

BLOOD LEAD CONCENTRATIONS IN ROAD-SIDE BATTERY CHARGERS IN NIGERIA

S.E. ASOGWA

University of Nigeria Teaching Hospital, Enugu, Nigeria

ABSTRACT

Self-employed road-side lead-acid battery chargers in Nigeria have been suspected of being at risk of lead poisoning. To get an idea of the amount of lead present in the organism of these workers, rather than make a deduction based on the effect of lead, a group of 43 male workers and a control group of 50 unexposed subjects of both sexes were taken in the study. Blood lead was examined in all of them by the method of atomic absorption spectrophotometry with the technique for spotting venous blood as recommended by the Occupational Health and Hygiene Laboratory of London where the estimations were done.

It was found that there were higher levels of blood lead in the exposed than in the unexposed group ($P < 0.01$). Three workers had "excessive" levels of blood lead (80–120 $\mu\text{g}/100\text{ ml}$) while 25 had "acceptable" levels. Since the number of battery chargers in a workshop was too small (less than ten) for their place of work to be recognized legally as a factory, recommendations were made to ensure that they work in groups of not less than ten to enable the Factory Inspectorate to inspect their workshops regularly.

The dimension of both industrial and non-industrial exposure to the hazards of lead poisoning in Nigeria has not been fully ascertained. In 1968 in a survey of a car battery factory in Nigeria employing 35 men two workers suspected of chronic lead poisoning, 16 with symptoms of lead poisoning, 8 with clinical symptoms of exposure to lead and only 9 without symptoms were found². Fume sampling showed lead fume pollution in several places. In the non-industrial exposure group self-employed lead accumulator battery workers (Fig. 1) – Sofoluwe and co-workers⁸ in a survey in Lagos estimated the degree of risk of 71 battery chargers. Urinary delta-aminolaevulinic acid (d-ALA) values were determined in these people and a control group (20 medical students). They were able to establish the fact that these workers were at risk and that in this trade, contrary to what was anticipated, the exposure by inhalation of lead fumes was more important than by oral route. The job of the battery charger was also described in detail.

The purpose of this study was to look again at the problem of lead with respect to self-employed lead workers in another part of the country (Enugu, the

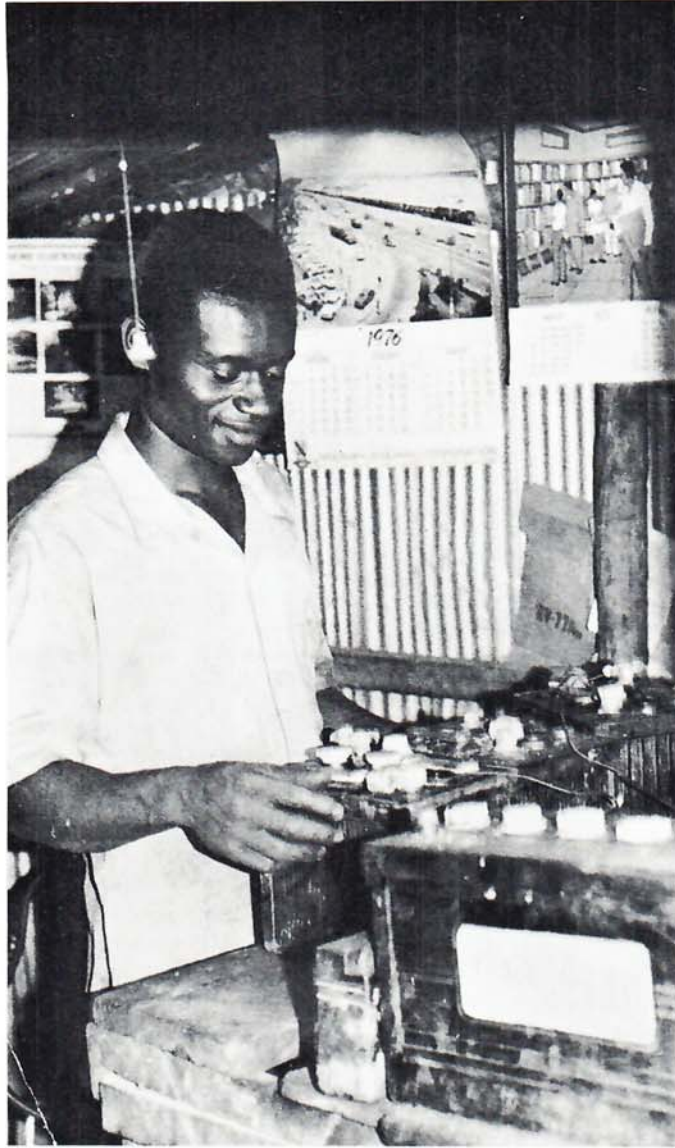


FIG. 1 - Inside view of a road-side battery workshop.

capital of Anambra State) and to try to estimate the degree of risk by quantifying the amount of lead in workers' blood by the comparatively new and very reliable method of atomic absorption spectrophotometry (AAS).

