The Diagnosis of Industrial Lead Poisoning

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A series of 100 lead workers from different industries, 91 at work and nine admitted to hospital with lead poisoning, was studied in order to define more clearly the clinical and biochemical criteria of lead poisoning in three stages—(A) a presymptomatic state of lead exposure (37 men), (B) a state of mild symptoms or mild anaemia (45 men), and (C) frank lead poisoning with severe symptoms and signs (18 men).

The tests used were haemoglobin, reticulocyte count, and blood lead, and urinary lead, coproporphyrin, δ-aminolaevulinic acid (ALA), and porphobilinogen (PBG) estimations. Of these, the urinary lead was similar for all three groups and the blood lead estimation was of less value for determining the clinical group of the men than the haemoglobin and urinary coproporphyrin or ALA estimations, which correlated well with the clinical assessment and with each other but showed no correlation with the urinary and blood lead levels. PBG levels became raised only with the onset of symptoms of lead poisoning.

A haemoglobin of 13 g./100 ml. (90%) or less is a cautionary sign. Urinary coproporphyrin above 80 μg./ 100 mg. creatinine (800 μg./litre), ALA above 2·0 mg./100 mg. creatinine (2·0 mg.%), and PBG above 0·15 mg./100 mg. creatinine (0·15 mg.%) were almost always associated with symptoms or signs and were therefore considered to be the upper safety limits. Although the blood lead level does not differentiate between lead toxicity and lead exposure, values above 60 μg. lead/100 g. blood should alert the physician to carry out other tests.

In addition to the above tests, blood pressure, blood urea, and serum uric acid estimations were performed on all the men in order to elucidate the possible role of lead in the production of renal damage. Blood pressure and serum uric acid levels were similar for all three groups but the blood urea level was raised in group C. The reason for this finding was not established.

It was found that scrap metal burning, battery manufacturing, and ship-breaking constituted the gravest lead hazards encountered in this survey whereas wire manufacture constituted the least. Workers in the most modern factory, a car-body pressing plant, gave average values just below the danger levels for the urinary coproporphyrin and ALA estimations despite apparently efficient protective measures. This finding underlines the importance of the medical supervision of lead workers.

Lead and its compounds have been used by man for at least six thousand years (Brunton, 1937) and today the demands of industry for lead rise steadily. Its toxic properties were first recognized by Hippocrates (Hunter, 1962) and over the centuries more has been written about the causation and symptomatology of poisoning by lead than by any other toxic agent (Johnstone, 1966).

The manifestations of overt lead poisoning are well documented, but the clinical relevance of the many tests used for its diagnosis have not yet been clearly defined. Much has been achieved in the disposal of, and protection from, lead dust and fume, the main hazards in the lead industry, and the routine screening of all statutory workers for

signs of developing lead toxicity is now required by law (Ministry of Labour, 1965). There are, however, no agreed criteria as to when a state of *lead exposure* merges into a dangerous clinical state of *lead toxicity*. Haemoglobin and basophilic stippled cell counts, urinary lead, coproporphyrin, and δ-aminolaevulinic acid (ALA) levels, and blood lead levels have all been correlated with lead exposure. The present study has accordingly been undertaken to determine the laboratory tests likely to be most useful in the early diagnosis of lead intoxication. An attempt has also been made to define the clinical and biochemical criteria of lead intoxication in three stages—(A) a presymptomatic state of lead exposure in which the men display neither symptoms nor signs, (B) a stage of mild symptoms, *e.g.*, occasional abdominal pain or mild

anaemia (12 to 13 g./100 ml.), and (C) severe symptoms and signs requiring hospital treatment.

Clinical Material

The survey included 100 subjects from several industries: nine patients admitted to hospital with acute lead poisoning from the ship breaking, scrap metal, and electric accumulator industries and 91 men studied at work from the car-body manufacturing, ship-breaking, electric accumulator, wiremaking, lead refining, and scrap metal industries. At the factory a brief history was taken and a clinical examination was performed; blood and random urine samples were collected, taking every possible precaution to avoid contamination of the samples. Any man with a history of peptic ulcer was excluded from the series. If deleading therapy was planned all investigations were completed before this was started.

The 100 men were classified into the above three groups: A, 37 men with neither symptoms nor signs; B, 45 men with either mild symptoms or mild anaemia (12 to 13 g./100, ml.); and C, 18 men with severe symptoms and signs. The latter group contained the nine hospital patients and nine men initially studied at work, of whom six were subsequently admitted to hospital.

Laboratory Methods

A haemoglobin and reticulocyte count, blood lead (Gonzales, Vance, and Helpern, 1954, modified by Goldberg, Smith, and Lochhead, 1963), blood urea (Skeggs, 1957) and serum uric acid (Caraway, 1955), and urine lead (Gonzales *et al.*, 1954), coproporphyrin (Rimington, 1961), ALA (Mauzerall and Granick, 1956), and creatinine (Hawk, Oser, and Summerson, 1953, adapted for the AutoAnalyzer) were performed on all men. The urinary lead, coproporphyrin, and ALA values were expressed relative to 100 mg. creatinine to eliminate as far as possible variations in concentration of the random samples. In a series of 24-hour urine collections performed on 20 normal adults, it was found that the scatter in values for lead, coproporphyrin, and ALA was least when expressed relative to 100 mg. urinary creatinine (Fig. 1). An estimation of urinary porphobilinogen (PBG) was performed on some of the men by the method of Mauzerall and Granick (1956) and was also expressed relative to 100 mg. creatinine.

