HARRISON RADIATOR DIVISION

GENERAL MOTORS CORPORATION

LOCKPORT, N.Y.

A Preliminary Study of the Lead Exposure Associated with the Normal Operation of a Plant Engaged in the Manufacture of Automobile Radiators and Heaters.

by

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in the

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KE 0003672

Introductory Comment on the Study of Occupational Lead Exposure

In view of the fact that the general methods of plant study employed by the Kettering Laboratory of Applied Physiology differ somewhat from those used generally in industrial surveys, it may be of some interest to refer to the available methods and to point out some of the advantages and disadvantages in their use.

(1) A single critical inspection of operating conditions in a plant, made by an experienced person, will provide a general idea of the opportunities for lead exposure and some knowledge of the location of the greatest hazards. Periodic inspectionsof this type will prove to be more useful than one for obvious reasons, and moreover they are a useful adjunct to any other method of study and control.

(2) A single clinical survey of groups of workmen representative of various occupational activities in the plant will define the general levels of lead exposure in terms of clinical evidences of lead absorption and will tend to show the most hazardous types of employment.

(3) Maintained medical supervision, with periodic examinations of all employees and with carefully recorded information as to the character and incidence of illness, the amount of lost time and the extent of the labor turnover, will eventually show the hygienic significance of plant conditions, as no other method can do, and if such data are combined with detailed knowledge of plant procedures,

including changes in equipment and plant practice, a clear composite picture of the hygienic situation can be obtained. All other means for evaluating the hygienic significance of lead exposure, as contrasted with its mere quantity, depend finally upon sound clinical data. Indeed the belief is well entrenched in the medical as well as the non-medical mind that individual susceptibility to lead intoxication is extremely variable and that even the smallest quantities of absorbed lead may, in time, result in lead poisoning in certain persons, while certain other persons may receive no injury from massive dosages. The complete acceptance of such a point of view,(which in our judgment is erroneous), implies that there is little or no value in any type of measurement of the quantity of lead absorbed or potentially available for absorption on the part of workmen. So long as such an opinion is applied with even partial effectiveness in medico-legal and trade-union phases of industrial practice, business management cannot afford to operate plants associated with any of the lead trades without competent and adequate medical service. Fortunately, evidence has accumulated to demonstrate the existence of a relationship between quantity of lead absorption and the occurrence of lead intoxication. Various expressions have been given to this relationship, and it cannot be said that there is complete agreement among authorities as to the safe limits of occupational lead absorption. Nevertheless it is now fairly generally accepted that there is a limit of lead exposure above which the danger of lead poisoning becomes progressively greater among a plant population, and below which cases of poisoning will either not occur at all or will be of an essentially harmless type. Thus it becomes feasible to estimate the significance of lead exposure in quantitative terms.

(4) Exposure to particulate lead compounds in dusty lead trades may be estimated by the analysis of air samples taken from representative parts of the plant. Obviously the use of this method involves certain assumptions; - (1) that the inhalation of particulate lead compounds constitutes the only important source of lead absorption on the part of the plant population; (2) that the lead particles found in air samples may be breathed in and retained within the lung to a more or less uniform extent by the workmen in the area. Various technical procedures have been employed to increase the practical reliability of this method. Frequent sampling over prolonged periods tends to reduce sampling errors. The manner of preparing samples for analysis also tends to exclude particles of lead compounds which because of their size are incapable of inhalation or of retention in the lung. Moreover the greater practical significance of inhaled lead in comparison with ingested lead has been fairly well demonstrated by experience and experiment, and is now almost universally recognized. On the other hand, lead poisoning can and does occur through ingestion of lead compounds, and unless precautionary measures are effectively taken against carelessness and uncleanly habits on the part of workmen, ingestion can occur to such an extent as to void conclusions arrived at by the analysis of air samples.

(5) It has been shown that the magnitude of the lead exposure in a plant can be determined by the study of the lead content of the excreta of the employees. Briefly stated, the lead content of a single evacuation of the feces of a workman serves to show the magnitude of his lead exposure for the period of from twenty-four to seventy-two hours previous to the collection of the sample. A large proportion of all inhaled lead is collected in the nose and

pharynx and eventually reaches the alimentary tract, through which it passes, in large measure unabsorbed. It becomes, thereby, an indicator of a man's gross lead intake for the period represented by the feces accumulated in the lower bowel - a period which experience has shown to be approximately twenty-four hours. The concentration of lead in a large sample of urine (collected over a period of forty-eight or seventy-two hours) seems to vary with the lead content of the body and therefore discloses the general magnitude of the lead absorption over a considerable period of time. The correlation of these data on the excreta with the observed conditions in industrial plants and with the clinical findings in the plant populations has not been made over a wide enough range of exposures to give a final expression of the relationships which obtain. To this extent every new industrial situation which comes under study is of intense interest, experimentally, in that the information made available fills one or more of the gaps in our understanding of the problem. Despite this distinctly experimental aspect of the work, the knowledge of plant conditions gained by this type of study is invaluable in hygienic control and is of the greatest practical value in the solution of individual clinical problems.

