straight regression line: $Pb_u = 0{\cdot}406\ Pb_b - 9{\cdot}571$ ($S_b = 0{\cdot}066$; $S_{yx} = 2{\cdot}398$; $r = 0{\cdot}88$) (Fig. 5). This was also found for ALA and lead in urine, $ALA = 0{\cdot}084\ Pb_u - 0{\cdot}154$ ($S_b = 0{\cdot}012$; $S_{yx} = 0{\cdot}200$; $r = 0{\cdot}90$). The mean values for ALA and lead in urine in the departments with the rotating system also seemed to be lower than in the other departments with corresponding blood lead levels. The values in the departments within the blood lead regions 50-60 and 60-70 µg/100 ml were pooled and their means were compared using Student's *t* test. Within the first region the ALA values were significantly different, but the values for lead in urine were not. The higher region showed the reverse.

Discussion

In our previous studies (Cramér and Selander, 1965; Selander *et al.*, 1966) the subjects were workers with symptoms and signs of lead poisoning of varying degrees, and the investigations were performed at least one week after the last exposure to lead. The

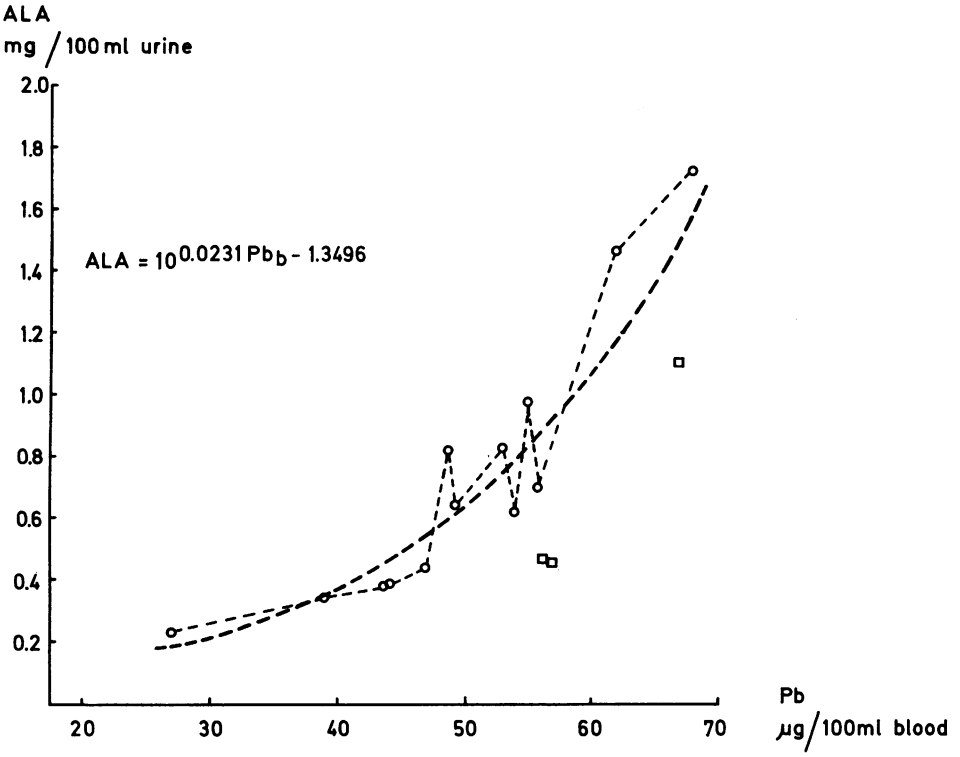

FIG. 4. Department mean values for ALA in urine plotted against those for lead in blood. The squares indicate the departments with a rotating system, which are not included in the regression calculations.

workers were also hospitalized and thus it was possible to obtain quantitative 24-hour urine samples. Accordingly, complete agreement between those results and the present ones is not to be expected, but a comparison may be of interest.

The investigation of Williams *et al.* (1969) from a storage battery factory is the one most comparable with ours, even though it included far fewer workers and was carried out under more standardized conditions. The range of the blood lead values, however, was the same as that of the present study. The laboratory results were given as the mean values of daily analyses during one week, and lead in air was also estimated using personal samplers.