Results

Haemoglobin and Reticulocyte Count Fifty-four men in this series (*i.e.*, 54%) had a haemoglobin level less than 13 g./100 ml. and 31 of these had a reticulocyte count greater than 2%. Only three of the 46 men with a haemoglobin greater than 13 g./100 ml. had a reticulocyte count greater than 2%.

All 37 men in group A had haemoglobins greater than 13 g./100 ml. by definition of this group. Two of these, however, had a raised reticulocyte count. Of the 45 men in group B, 36 had haemoglobins of 13 g./100 ml. or less while 19 had a raised reticulocyte count. Of the 18 men in group C, all had haemoglobins of 13 g./100 ml. or less and 13 had a raised reticulocyte count.

Urinary Lead The normal range for urinary lead, calculated on the 20 normal adults, was 0–18·09 μg. lead/100 mg. creatinine, with a mean of 3·89 μg./100 mg. creatinine. The means for this estimation were 22·41, 27·49, and 27·68 μg. lead/100 mg. creatinine for groups A, B, and C respectively (Table I). None of these differed significantly (P>0·3) from the other although all were signi-

TABLE I

MEAN VALUES AND STANDARD DEVIATIONS OF URINARY LEAD, URINARY COPROPORPHYRIN, URINARY ALA, AND BLOOD LEAD FOR THE THREE GROUPS

Group	Urine			Blood Lead (μg./100 g.)
	Lead (μg./100 mg. creatinine)	Coproporphyrin (μg./100 mg. creatinine)	ALA (mg./100 mg. creatinine)	
A	22·41 (16·21)	54·56 (38·68)	1·48 (1·24)	51·72 (33·81)
B	27·49 (21·87)	110·07 (67·41)	2·62 (1·46)	73·69 (44·21)
C	27·68 (16·58)	143·55 (72·41)	4·29 (1·95)	83·94 (44·14)

Standard deviations are shown in parentheses.

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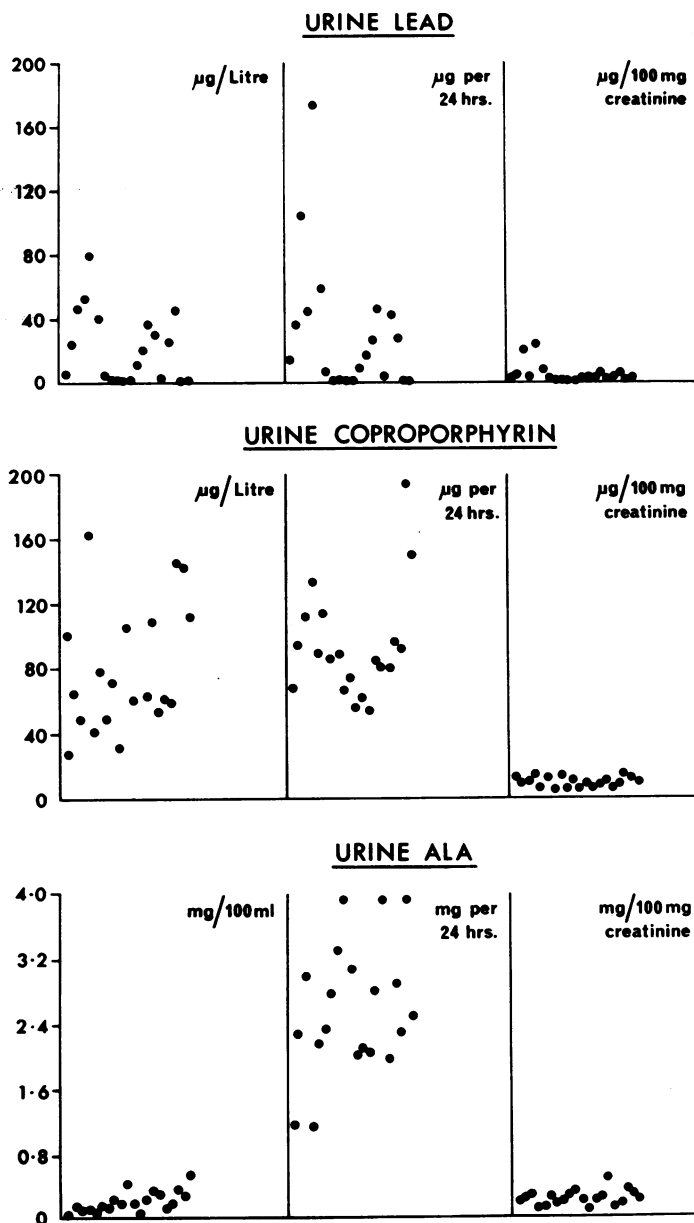## URINE LEAD

URINE COPROPORPHYRIN

URINE ALA

FIG. 1. Comparison of the values for urinary lead, coproporphyrin, and δ-amino-laevulinic acid (ALA) in 20 normal adults.

ficantly higher than the normal mean. Urinary lead did not correlate with any other estimation (r = 0·17).

Urinary Coproporphyrin The normal range for urinary coproporphyrin was 2·04-15·64 μg./ 100 mg. creatinine, with a mean value of 8·84 μg./ 100 mg. creatinine. Five subjects in group A and four in group B were within the nomal range or just above the upper limit of normal (Fig. 2). No subject in group C was normal.