SUBJECTS AND METHODS

The exposed group (A) consisted of 43 lead-acid battery workers and the control group (B) of 50 unexposed subjects (Table 1). Both groups were aged 10–35 years. Group A were all male workers while Group B was made up of both male (24) and female workers (26). Those whose blood was examined in Group B were selected randomly. All the blood was collected by venepuncture and specimens were prepared by the technique for the spotting of venous blood as recommended by the Occupational Health and Hygiene Laboratory of London⁶. After the blood was spotted on filter paper it was sent to the

TABLE 1
Number of workers and blood levels of lead ($\mu\text{g}/100\text{ ml}$).

Study group	Normal (less than 40)	Acceptable (40–80)	Excessive (80–120)	Dangerous (over 120)	Total
A (Exposed)	15	25	3	0	43
B (Unexposed)	49	1	0	0	50

$t = 2.64$; $Df = 91$; $P < 0.01$

Occupational Health and Hygiene Laboratory of London where blood lead was estimated by the AAS method. All the blood samples were taken and spotted by one person (the investigator) who used a strictly aseptic technique. Haemoglobin estimations were carried out on the remaining blood samples. All the people in the exposed group were questioned about symptoms and were briefly examined at their workshops for signs of lead poisoning.

RESULTS

It was found that there was a statistically significant difference ($P < 0.01$) between the levels of blood lead in the two groups (Table 1). The levels of blood lead in the unexposed group (Group B) were within the expected normal values while those of the exposed group (Group A) showed evidence of abnormal lead absorption. Only one person in the control group (B) had blood lead above the normal value but it was still within the acceptable level (for industrial lead workers). People with high blood lead were mostly those who had been in the trade for a period of 1–4 years (Table 2). Those aged between 20–24 years were about a half of the study group and they also were the most affected age group (Table 3). The three workers with excessive levels of blood lead were within this age group. The mean haemoglobin was 14.5 g/100 ml with a range of 11.2–16.5 g/100 ml (Table 4). There was poor linear correlation between blood lead and duration of exposure on the one hand and blood lead and haemoglobin levels on the other. The correlation coefficients (r) were 0.01 and 0.39 respectively. The characteristic blue line (Burton's line) in the gum usually seen in those exposed to

TABLE 2
Blood lead ($\mu\text{g}/100\text{ ml}$) and duration of exposure (Group A).

Duration of exposure (years)	Normal (less than 40)	Acceptable (40-80)	Excessive (80-120)	Dangerous (over 120)	Total
Less than 1	5	4	-	-	9
1-4	6	7	3	-	16
5-9	2	7	-	-	9
10-14	2	4	-	-	6
15-19	1	2	-	-	3
Total	16	24	3	-	43

TABLE 3
Blood lead ($\mu\text{g}/100\text{ ml}$) and age of lead workers.

Age (years)	Normal (less than 40)	Acceptable (40-80)	Excessive (80-120)	Dangerous (over 120)	Total
Less than 15	-	1	-	-	1
15-19	2	4	-	-	6
20-24	8	11	3	-	22
25-29	4	7	-	-	11
30-34	-	3	-	-	3
Total	14	26	3	-	43

TABLE 4
Age group and haemoglobin of the exposed Group A.

Age group (years)	Haemoglobin (g/100 ml)			Total
	Less than 10	10-15	Over 15	
Less than 15	-	1	-	1
15-24	-	21	9	30
25-34	-	5	7	12
35-40	-	-	-	-
Total	-	27	16	43

lead associated with poor oral hygiene, was not thought important in the coloured races. There were no symptoms or signs suggestive of lead poisoning in the study group.