Individual values

ALA in urine versus lead in blood In a previous study we found a linear relationship between ALA in urine and lead in blood (ALA = 0·076 Pb_b − 2·179) within the blood lead region 40-130 µg/100 ml (Selander *et al.*, 1966). In the present investigation this also holds true for the population of workers with a blood lead level of 40 µg/100 ml or higher (Fig 6) but here the slope of the regression line (ALA = 0·033 Pb_b − 1·037) was not as steep, *i.e.*, a given blood lead level corresponds to a lower ALA level. This difference can be explained if the blood lead level after interrupted exposure decreases faster than the ALA excretion. In this investigation the far greater spread of the ALA values at higher blood lead levels is explained by the nature of this study, which was performed during routine conditions. It was impossible to know for how long a worker had maintained a given blood lead level, and obviously the ALA response is determined not only by the blood lead value but also by the duration of exposure.

For the workers with blood lead levels below 40 µg/100 ml the relationship between ALA in urine and lead in blood was also linear (ALA = 0·007 Pb_b + 0·062) (Fig. 6). The regression lines for the two populations were statistically significantly different and were nearly tangential to the common curvilinear regression line in Figure 1. This further strengthens the assumption that it is not correct to assume a straight linear relationship over a wide range of values for blood lead and urinary ALA.

A feature common to those workers with particularly high ALA values in relation to their

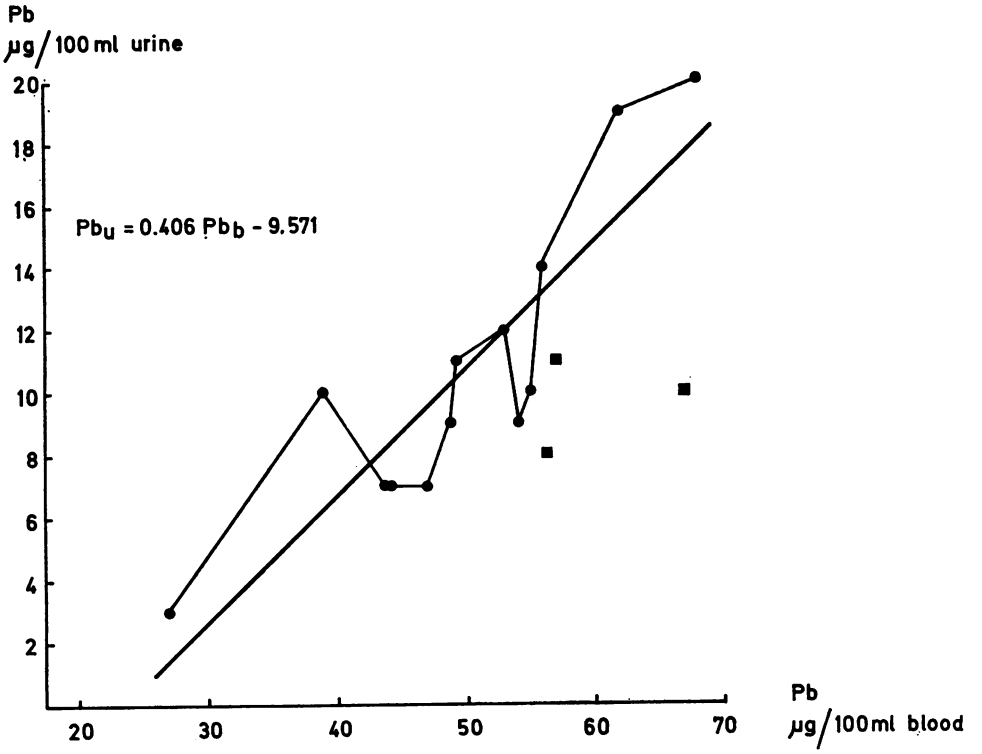

FIG. 5. Department mean values for lead in urine plotted against those for lead in blood. The squares indicate the departments with a rotating system, which are not included in the regression calculations.