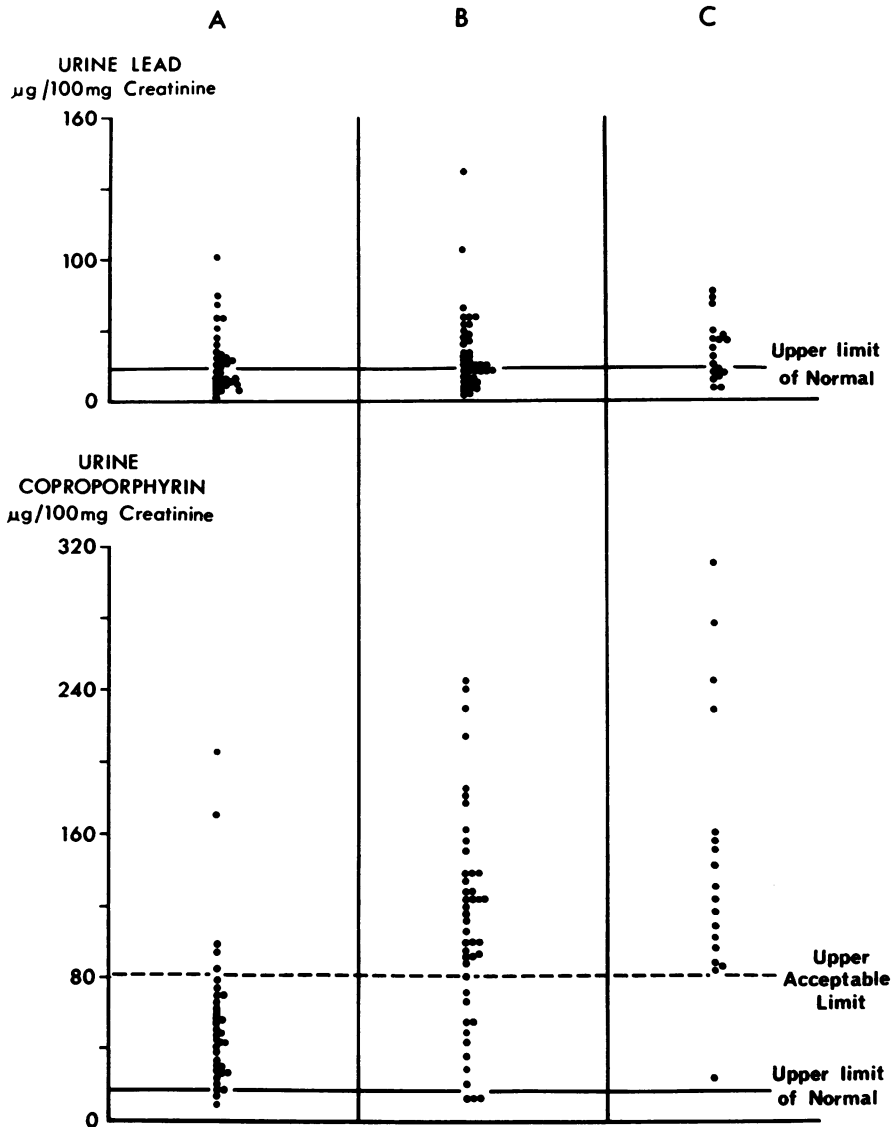The means for this estimation were 54·56, 110·07,

FIG. 2a. Comparison of the values for urinary lead and urinary coproporphyrin for the three
groups A, B, and C.

and 143·55 μg. coproporphyrin/100 mg. creatinine
for groups A, B, and C respectively (Table I), all
of which differ significantly from each other (P<
0·05). All, moreover, are greatly in excess of the
normal mean.

Although the scatter is large (Fig. 2), giving
considerable overlap in three groups, the results
indicate that the occurrence of a coproporphyrin
level of 80 μg./100 mg. creatinine or over is com-
patible with the presence of symptoms. Values

above this level include all but one man (94%) of
group C, 75% of group B, and only 14% of group A.
A similar separation occurs when the urinary
coproporphyrin results are expressed in μg./litre,
when a coproporphyrin level of 800 μg./litre or
over is compatible with the presence of symptoms
indicative of toxicity.

Urinary δ-Aminolaevulinic Acid The nor-
mal range for this estimation was found to be 0·04-