DISCUSSION AND CONCLUSIONS

The diagnosis of lead poisoning and control of lead exposure among workers are based on clinical examinations, laboratory studies and environmental measurements³. The clinical effects of lead have been well documented¹. In a

study of male adolescents and young adults treated for disorders of behaviour or learning disability Moor and Fleischman⁵ found that they exhibited increased systolic pressure, decreased hand-eye coordination and shortened reaction times all of which significantly correlated with the lead (in hair) concentration considered to be within normal range. Failure to find clinical evidence of the effects of lead on these (Nigerian) workers was more likely due to the fact that they were not examined in any great detail at their workplaces than that there were none at all. The study of Shannon and co-workers⁷ showed little evidence of harmful effects of lead exposure as measured by sickness absence in men with blood lead levels of up to 80 $\mu\text{g}/100\text{ ml}$ (3.86 $\mu\text{mol/l}$). Hence at the levels of blood lead in these battery chargers one would not expect them to be overtly ill. The positive linear correlation between blood lead and duration of exposure is negligible. This is not surprising in view of the fact that both the duration of exposure and the concentration of lead in the work environment varies between individual workers. If a study is carried out in an individual exposed to a known amount of lead over a certain period, the relationship expected is an increase in lead with the duration of exposure as lead is a cumulative poison. With regard to the relationship between blood lead and haemoglobin, the positive linear correlation ($r = 0.39$) would give the impression that haemoglobin rises with the concentration of lead in the body. This is contrary to the known effect of lead on the haemopoietic system which is anaemia. Stoke⁹ in his series also found a decrease in haemoglobin with an increase in blood lead in the order of 250 $\mu\text{g}/100\text{ ml}$ or above. It seems that in the group studied the level of lead was not high enough to produce a significant reduction in haemoglobin and that the apparent increase in haemoglobin with increase in blood lead is spurious.

In view of the fact that the workplaces of these battery chargers do not meet the definition of a factory, it is difficult for the Factory Inspectorate to do anything at this stage. The following recommendations would, it is hoped, help in safeguarding the lives of these workers without depriving them of their means of livelihood:

1. Government legislation should make it unlawful to engage in lead-acid battery work without a licence.
2. All workplaces where lead-acid battery is handled should be registered with the Factory Inspectorate.
3. Lead-acid battery factories should be used as workplaces only. Smoking and consumption of food and beverages at workplaces should be prohibited.
4. A registration should only be granted to a battery workshop if there are provisions for more than ten people licensed to work in it (thus meeting the definition of a factory as contained in Laws of the Federation of Nigeria and Lagos⁴ and if the building is satisfactorily constructed and provided with an adequate ventilation system.

With these conditions fulfilled, the Factory Inspectorate would then be able to inspect the factories and institute special monitoring in workplaces where workers are exposed to lead. Doctors in Nigeria and in all countries where self-

-employed workers are engaged in lead-acid battery work should bear in mind the possibility of lead poisoning from this source. It is therefore necessary for detailed occupational histories to be taken always in cases with symptoms and signs remotely suggestive of exposure to lead and to bear plumbism in mind as a differential diagnosis of encephalitis, poliomyelitis, abdominal colic, etc.

ACKNOWLEDGEMENT

I should like to express my gratitude to Drs H.M.P. Sayers, Allan Smith and their staff of the Occupational Health and Hygiene Laboratory, London for the assistance with blood lead estimation. To Mr B. C. Welukalu of the Census Office Enugu, Nigeria, I am obliged for the statistical analysis.

REFERENCES

1. *Anonymous*. Diagnosis of inorganic lead poisoning: A statement. *Br. Med. J.*, **4** (1968) 501.
2. *International Labour Office*. ILO Report to the Government of the Federation of Nigeria on Occupational Health. ILO/OTA/Nigeria. R.14. International Labour Office, Geneva, 1969.
3. *Lane, R.E.* Industrial lead poisoning. *Br. Med. J.*, **2** (1943) 515-520.
4. *Law of the Federation of Nigeria and Lagos*. Volume III, 1968, Ordinances, Chapters 63-96 Factories, 1371-1374 Federal Government Printers, Lagos, Nigeria, 1968.
5. *Moore, L.S. and Fleischman, A.I.* Subclinical lead toxicity. *J. Orthomol. Psychiatry*, **4** (1975) 61-70.
6. *Sayers, M.H.P.* Personal communication.
7. *Shannon, H.S., Williams, M.K. and King, E.* Sickness absence of lead workers and control, *Br. J. Ind. Med.*, **33** (1976) 236-242.
8. *Sofoluwe, G.O., Adegbola, A. and Akinyanju, P.A.* Urinary delta-aminolevulinic acid determinations among workers charging lead accumulation batteries in Lagos, Nigeria. *Arch. Environ. Health*, **23** (1971) 18-22.
9. *Stoke, J.* Plumbism in Rhodesia. *Cent. Afr. J. Med.*, **21** (1975) 119-124.