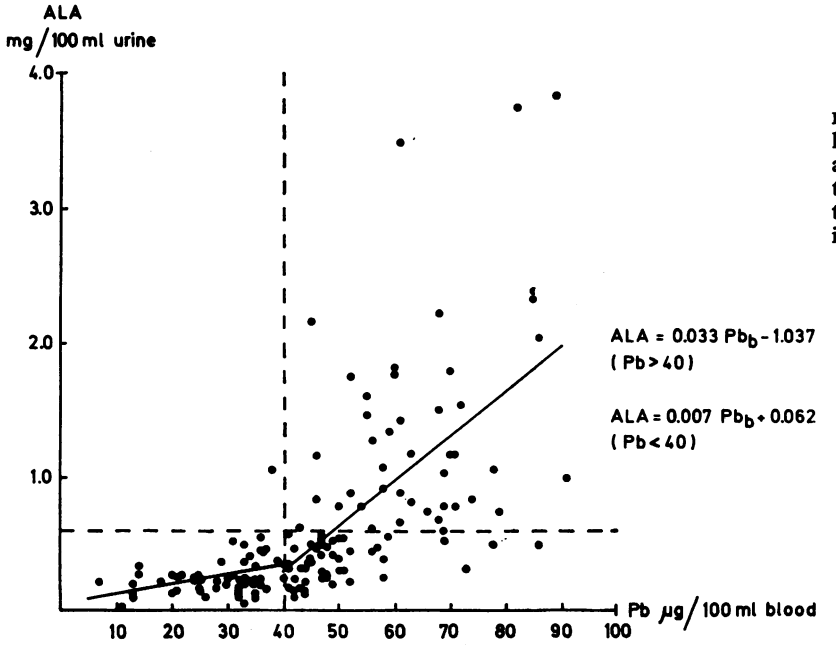

FIG. 6. ALA in urine versus lead in blood. Regression lines are drawn for the two populations of workers separated by the upper normal limit for lead in blood.

blood lead levels was that several times during the last few years they had shown signs of a metabolic influence of lead and that their ALA excretion had seldom been allowed to fall to normal values. Consequently, one ought to attach great importance to high or increasing ALA values, even if the blood lead levels are not correspondingly high. Furthermore, if a worker has been removed from lead-exposed work, he must not be allowed to return until the ALA excretion is quite normal.

Williams *et al.* (1969) expressed the relationship between ALA and lead in blood with a single straight line (ALA = 0·0321 Pb_b − 0·486). This line is quite comparable with our corresponding, but rejected, regression line (ALA = 0·024 Pb_b − 0·479). The authors did not give the individual results, but the spread of the points in the diagram suggests the possibility of a curvilinear regression line, which would better reflect the relationship between the variables.

From the same diagram it is also evident that the ALA values for the workers not exposed to lead were higher than our corresponding values. This probably results from the fact that these authors used a simplified method for the ALA analyses (Williams and Few, 1967), which gives somewhat higher values because it does not eliminate the porphobilinogen content of the samples.

Lead in urine versus lead in blood A linear relationship between lead in urine and lead in blood was found for blood lead values of 40 μg/100 ml or higher (Pb_u = 0·288 Pb_b − 4·568) as well as for the whole material. These two regression lines were almost identical. A straight regression line (Pb_u = 0·563 Pb_b − 18·204) can also be calculated from the figures in our earlier investigation (Selander *et al.*, 1966). But here also, as for ALA versus lead in blood, the slopes of the regression lines in the two studies were not the same. This means that in the present study, carried out during lead exposure, a given lead level in blood corresponded to a lower lead excretion in urine.

An explanation analogous to that proposed for ALA versus lead in blood is probable, *i.e.*, the blood lead values drop faster than the urinary lead values after cessation of the lead exposure. The regression line for the present investigation (Pb_u = 0·265 Pb_b − 3·305) closely corresponded to that found in the investigation of Williams *et al.* (1969), (Pb_u = 0·246 Pb_b − 3·54), which was also performed during lead-exposed work.