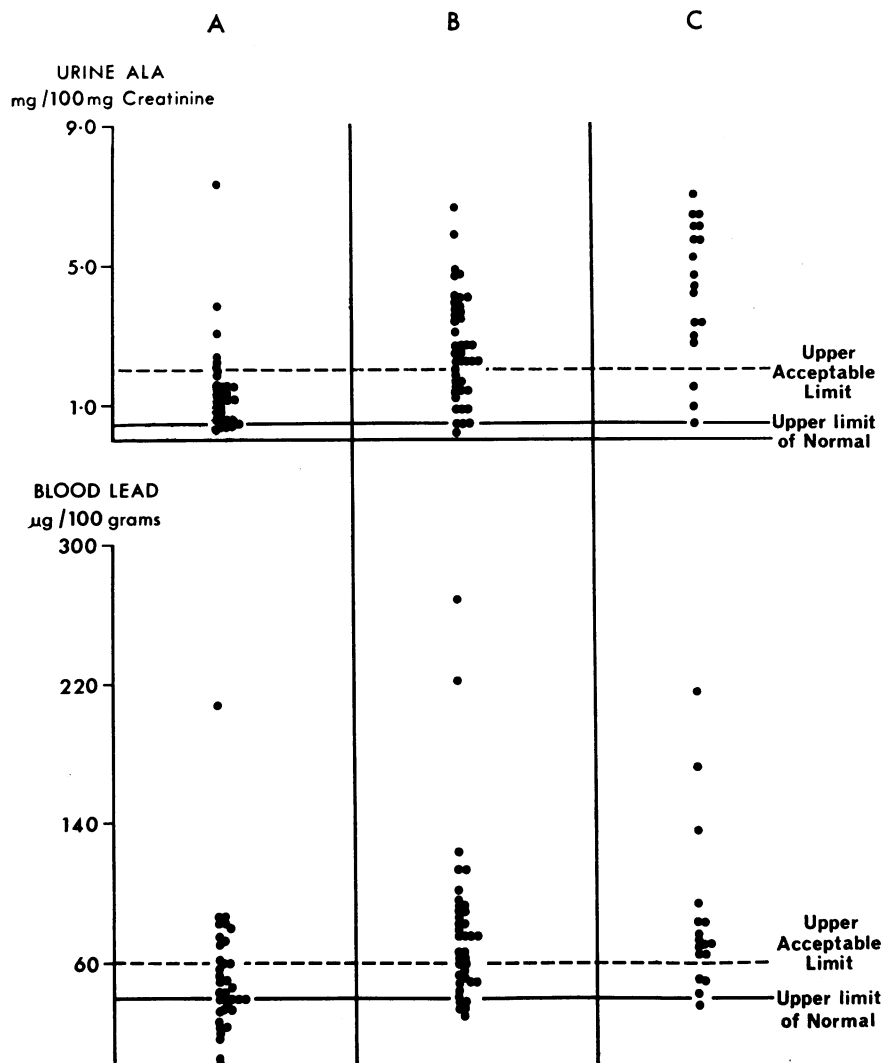*Sheila L. M. Gibson, J. C. Mackenzie, and A. Goldberg*

FIG. 2b.   Comparison of the values for urinary ALA and blood lead for the three groups A, B, and C.

0·44 mg. ALA/100 mg. creatinine with a mean value of 0·24 mg./100 mg. creatinine. Three subjects from group A, two from group B, and one from group C are within the normal range (Fig. 2). The three subjects in group A correspond to three of the five in group A with normal coproporphyrin levels, while the two in group B correspond to two of the four with normal coproporphyrin levels in that group. The one man in group C with urinary ALA within the normal range had a high urinary coproporphyrin (122·54 μg./100 mg. creatinine).

The mean urinary excretions of ALA for groups A, B, and C respectively are 1·48, 2·62, and 4·29 mg. /100 mg. creatinine (Table I). These are all considerably higher than the normal mean and differ significantly from each other ($p < 0·05$). Again, however, considerable overlap is evident (Fig. 2) but any urinary ALA value of 2 mg./100 mg. creatinine or over is compatible with symptoms of lead poisoning indicative of toxicity. This corresponds to the value of 80 μg. coproporphyrin/ 100 mg. creatinine (Figs 2 and 4). A high degree of correlation was found between urinary coproporphyrin and urinary ALA (Fig. 4) ($r = 0·79$).

Urinary Porphobilinogen This estimation was performed on the men with acute lead intoxication admitted to hospital during the course of this study and in all cases was found to be slightly raised. On the other hand, random PBG estimations performed on some of the men studied at work had all been found to be normal. In view of this fact, in all subsequent cases an estimation was made of the urinary PBG, which was determined in 62 of the total of 100 men studied, 16 of group A, 30 of group B, and 16 of group C.

The normal range is 0·02-0·14 mg./100 mg. creatinine and the normal mean 0·08 mg./100 mg. creatinine. The means were 0·08, 0·12, and 0·34 mg./100 mg. creatinine for groups A, B, and C respectively. While the mean of B is just significantly higher than that of A ($p = 0·05$), the mean of C is significantly higher than those of either A or B ($p < 0·01$). No individuals in group A, eight in group B, and all but two in group C had a raised PBG (Fig. 3). All patients admitted to hospital with acute lead intoxication had PBG values above the normal range.

Blood Lead The upper limit of normal for this estimation is 40 μg./100 g. blood, in this department, with a range of 11·40-49·10 μg./100 g. (Goldberg *et al.*, 1963). This is lower than the figure of 60 μg./100 g. blood given by Kehoe, Thamann, and Cholack (1935) or that of Byers (1959) of 60-80 μg./100 g. blood.

