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Incidence of Hypertension among Lead Workers

A Follow-up Study based on Regular Control over 20 years

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Three hundred and sixty-four workers employed in an accumulator factory had at least three blood-pressure determinations during 1962 in a study of the relation between lead exposure and the incidence of hypertension. In this group 46 workers were found to have hypertension; the expected incidence was 51.

Two hundred and seventy-three of the total group, all over 35 years, had been employed for a sufficiently long time to be considered as having had a long-term exposure to lead. On the basis of urinary coproporphyrin tests, they were divided into a 'lead-affected' group (141) and a 'non-lead-affected' group (132). There were 22 persons with hypertension in the former group, and 20 in the latter. There was no significant difference in the appearance of hypertension in these two groups either from the standpoint of age or from the duration of exposure to lead.

Two hundred and sixty-five workers had been employed at the factory for 10 or more years, and 82 of these for more than 20 years. There was a positive correlation between the incidence of hypertension and the duration of employment, but no difference between the 'lead-affected' and 'non-lead-affected' groups. This observation is understandable in view of the increasing incidence of hypertension with advancing age.

The study shows that workers in an accumulator factory, in which the working conditions are inspected and controlled regularly and in which the workers themselves are examined regularly for the influence of lead, are not more prone to hypertension than the general population. In view of the possibility of vascular damage after exposure to lead, blood pressures in lead-workers should be watched, and treatment started early if hypertension is found.

Workers in the lead industry have been known to run the risk not only of acute lead poisoning with its well-known manifestations of anaemia, colic, neuritis, encephalopathy, etc., but also of chronic poisoning. Depending on the degree and duration of exposure, this latter is said to be followed by arteriosclerosis and cardio-nephro-cerebrovascular lesions of varying degrees. In England and Australia it has been considered that hypertension and chronic nephritis are relatively common consequences of working in the lead industry (Legge and Goadby, 1912; Oliver, 1914; Teleky, 1937; Fishberg, 1939; Nye, 1933), and in England chronic Bright's disease has been accepted as a manifestation of industrial lead poisoning and is therefore compensable. On the other hand, in America such a connexion has not been so readily accepted; according to Lane (1949) this is perhaps due to the greater turn-over of workers and consequently the shorter duration of exposure. In the tight communities in England there has been a good opportunity to observe the long-term effects of lead. The legislation introduced there in the 1920s regulating and improving plant hygiene has brought about a progressive decrease in deaths due to lead poisoning and has led to a virtual disappearance of chronic Bright's disease (Lane, 1949). Dingwall-Fordyce and Lane (1963) suggest that the excessive number of deaths due to cerebrovascular disease seen in the first quarter of this century among those with heavy lead exposure is now much reduced.

The fact that a short-term heavy exposure to lead can cause acute lead poisoning and that a long-term exposure can cause eventually a chronic and often fatal illness can be considered to be well documented. The manner in which a lesser exposure over a long period of time (highly applicable to present-day working conditions) affects the human organism is difficult to determine. Lane (1949) considered that continued surveillance of the blood picture, blood pressure, and urine in these workers remains necessary.

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The present investigation was carried out in order to obtain data on the incidence of hypertension in a large accumulator factory.

Investigation

At the accumulator works, AB Tudor in Nol, 30 km. north of Gothenburg, lead accumulators have been produced since the 1930s. When the plant opened, the working conditions were primitive, and regular medical check-ups and laboratory studies were not carried out. The main lines of control, which included regular medical examinations and various laboratory studies, were set down in 1941 by the late Professor Martin Odin, who himself was the plant physician from that time until his death in 1959. The routine investigations have consisted of blood pressure, haemoglobin, basophilic stippling of red blood cells, urine sediment, and a semi-quantitative urinary coproporphyrin determination according to the technique of Fischer and Waldenström (Waldenström, 1942). (An ether extract of urine is acidified with hydrochloric acid and examined in the spectroscope in cuvettes of different thickness. The smallest extract layer which still gives absorption lines at 550 mμ as well as at 600 mμ is determined. A layer of 20 mm. is considered to indicate metabolic influence from lead; a layer of 10 mm. or less indicates lead intoxication.) These studies are carried out every two months on those exposed to lead.

The present study has been carried out for two main reasons:

1. The turn-over of workers at this factory has been very low, so that a large number of individuals have had a long-term exposure to lead. The medical control has been carried out according to the same principles for the whole time so that a comparison between individuals is possible.

2. Data on the incidence of hypertension among lead workers with such a long period of surveillance are not available in the literature. It was possible to estimate the expected incidence of hypertension in our workers by making use of the extensive data of Humerfelt (1963) collected in Bergen, Norway. A similar broad investigation had not been available previously.