ALA in urine versus lead in urine The regression line (ALA = 0·069 Pb_u + 0·013), describing the relationship between ALA and urinary lead in the present study, was almost parallel to that (ALA = 0·078 Pb_u+1·201) which can be calculated from our earlier study (Selander *et al.*, 1966), but it is displaced so that a given lead value corresponds to an ALA value which is about 1·2 mg/100 ml lower than in the former study, where the subjects were investigated some time after cessation of exposure.

This difference may depend on the composition of the two groups, the earlier one consisting of workers, imperfectly controlled, with symptoms of lead poisoning, the present one consisting of well-controlled workers without symptoms. Probably the lead excretion decreases faster than the ALA excretion after cessation of exposure (Stopps, 1968), which may contribute to the different results. As can be seen in Fig. 3, there were a number of workers showing especially high ALA values in relation to their urinary lead excretion. These workers were the same as those who had especially high ALA values in relation to their blood lead levels, *i.e.*, those workers who had several times shown signs of a metabolic influence of lead and who had an ALA excretion which was seldom allowed to fall to normal values. Haeger-Aronsen (1960), de Bruin and Hoolboom (1967), and Williams *et al.* (1969) have reported regression lines with steeper slopes than ours. However, the first two investigations consisted exclusively of workers with a lead excretion higher than normal. As mentioned before, the ALA method used by Williams, King, and Walford (Williams and Few, 1967) gives somewhat higher values than the original method of Mauzerall and Granick (1956), used by us, and it is possible that the difference is more pronounced the higher the ALA values are, since the porphobilinogen excretion in urine may be moderately increased with increasing ALA excretion (de Bruin and Hoolboom, 1967). Stopps (1968) reported a curvilinear relationship between ALA and lead in urine. His material was, however, to some extent different from ours and from those previously referred to, as it included a number of workers with remarkably high ALA values, up to 16 mg/100 ml, and these high values are probably responsible for the curvilinearity of the regression line. The great spread of the ALA values at higher urinary lead levels may be explained by differences between the workers with regard to the duration of a certain exposure. That is analogous to the explanation given for the spread of the ALA values at higher blood lead levels in our present report.

de Kretser and Waldron (1963) considered that they could not find a positive correlation between the urinary excretion of ALA and lead. Despite that, the diagram in the paper gives the impression of a positive correlation between the variables. Unfortunately, the authors did not give the results of any correlation or regression calculations, nor the individual values. Their conclusion that 'a raised ALA' urinary lead was always associated with a raised ALA, is hard to understand. It is correct only if one is

willing to accept the authors' upper normal limit for lead in urine, *i.e.*, 20 μg/100 ml. However, most workers in this field consider a lead excretion of about 20 μg/100 ml urine to be excessive (Lane *et al.*, 1968).

Department mean values

Reports of department mean values are not common. Haeger-Aronsen (1960) found the highest ALA values in plate finishing, assembly, pasting + oxide mixing, and plate forming, while the mean values were within normal limits in lead storage, casting, and charging. Tsuchiya and Harashima (1965) reported great variations of the lead concentration in air between different departments, while despite this the lead excretion in urine was roughly of the same magnitude, 15·2-16·5 μg/100 ml.

The same variations of the exposure between different departments were also reported by Williams *et al.* (1969). The highest mean values were found in machine pasting and hand pasting + forming but were considerably lower in casting. They also found, however, that the mean values for lead in blood and for lead and ALA in urine showed the same pattern as those for lead in air, even if the ALA value in hand pasting was lower than expected in relation to the other indices. There were large individual variations of exposure and response between workers in the same departments.

A comparison between factories is difficult because of the variations in the hygienic standards, but the investigations referred to give a similar indication of those departments which offer the greatest lead hazards in a storage battery factory. The results are also in fairly good agreement with ours.