The mean levels for groups A, B, and C respectively were 51·72, 73·69, and 83·94 μg. lead/100 g. blood (Table I). Fifteen men (41%) in group A, seven men (16%) in group B, and one man (6%) in group C had values within the normal range. The mean levels for all groups are significantly higher than the normal mean ($p < 0·01$) and A and B and A and C differ significantly ($p < 0·05$), but B and C do not ($p = 0·2$). With this estimation there is a considerably greater overlap in the three groups than with either the urinary coproporphyrin or the urinary ALA estimations (Fig. 2) but 30% of group A, 60% of group B, and all but four (78%) in group C have levels greater than 60 μg. lead/100 g. blood, similar to the numbers in each group with urinary ALA levels greater than 2 mg./100 mg.
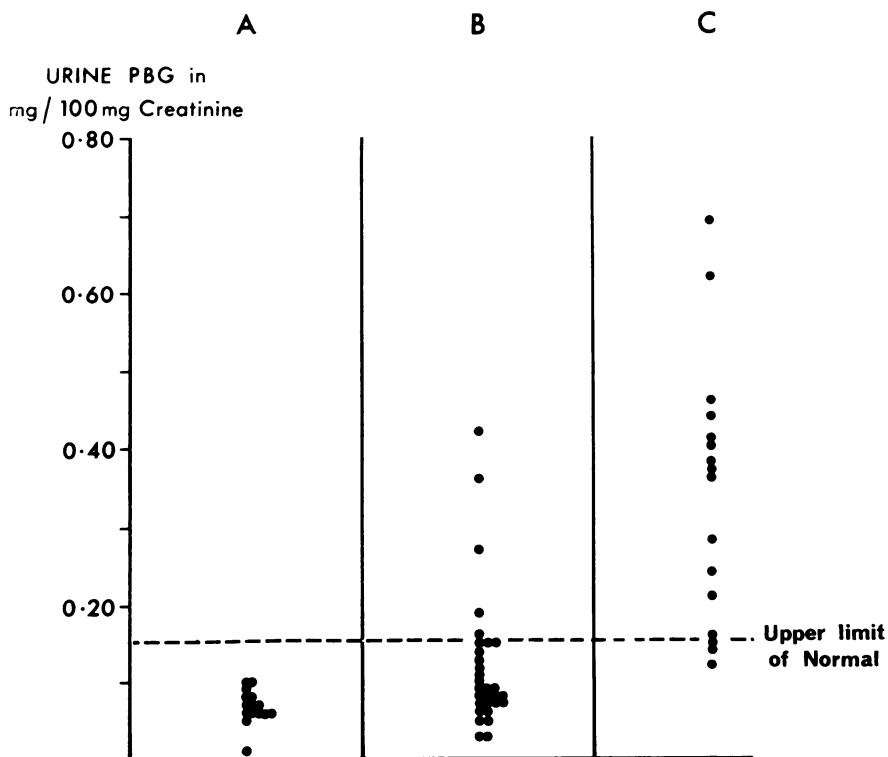

FIG. 3. Comparison of the values for urinary porphobilinogen (PBG) for the three groups A, B, and C.

creatinine (Table IV). This figure of 60 μg. lead/100 g. blood is therefore proposed as the upper acceptable limit for this estimation although in the whole series no significant correlation was found between blood lead and either urinary coproporphyrin (r = 0·16) or urinary ALA (r =0·16).

Blood Pressure, Blood Urea, Serum Uric Acid, Age, and Duration of Exposure Blood pressure, blood urea, and serum uric acid were estimated in all individuals in an attempt to elucidate whether or not lead exposure predisposes to hypertension and renal damage. The mean diastolic blood pressures of groups A, B, and C were 82, 82, and 74 mm. of mercury respectively. The mean ages of the groups were 37, 39, and 37 years respectively, and the mean durations of exposure 7, 8, and 9 years respectively (Table II).

The serum uric acid values were likewise similar for each group, the mean values being 4·7, 4·8, and 4·7 mg./100 ml. serum for A, B, and C respectively (Table II).

The mean values for blood urea were 34·4, 38·2, and 42·3 mg./100 ml. blood for A, B, and C respectively (Table II), and although the means of A and B and B and C are not significantly different, the means of A and C are significantly different (P<0·01). Five men in group A, six in group B, and six in group C showed trace amounts of proteinuria as tested by the 'albustix test'. Addis counts, a count of the white cells in a centrifuged 10-ml. sample of urine, performed by the method of McGeachie and Kennedy (1963), on 15 men from group A, 15 from group B, and 10 from group C showed no abnormality. These included all men with proteinuria.

Discussion

Any worker exposed to lead dust or fume will absorb some lead and will therefore excrete lead in the urine (Kehoe, Thamann, and Cholak 1933; Kehoe, Cholak, Hubbard, Bambach, and McNary, 1943).

Not all men, however, with evidence of lead exposure are in danger of developing symptoms of lead toxicity (Kehoe, 1964; Lane, 1964). The difficulty is to recognize when a 'state of exposure' merges into a 'state of toxicity'. Even when some of the symptoms and signs of lead poisoning appear, difficulties may still be encountered as few are specific for lead poisoning.

Much has been published on the tests considered to be most helpful in assessing the state of the lead worker with respect to lead. Apart from a brief history and clinical examination the tests most commonly used by factory doctors are serial haemoglobins, stippled cell counts, and a semi-quantitative urinary coproporphyrin estimation (Cramér and Dahlberg, 1966). These tests are easily and rapidly performed and are thus applicable to the frequent checking of large numbers of workmen. King and Thompson (1961) and Johnstone (1964), however, regard basophilic stippling as being unreliable, and Lane (1964) advocates the use of blood and urine lead estimations and considers that the urinary coproporphyrin test is too sensitive.

Clinical Significance of Laboratory Tests Urinary coproporphyrin (de Langen and ten Berg, 1948; Waldman and Seideman, 1950; Maloof, 1950; Brooks, 1951; Malcolm, 1965) and urinary ALA (Haeger, 1957; Cramér and Selander, 1965) have been considered useful in the detection of lead exposure but in all these papers no real difference has been drawn between exposure and intoxication.