Material and Methods

Investigations were carried out on a total of 364 employed workers. Two hundred and sixty-five had worked at the company for 10 or more years, and, of these, 82 had worked there for at least 20 years. Blood pressures were recorded regularly. All readings were taken using the left arm with the workman in the supine position and after a rest of approximately one minute. A Recklinghausen mercury sphygmomanometer was used. During the year 1962 all subjects had at least three determinations of blood pressure by the cuff method, and the values obtained were categorized according to lead exposure and duration of employment. An individual has been considered to be hypertensive if the average diastolic pressure was 95 mm. Hg or over. An individual has been considered to be 'lead-affected' if at any time during his employment he had a positive urinary coproporphyrin test (20 mm. layer or less).

Results

Table I shows the frequency distribution of diastolic blood pressure in the 364 workers grouped according to age. Forty-six workers had hypertension. From Humerfelt's material, the expected number was 51. The material has been divided into a 'lead-affected' group (A) and a group considered to be 'non-lead-affected' (B). The former group consisted of 155 and the latter 209 persons. The Figure shows the age distribution for the two groups. The greater part of those in group B under the age of 35 years began employment during the decades 1950 and 1960, and for the most part have not been

TABLE I

FREQUENCY DISTRIBUTION OF DIASTOLIC BLOOD PRESSURE WITHIN THE VARIOUS AGE GROUPS

Diastolic B.P.	*Age*											*Total*
	15-	20-	25-	30-	35-	40-	45-	50-	55-	60-	65-	
60		1										1
70	5	3	2	2	3	3	1	3	2	1		25
75	3	4	3	3	1	3	6	1	1			25
80	10	10	8	9	16	18	13	15	12	4	2	117
85	2	3	3	3	5	7	6	8	4	5		46
90	3	3	4		11	22	11	17	21	11	1	104
95			1		1	1	3	3		4		13
100				1		1	1	3	2	6	1	15
105						1	1			2		4
110					1	1	4			3		9
115									2			2
120									1			1
130			1				1					2
Total	23	24	22	18	38	57	47	50	45	36	4	364

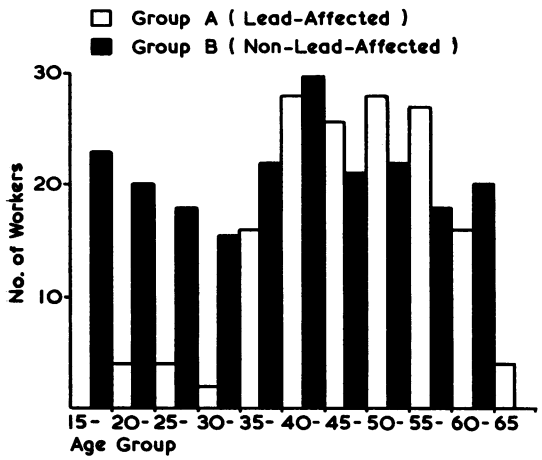

FIG. 1. Distribution according to age of lead-affected workers (group A) and workers not affected by lead (group B).

exposed to lead. There were altogether 87 workers under the age of 35, and of these 77 had had no exposure to lead. Therefore, in this material, one can only regard those over the age of 35 as being lead workers.

Table II shows the incidence of hypertension in groups A and B considering only workers of 35 years of age or older, *i.e.*, exposed to lead. From this table it can be seen that, as a whole, the 'lead-affected' group did not have a higher incidence of hypertension than the 'non-lead-affected' group.

TABLE II

INCIDENCE OF HYPERTENSION IN 'LEAD-AFFECTED' AND 'NON-LEAD-AFFECTED' WORKERS AGED 35 YEARS OR MORE

	Hyper-tensive	*Normo-tensive*	*Total*
Group A 'Lead-affected'	22	119	141
Group B 'Non-lead-affected'	20	112	132
Total	42	231	273

In Table III the two groups have been further subdivided according to age. This analysis shows that the observed incidence of hypertension according to age was not greater than expected, and that there was no significant difference between the 'lead-affected' and 'non-lead-affected' groups.

In Table IV the individuals have been grouped according to the duration of exposure to lead, in order to see if this parameter, in either group, correlated with the development of hypertension. If

TABLE III

AGE DISTRIBUTION OF 'LEAD-AFFECTED' AND 'NON-LEAD-AFFECTED' HYPERTENSIVES

	Age		
	35	45	55
Group A 'Lead-affected'	6	8	8
Group B 'Non-lead-affected'	1	7	12
Total	7	15	20
Expected*	10	18	21

*From data of Humerfelt (1963).