Above all, these department mean values give information about the conditions in a given factory and indicate where preventive measures should first be initiated. It is evident from Figs. 4 and 5 that two departments, scrap metal works and machine pasting, had unacceptable mean values for the excretion of both lead and ALA. The mean values for lead in blood were between 60 and 70 μg/100 ml. These values indicate strongly that both departments should be subject to preventive measures. In the departments with a mean value for lead in blood of up to 47 μg/100 ml, the mean excretion of lead and ALA was quite acceptable and there were only isolated cases showing a slightly raised ALA excretion, indicating a metabolic influence of lead. These departments were: no exposure, grid sawing, grid casting, submarine assembly, and assembly line I. Between these two groups were a number of departments with mean values for lead in blood of about 50 μg/100 ml and up to 60 μg/100 ml. This zone showed a greater instability with regard to the excretion of lead and ALA. Even if the metabolic influence on a certain group as a whole was fairly slight, there were always several individuals who showed unacceptable ALA values, and in the departments of this zone the control of the individual worker must be sharpened. The rotating system, used in three of the departments, had a good effect, producing a tendency to lowered excretion values for lead and ALA for the degree of exposure, expressed as the lead concentration in blood. In all probability this was due to an excretion of the easily accessible lead of the body during the three-week periods the workers spent in departments without lead exposure, and an accumulating lead body burden during exposure. Longitudinal studies of these workers and of workers with a constant lead exposure would be of value and would give information on how to prevent the metabolic influence of lead in a consistent way. The rotating system, or modifications of it, must, however, not be allowed to replace other hygienic measures, although it appears to have advantages, especially where good prophylaxis is difficult to install, or for notoriously careless workers.

Limit values It is of particular interest to relate the results presented here to recommended or agreed limit values.

From January 1 1967, in Sweden these are as shown in Table 1.

In November 1968, a statement on the diagnosis of inorganic lead poisoning was published in the *British Medical Journal* signed by several workers in this field. The different limit values are given Table 2.

Some days later, in November 1968, a conference on inorganic lead was arranged in Amsterdam with participants from several countries in Europe and from the United States of America. The aim of the conference was, among other things, to discuss the interrelationships between different biochemical

TABLE 1

	Acceptable	*Acceptable for work but with precaution*	*Unacceptable*
Blood lead	< 50 μg/100 ml	50-70 μg/100 ml	> 70 μg/100 ml
Urinary ALA	< 1·5 mg/100 ml	1·5-2·5 mg/100 ml	> 2·5 mg/100 ml

TABLE 2

	Normal	*Acceptable*	*Excessive*	*Dangerous*
Blood lead (μg/100 ml)	< 40	40 - 80	80 - 120	> 120
Urinary lead (μg/100 ml)	< 8	8 - 15	15 - 25	> 25
Urinary CP (μg/100 ml)	< 15	15 - 50	50 - 150	> 150
Urinary ALA (mg/100 ml)	< 0·6	0·6 - 2	2 - 4	> 4

indices of lead absorption and to settle permissible limit values for these indices. It was concluded that the upper limits for acceptable lead absorption or acceptable effects of such absorption were:

Blood lead	70 μg/100 ml
Urinary lead	13 μg/100 ml
Urinary CP	30 μg/100 ml
Urinary ALA	1·0 mg/100 ml
Lead in air	150 μg/m³

It was emphasized that these figures should be used as guides and that they do not indicate the limit between safe and unsafe. Because of the wide confidence limits of the calculations no undue emphasis should be placed on a single index.

These various limit values must be a matter of debate. For example, it is open to doubt if there is any real difference between groups 3 and 4 in the statement given in the *British Medical Journal*. If a lead exposure is considered excessive, then it ought to be dangerous, too. However, on the whole, there is good agreement between the statement in the *British Medical Journal* and the conclusion reached at the Amsterdam conference, even if the acceptable limit values, agreed upon at the latter, are somewhat more restrictive, especially with regard to the ALA limit value.

There is also agreement with the Swedish limit between acceptable and unacceptable values for lead in blood, while the Swedish ALA limit seems somewhat high.