The presence of anaemia and of raised levels of urinary coproporphyrin, ALA, and PBG reflect significant effects on haem and haemoglobin metabolism and hence are more likely to reflect the degree of lead toxicity than blood and urine lead levels which merely indicate the degree of lead absorption (Schrenk, 1959). Individuals, moreover, vary in their sensitivity to toxic agents. It is, therefore, not surprising that no correlation was found between tests which measure exposure to lead (urinary and blood lead) and those which measure interference with tissue enzymes caused by

TABLE II

MEAN VALUES FOR AGE, LENGTH OF TIME WORKED, DIASTOLIC BLOOD PRESSURE, BLOOD UREA, AND SERUM URIC ACID FOR THE THREE GROUPS

Group	Diastolic B.P. (mm. Hg)	Age (yrs)	Blood Urea (mg./100 ml.)	Serum Uric Acid (mg./100 ml.)	Time Worked (yrs)
A	82	37	34·4	4·7	7
B	82	39	38·2	4·8	8
C	74	37	42·3	4·7	9

TABLE III

CORRELATIONS FOUND BY PREVIOUS AUTHORS COMPARED WITH THOSE FOUND IN THE PRESENT STUDY

Correlation	Author	
Positive correlation between urinary coproporphyrin and urinary lead excretion	Waldman and Seideman	(1950)
	Maloof	(1950)
	Haeger-Aronsen	(1960)
	Cramér and Selander	(1965)
Positive correlation between urinary ALA and urinary lead excretion	Haeger-Aronsen	(1960)
	Cramér and Selander	(1965)
Positive correlation between urinary coproporphyrin and blood lead	Brooks	(1951)
No correlation between semi-quantitative urinary coproporphyrin and blood lead	Meek, Mooney and Harrold	(1948)
No correlation between urinary coproporphyrin and ALA excretion	de Kretser and Waldron	(1963)
Positive correlation between urinary ALA and PBG and clinical manifestations	Cramér and Selander	(1965)
Positive correlation between urinary coproporphyrin and ALA excretion ($r = 0.79$)		
Positive correlation between urinary coproporphyrin and haemoglobin level ($r = 0.54$)		
Positive correlation between urinary ALA and haemoglobin level ($r = 0.57$)		
Positive correlation between urinary coproporphyrin and ALA and clinical assessment	Observations from present study	
Positive correlation between rise in urinary PBG and presence of symptoms ($p < 0.01$)		
No correlation between urinary coproporphyrin or ALA and blood lead ($r = 0.16$)		
No correlation between urinary coproporphyrin or ALA and urinary lead ($r = 0.17$)		

lead (haemoglobin level, and urinary coproporphyrin, ALA, and PBG levels). Furthermore, the tests indicative of this interference correlate more closely with the clinical assessment than do those which reflect exposure. The haemoglobin level could not be correlated directly with the clinical grouping as it formed a part of it, but the haemoglobin level correlates significantly with the urinary coproporphyrin ($r = 0.54$) and ALA ($r = 0.57$) ($p < 0.05$ in both cases). This agrees with the findings of Chisolm and Harrison (1956) and of Haeger-Aronsen (1960) that the urinary excretion of coproporphyrin and of ALA respectively are sensitive indices of 'metabolically active' and presumably toxic lead in the body.

The results of the present study confirm that the urinary coproporphyrin level is a very sensitive test of exposure to lead (Lane, 1964) and also indicate that it is of value in the total appraisal of the individual's reaction to lead. Studies in experimental lead poisoning in rabbits (Gibson and Goldberg, unpublished data) show that the urinary coproporphyrin rises immediately on exposure to lead and that the urinary ALA shows a rise after about one to two weeks of continual dosage. Urinary PBG begins to rise above normal levels only after several weeks of continual dosage. This animal work confirms the results of the present study in lead workers that the urinary coproporphyrin and ALA become abnormal on exposure to lead, whereas a rise in the urinary PBG is a much later sign occurring only with the onset of symptoms. This finding is of particular significance because of the close similarity between the clinical manifestations of lead poisoning and acute intermittent porphyria (Dagg, Goldberg, Lochhead, and Smith, 1965) in which there is often a marked rise in urinary PBG as well as ALA with the onset of acute symptoms.

Evidence of Toxicity The majority of men in group A have urinary coproporphyrin values below 80 μg./100 mg. creatinine (equivalent approximately to 800 μg./litre urine) whereas all but one man in group C and 75% of group B have values above this level (Fig. 2.) The majority of the men in group A have urinary ALA values below 2·0 mg. ALA/100 mg. creatinine (2·0 mg./100 ml. urine), while all but three men in group C and 60% of group B have values above this level (Fig. 2). Furthermore, the value of 80μg. urinary coproporphyrin/100 mg. creatinine corresponds to that of 2·0 mg. urinary ALA/100 mg. creatinine when