TABLE IV

INCIDENCE OF HYPERTENSION AND NORMOTENSION IN WORKERS AGED 35 YEARS OR MORE, ACCORDING TO DURATION OF EMPLOYMENT

Duration of Employment (years)	*Group A 'Lead-affected'*		*Group B 'Non-lead-affected'*	
	Normo-tensive	*Hyper-tensive*	*Normo-tensive*	*Hyper-tensive*
10-	10	3	32	3
15-	57	14	51	13
20-	38	3	24	3
25-	5	2	5	—
30-	2	—	—	—
Total	112	22	112	19

duration of exposure has any influence on the development of hypertension, it could be expected that the 'lead-affected' group might show an earlier occurrence of hypertension than the 'non-lead-affected' group. This table shows that there was no significant difference between the two groups in this respect.

Discussion

The effect of lead on the kidneys has been investigated with varying results. In animal experiments, however, one sees usually two types of injury, tubular and vascular. The tubular type is seen with acute, severe exposure (Bell *et al.*, 1925), so severe that such an exposure can hardly be considered to be applicable to the low-dose, long-term exposure seen in man, which, on the other hand, is considered to cause blood vessel damage (Calvery, 1938; Fishberg, 1939). Fishberg (1939) has described arteriosclerotic changes in the renal vessels of lead workers, and in some cases even arteriolar necrosis, such as is seen in malignant hypertension.

The clinical observations of Oliver (1914) and Legge and Goadby (1912) gave strong support to the concept that chronic Bright's disease was a con-

sequence of severe, chronic lead exposure. In the United Kingdom the evidence for this is considered to be so strong that chronic renal disease discovered in a lead worker is sufficient for compensation as an occupational injury. In 1933 Nye reported 34 patients who in their youth had received such a severe lead poisoning that they developed peripheral neuropathy. In later life, 24 of these were shown to have kidney damage and hypertension. Lane (1949) described nine deaths from renal disease with hypertension in a group of about 150 workers, all of whom had had approximately the same degree and duration of exposure. All these men had been severely exposed to lead during the 'bad days', and the mean age at death of this group was 48·4 (range 42-52) years. In only one case could an hereditary factor for hypertension be considered on the basis of the family history, but here also the father had died at the age of 49 years of lead poisoning. The parents of the other patients had lived to be 60 to 80 years old. Aub, Fairhall, Minot, and Reznikoff in their monograph on lead poisoning (1926) say that it has never been demonstrated satisfactorily that lead causes an increase in blood pressure, and Hamilton (1934), after a review of the literature, concluded that there was no evidence for an increased frequency of hypertension in lead workers.

Dingwall-Fordyce and Lane (1963) undertook a retrospective study of 425 lead workers pensioned during the years 1926-60. Of these, 184 had died by the time of the investigation, and they analysed the cause of death with respect to the degree of lead exposure. A similar analysis was carried out on 153 workers in the accumulator industry who died during the years 1946-61 while still employed as lead workers, before they had reached pensionable age. They found a significant excess of deaths from all causes among those pensioners who had been exposed to the greatest lead hazard. They also found that deaths from vascular lesions of the central nervous system were markedly increased over the expected rate in the groups with the greatest exposure to lead. They suggested that in the first quarter of this century men who were exposed to lead may have had their lives shortened. However, with improved working conditions, it appeared to them that the excess of central nervous system vascular lesions had decreased. This retrospective study, however, did not investigate the relation between lead exposure and hypertension, which is important not only in the development of vascular lesions but also in regard to future measures for the surveillance of workers in the lead industry.

The reduction in the excessive death rate following legislation in England in the 1920s, which markedly improved working conditions, also speaks strongly in favour of a connexion between severe lead exposure and reno-vascular and cerebrovascular insults. It is possible that it was just these improved working conditions, decreasing markedly the exposure to lead, that prevented Belknap (1936), Dreessen, Edwards, Reinhart, Page, Webster, Armstrong, and Sayers (1941), and Lane (1949) from finding any significant increase in the frequency of hypertension in workers in the accumulator industry. Most of the workers in the first two of these studies had been exposed to lead for less than 10 years, whereas those investigated by Lane, electric accumulator pasters, had 20 or more years' exposure. In the present study, 265 workers had been exposed for more than 10 years, and 82 of these for more than 20 years. An increased frequency of hypertension could not be demonstrated either in the total material or when broken down according to age group. Furthermore, a subdivision into 'lead-affected' and 'non-lead-affected' groups also failed to demonstrate any significant difference in the incidence of hypertension.

Probably the increased incidence of cerebrovascular disease in lead workers should be related to vascular damage rather than to hypertension. An existing hypertension would however increase the risks from vascular damage. It appears essential to determine blood pressures in lead-exposed workers regularly and to start adequate treatment at an early stage.

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