As can be seen from the regression lines in Figs. 1 and 2, our results show that the relationships between lead in blood, lead in urine, and ALA in urine were in good agreement with the conclusions reached at the Amsterdam conference: a blood lead concentration of 70 μg/100 ml corresponded to an ALA excretion of 1·1 mg/100 ml. and a urinary lead excretion of 15 μg/100 ml. This is valid for individuals. For groups of workers these limit values are too high to be safe. Figures 4 and 5 show that a group mean value for lead in blood of about 70 μg/100 ml gave unacceptable mean values for ALA and urinary lead, and even in the departments where the mean values for lead in blood were between 50 and 60 μg/100 ml and where the mean values for ALA and urinary lead were lower than the tolerable limits for individuals, there were several workers with unacceptable ALA and urinary lead values.

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APPENDIX TABLE

(a) VALUES FOR MEN IN DEPARTMENTS WITHOUT ROTATING SYSTEM

Worker no.	*Lead in blood (μg/100 ml)*	*Lead in urine (μg/100 ml)*	*ALA in urine (mg/100 ml)*
No exposure (36 workers)			
4	20	3	0·13
6	36	7	0·56
16	28	2	0·17
59	24	2	0·22
86	21	3	0·25
94	25	2	0·15
122	33	6	0·24
188	14	3	0·34
225	20	2	0·27
234	33	2	0·37
237	35	2	0·13
268	13	2	0·13
286	32	3	0·22
297	25	7	0·22
318	24	3	0·25
322	18	3	0·22
327	7	2	0·22
335	29	6	0.37
354	22	2	0·27
365	33	2	0·25
373	28	2	0·22
374	14	2	0·27
446	33	6	0·05
463	30	3	0·25
478	40	5	0·25
492	35	2	0·20
520	11	2	0·03
523	45	8	0·37
561	42	4	0·10
575	26	2	0·10
605	13	4	0·10
784	34	2	0·22
794	32	2	0·15
901	47	9	0·53
960	47	3	0·30
983	13	2	0·20
Median	28	3	0·22
Mean	27	3	0·23
S.D.	10	2	0·11
Grid sawing (4 workers)			
51	32	8	0·17
383	47	8	0·42
680	25	5	0·22
757	51	18	0·55
Median	47	8	0·42
Mean	39	10	0·34
S.D.	12	6	0·18
Grid casting (9 workers)			
31	25	2	0·17
67	36	7	0·45
163	52	10	0·45
208	57	15	0·48
215	32	2	0·13
240	61	12	0·89
406	37	4	0·47
506	48	6	0·27
906	44	2	0·13
Median	44	6	0·45
Mean	44	7	0·38
S.D.	12	5	0·24
Submarine assembly (10 workers)			
35	35	8	0·20
41	44	5	0·15
98	34	6	0·42
156	32	2	0·10
233	58	16	0·92
277	69	5	1·04
298	36	5	0·25
402	48	6	0·25
490	48	13	0·48
508	35	3	0·10
Median	44	6	0·25
Mean	44	7	0·39
S.D.	12	4	0·34
Assembly line I (18 workers)			
10	44	2	0·22
23	36	10	0·45
42	52	8	0·22
60	43	5	0·17
61	33	5	0·50
83	31	4	0·27
197	66	10	0·75
206	49	2	0·20
209	61	15	0·67
345	30	2	0·20
397	56	9	0·45
398	49	4	0·53
657	78	8	0·50
877	54	3	0·79
926	46	14	0·84
1 141	35	8	0·34
1 148	41	8	0·17
1 153	41	11	0·58
Median	46	8	0·45
Mean	47	7	0·44
S.D.	13	4	0·23
Assembly line II (8 workers)			
1	58	5	0·39
43	33	3	0·20
295	43	12	0·63
1 120	52	20	0·89
1 122	70	13	1·80

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APPENDIX TABLE (continued)