ALA
mg/100mg Creatinine

$$y = 1.21 + 0.022 \ (x - 45.88)$$
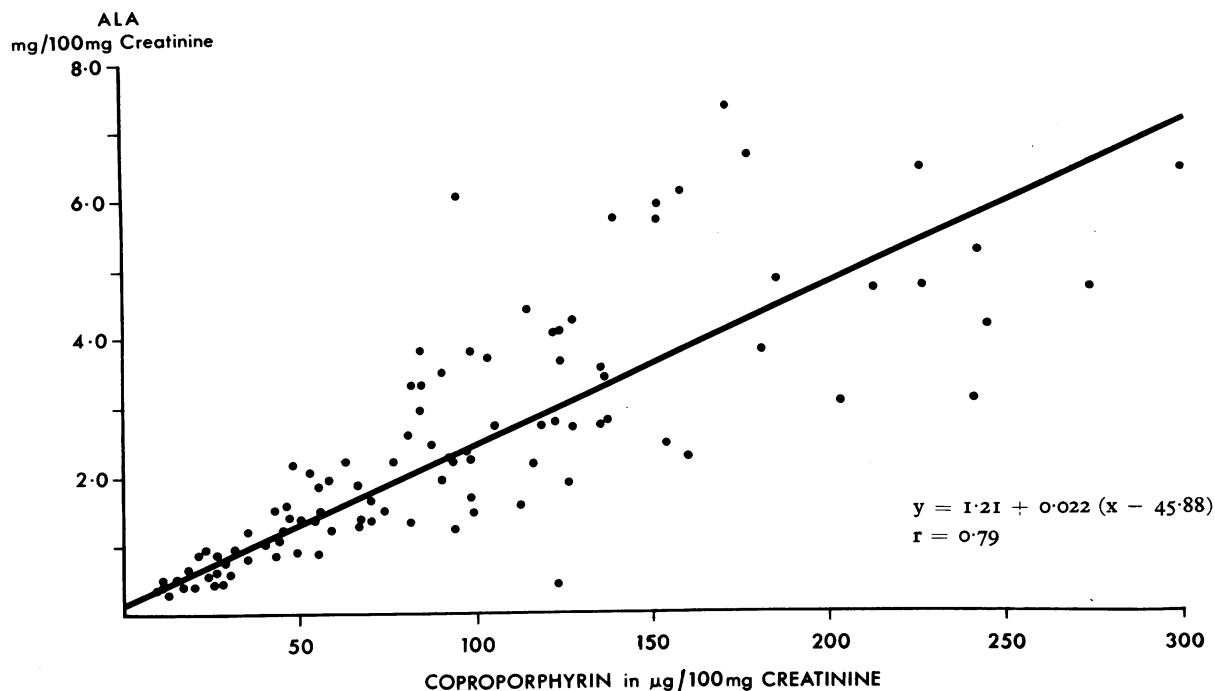$$r = 0.79$$

COPROPORPHYRIN in µg/100mg CREATININE

FIG. 4.   Graph of urinary ALA against urinary coproporphyrin for the whole series of 100 men.

correlating these estimations (Fig. 4). It is therefore considered that these values should be taken as indicative of dangerous lead absorption. The numbers of men above and below the critical limits in groups A, B, and C are presented in Table IV and compared with the number with blood lead levels greater and less than 60µg. lead/100 g. blood. The value for coproporphyrin agrees with that proposed by Lane (1964) of 800 µg./litre. Any man with a urinary PBG value of 0·15 mg./100 mg. creatinine or over should be considered in imminent

danger of developing symptoms of lead poisoning if these are not already present. As the urinary coproporphyrin level is such a sensitive test, any man with a normal level is unlikely to have suffered recent exposure to lead. A raised urinary PBG level is also of value where doubt arises as to whether the symptoms of colic and constipation are in fact due to lead poisoning. The level never rises to that found in the acute stages of acute intermittent porphyria.

Table V summarizes the evidence of toxicity

TABLE IV

COMPARISON OF THE NUMBERS OF MEN IN GROUPS A, B, AND C WITH URINARY COPROPORPHYRIN, URINARY
δ-AMINOLAEVULINIC ACID, AND BLOOD LEAD GREATER AND LESS THAN THE CRITICAL LEVELS

Group	A (37 men)		B (45 men)		C (18 men)	
	Above Critical Level	*Below Critical Level*	*Above Critical Level*	*Below Critical Level*	*Above Critical Level*	*Below Critical Level*
Urinary coproporphyrin[1]	5	32	33	12	17	1
Urinary ALA[2]	8	29	28	17	15	3
Blood lead[3]	11	26	27	18	14	4

[1] Critical level 80 µg./100 mg. creatinine
[2] Critical level 2·0 mg./100 mg. creatinine
[3] Critical level 60 µg./100 g. blood

TABLE V

EVIDENCE OF TOXICITY

Series	Safe Range	Danger Levels
Lane (1964)	Blood lead less than 80 μg./100 ml. blood Urinary lead less than 150 μg./litre Haemoglobin over 13 g.% (90%) Punctate basophilia less than 10,000/10⁶ red cells Urinary coproporphyrin + + + (500 μg./litre) or less	Blood lead over 80 μg./100 ml. blood Urinary lead over 200 μg./litre Haemoglobin less than 13 g.% or falling haemoglobin Punctate basophilia of 5-20,000/10⁶ red cells Urinary coproporphyrin + + + + (800 μg./litre)
Present study	Haemoglobin over 13 g.% (90%) Urinary coproporphyrin less than 80 μg./100 mg. creatinine (800 μg./litre) Urinary ALA less than 2·0 mg./100 mg. creatinine (2·0 mg.%) Blood lead less than 60 μg./100 g. blood.	Haemoglobin less than 13 g.% or falling haemoglobin Urinary coproporphyrin over 80 μg./100 mg. creatinine (800 μg./litre) Urinary ALA over 2·0 mg./100 mg. creatinine (2·0 mg.%) Blood lead over 60 μg./100 g. blood Urinary PBG over 0·15 mg./100 mg. creatinine (0·15 mg.%)

found in this series and compares it with that proposed by Lane (1964). The value of 60 μg. lead/100 g. blood is included in this table although this estimation is evidence of exposure and not strictly of toxicity.

Renal Damage Due to to Lead Poisoning
Lane (1964) has stressed the importance of recognizing a state of latent lead toxicity in which workers have absorbed toxic quantities of lead and, although apparently well, may develop renal damage. While there seems to be little doubt that renal damage can follow lead poisoning in children (Henderson, 1955; Henderson and Inglis, 1957) and lead poisoning in adults resulting from oral ingestion of lead over long periods (Morgan, Hartley, and Miller, 1966; Danilović, 1958), a marked relationship is not generally accepted in industrial lead exposure (Aub, Fairhall, Minot, and Reznikoff, 1926; Kehoe, 1964; Johnstone, 1964).

No conclusive evidence was obtained on this point from this survey. The raised blood urea level of group C cannot be explained either by age or by hypertensive renal damage. It was at first considered to be due to a transient drop in the glomerular filtration rate (GFR) as a result of abdominal colic and vomiting, causing a pre-renal azotaemia, but the GFR, as measured by the creatinine clearance test, was normal in nine patients in whom this was carried out within three to five days of admission. This also indicates that it was unlikely to have been due to transient intrinsic renal damage. The presence of proteinuria bore no

relation to the degree of exposure or intoxication. A fuller survey of the effects of lead on the kidney will be the subject of a future publication.

Relative Hazard of Different Industries
Seven industrial firms co-operated in this survey providing 98 of the 100 men studied. Some firms had new factories with modern engineering precautions to reduce the lead hazard, e.g., the car-body manufacturing plant, while others, e.g., the wire-manufacturing plant and the lead refinery, were old. Others, e.g., ship-breaking and scrap-metal burning, presented a hazard less easy to control than in car or battery manufacture. It was, therefore, felt that it would be of interest to compare the average values of haemoglobin, blood lead, urinary lead, coproporphyrin, and ALA obtained for the different firms. These were accordingly tabulated along with the length of service (Table VI).

The workers in four of the firms, battery manufacture, scrap-metal burning, and the two ship-breaking firms, show mean urinary coproporphyrin values greater than 80 μg./100 mg. creatinine, urinary ALA values greater than 2·0 mg./100 mg. creatinine, and blood lead values in excess of 60 μg./100 g. blood, while in three of the firms the mean haemoglobin values are less than 13 g./100 ml.

The workers in the car-body plant and lead refinery have mean haemoglobin values just over and mean urinary coproporphyrin and ALA values just under the critical levels, while the mean blood lead value for those in the new car-body plant is greater than 60 μg./100 g. blood and that for those in the old lead refinery is less than 60 μg./100 g.

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TABLE VI

AVERAGE VALUES FOR HB, URINARY LEAD, URINARY COPROPORPHYRIN, URINARY ALA, BLOOD LEAD, AND LENGTH OF
SERVICE FOR THE SEVEN FIRMS

| Firm | No. of Workers | Hb (g./100 ml.) | Urine | | | Blood Lead (μg./100 g.) | Time (yrs) |
| | | | Lead (μg.) | Coproporphy-rin (μg.) | ALA (mg.) | | |
				per 100 mg. creatinine			
Battery manufacturers	9	11·81	20·89	135·65	3·27	65·49	9¾
Scrap-metal firm	13	11·55	38·39	130·49	4·19	94·33	9¼
Ship-breaking firm 1	20	13·60	29·50	106·75	2·80	63·92	6¾
Ship-breaking firm 2	16	12·74	28·98	95·52	2·59	81·06	14½
Car-body factory	20	13·40	23·29	76·57	1·80	64·73	1
Lead refinery	12	13·70	14·49	73·11	1·88	45·63	3¼
Wire factory	8	14·75	19·11	43·10	1·02	46·26	12¾

blood. The least hazard in this series was encountered in the wire-manufacturing factory, where 50% of the workers were excluded from the survey as they had no evidence of exposure to lead.

It was not surprising to find that industries such as scrap metal burning and ship-breaking should show a generally dangerous level of exposure to lead, since lead fume, often produced in confined spaces, is difficult to control and the wearing of masks difficult to enforce when continual supervision of the men at work is not feasible. In the wire-manufacturing factory, on the other hand, lead was used only as a molten bath and the exposure hazard was less severe.

Workmen in the lead refinery and the car-body plant had similar mean values. The lead refinery was an old factory whereas the car plant was new, having been in operation for just under two years, with the mean length of service one year. It was equipped with modern devices for the removal of lead dust, the lead hazard in this industry, and the high level of exposure was probably largely due to the workmen removing the excess lead solder while not in the specially installed 'discing booths'.

In conclusion it may be said that in some lead industries by their very nature, e.g., scrap-metal burning and ship-breaking, and in others despite modern technical control, a considerable lead hazard still exists. From the results of this survey it is felt that routine haemoglobin and urinary coproporphyrin, ALA, and PBG estimations are of most value in screening the men at risk. A steadily falling haemoglobin or a haemoglobin below 13 g./100 ml., if other causes for this are excluded, is a warning sign, while the upper safety limits for urinary coproporphyrin and urinary ALA are 80 μg./100 mg. creatinine (800 μg./litre) and 2·0 mg./100 mg. creatinine (2·0 mg.%) respectively.

Ideally, values of this order should never be encountered. A rise in urinary PBG above 0·15 mg./100 mg. creatinine indicates acute lead poisoning. The blood lead estimation does not differentiate between lead exposure and lead toxicity but values above 60 μg. lead/100 g. blood should alert the physician to carry out other tests.

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