(a) VALUES FOR MEN IN DEPARTMENTS WITHOUT ROTATING SYSTEM

Worker no.	*Lead in blood (μg/100 ml)*	*Lead in urine (μg/100 ml)*	*ALA in urine (mg/100 ml)*
1 136	31	16	0·53
1 166	21	2	0·15
1 168	86	15	0·50
Median	52	13	0·53
Mean	49	11	0·64
S.D.	22	7	0·53
Forming (6 workers)			
189	45	12	2·17
278	71	14	0·79
384	39	3	0·38
385	58	18	1·08
423	37	2	0·17
820	44	2	0·32
Median	45	12	0·79
Mean	49	9	0·82
S.D.	13	7	0·74
Soldering (9 workers)			
74	42	2	0·15
193	41	10	0·32
262	50	19	0·79
266	52	17	1·76
291	49	6	0·42
448	85	14	2·34
538	56	11	0·63
703	59	11	0·56
1 161	42	14	0·47
Median	50	11	0·56
Mean	53	12	0·83
S.D.	14	5	0·73
Casting (17 workers)			
34	46	12	0·47
37	56	10	1·28
62	71	20	1·18
118	86	10	2·05
142	45	2	0·38
276	41	10	0·34
359	68	16	0·68
422	69	3	0·53
425	42	11	0·24
427	63	12	0·82
440	50	9	0·55
485	47	7	0·24
524	50	11	0·30
528	50	5	0·39
562	47	9	0·56
658	35	4	0·24
828	58	5	0·25
Median	50	10	0·53
Mean	54	9	0·62
S.D.	13	5	0·48
Gridsmearing (9 workers)			
15	91	18	1·00
160	43	6	0·32
181	68	5	1·51
252	45	5	0·50
272	61	20	1·43
281	60	12	1·78
349	25	8	0·27
529	47	5	0·55
806	55	9	1·47
Median	55	8	1·00
Mean	55	10	0·98
S.D.	18	6	0·58
Manual assembly (7 workers)			
14	45	16	0·37
107	78	17	1·06
173	38	14	1·06
226	79	10	0·75
429	46	28	1·17
430	73	9	0·32
564	36	4	0·20
Median	46	14	0·75
Mean	56	14	0·70
S.D.	19	8	0·41
Machine smearing (6 workers)			
11	69	14	0·79
134	74	20	0·84
229	59	22	1·35
242	60	33	1·83
247	47	4	0·50
299	61	20	3·50
Median	61	20	1·35
Mean	62	19	1·47
S.D.	9	10	1·10
Scrap lead works (11 workers)			
17	89	38	3·85
53	72	16	1·55
65	55	17	1·61
104	82	25	3·76
201	70	26	1·18
293	63	18	1·18
330	51	6	0·30
343	85	36	2·40
357	41	2	0·34
361	68	23	2·23
841	69	14	0·60
Median	69	18	1·55
Mean	68	20	1·73
S.D.	15	11	1·23

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APPENDIX TABLE (continued)

(b) VALUES FOR MEN IN DEPARTMENTS WITH ROTATING SYSTEM

Worker no.	*Lead in blood (μg/100 ml)*	*Lead in urine (μg/100 ml)*	*ALA in urine (mg/100 ml)*
Tube filling (11 workers)			
3	62	5	0·20
13	62	11	0·70
22	30	7	0·39
71	34	3	0·22
115	48	2	0·24
124	60	14	0·55
127	69	12	0·39
133	63	4	0·24
135	67	4	0·27
280	72	23	1·78
920	43	7	0·17
Median	62	7	0·27
Mean	56	8	0·47
S.D.	14	6	0·46

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Worker no.	*Lead in blood (μg/100 ml)*	*Lead in urine (μg/100 ml)*	*ALA in urine (mg/100 ml)*
Ball mills (7 workers)			
113	50	13	0·48
177	49	10	0·45
216	73	25	0·94
289	52	6	0·38
290	78	2	0·13
364	50	11	0·34
420	48	12	0·47
Median	50	11	0·45
Mean	57	11	0·46
S.D.	13	7	0·25
Paste mixing (9 workers)			
2	76	18	2·60
29	64	3	0·32
167	64	5	0·30
191	77	5	0·32
235	60	17	1·47
370	56	5	0·38
410	43	5	0·24
437	70	13	0·85
768	89	23	3·52
Median	64	13	0·38
Mean	67	10	1·11
S.D.	13	7	1·19