It is desirable that initial studies made in a plant be supplemented by further observations. If one is to know, with certainty, whether or not a given magnitude of exposure is capable of producing lead intoxication, he must be sure that the conditions originally observed are maintained (or improved) and he must also be sure that illnesses that develop among the employees are adequately studied and recorded. The staff of this Laboratory has every interest in promoting the most adequate type of plant medical service and in rendering every possible aid in doubtful or difficult cases, to the end that the conclusions once arrived at may be subjected to critical check. As industrial hygienists we attempt to recognize and to eliminate hazardous lead exposure, while at the same time, as experimentalists, we seek to build a sound foundation for a sharp and practical differentiation of hazardous from non-hazardous exposure. From the viewpoint of plant management these may appear to be somewhat incompatible objectives, but they need not be if adequate and accurate information is available. Actually both objectives may be achieved after an initial thorough survey, if good clinical data on the employees can be had and if questionable cases can be studied by adequate laboratory methods.

The Technique and Scope of the Present Investigation.

For a variety of reasons, this study was carried out on a small scale - too small in fact to justify more than tentative conclusions as to the conditions existing in the plant as a whole. It is probable that the data portray the situation fairly with respect to lead exposure, for reasons which will be pointed out in later paragraphs, but they cannot properly be regarded as more than the results of selective sampling rather than the general random sampling which would be required to detect unrecognized factors in the exposure.

A preliminary inspection of the plant was made, on the basis of which employees were selected for study from what appeared to be the commonest and the most hazardous occupations. Moreover, a number of subjects with the longest records of employment were chosen, and in recognition of the generally accepted belief that women are more susceptible than men to injury from lead absorption, several female employees were included. The total number of employees was approximately

2500, of whom about 500 were women. Most of the employees worked in large groups that performed the same type of work and had the same basic conditions of exposure. Thus some 1500 persons worked on assembly lines in large rooms in which the lead exposure was of the same type and of the same general order of magnitude. In such a group gross variations in individual lead absorption are scarcely to be anticipated, except on the basis of variability of conduct in matters of cleanliness and personal hygiene. Thus there is some justification for believing that the thirty-six persons selected for examination (of which five were women) have provided fairly characteristic evidence of the lead exposure associated with the various types of work in the plant.

Table I shows the age grouping of the persons selected for examination. A wide range has been covered. It is of some importance to note that the mean age of the group examined is unusually high, fourteen out of the thirty-six being forty-five years of age or older. The group is probably not characteristic of the plant population, and the fact has no particular significance with respect to any increased likelihood of the occurrence of lead poisoning, since older people, if different in any wise, are believed to be somewhat less susceptible than the young. However, certain of the degenerative diseases of middle and late life have been linked up with the effects of lead absorption. Arterio-sclerosis, for example, has long been regarded (on inadequate evidence) as a sequel of prolonged lead exposure, and although it is quite clear that there are other causes, the coincidence of arterio-sclerosis and a history of prolonged employment in a lead trade is regarded by many physicians and medical writers as sufficient evidence to warrant the conviction of lead as the offending agent. On this account, and in illustration of the point under discussion, the frequency distribution of systolic blood pressures as observed in the group is listed in Table 2, and the ages of the persons showing abnormally high blood pressure are shown in parenthesis. The significance of these data will be referred to later.

Table 3, in which the subjects are grouped according to the duration of their lead exposure in the plant, merely shows that the group has been well selected with respect to variety in length of exposure. As a sampling procedure this is advantageous. The significance of prolonged exposure to lead compounds is well appreciated - indeed too much emphasis has been placed upon the time factor by the theorists in industrial hygiene. It is not so well recognized that lead intoxication occurs more commonly among new employees than among the habituated (perhaps naturally selected) and long employed members of a plant population.

Each person in the selected group was subjected to detailed questioning. Every effort was made to gain his confidence and to induce him to talk freely about any ailment, illness or injury which might, in his mind, be associated with his employment. Leading questions were avoided at first so as not to color the picture, but were employed subsequently with reference to specific symptoms, so as to miss no significant information. The employee was assured that any information which he might wish to withhold from his employer would be kept in confidence, to the end that his position would in no way be endangered by any statement he might make. (Confidential information was noted on the original examination record and deleted from examination records furnished with this report. None of it had any relation to lead exposure or lead intoxication.) Information as to general plant conditions, the attitude of other workmen toward the potential hazards of the plant, and the occurrence of illness among other employees, was sought from each employee, after explaining the reasons for collecting such information. The men appeared to respond with the utmost frankness.

A careful physical examination was given each person, together with such laboratory tests as might be expected to yield data specifically useful in the recognition of abnormal lead absorption. The scope of the examination is shown by the individual records. It is believed that no specifically useful clinical or laboratory test was omitted. Obviously, the most specific information was to be obtained from the analytical data. It has been our experience, that the most meticulous precautions must be employed in avoiding the contamination of samples of the excreta of workmen in lead trades. For this reason, in part, it was believed to be advisable to collect a limited number of samples of blood and small samples of urine at the time of the physical examinations. These, obtained by the examiner in person, could scarcely be contaminated under the excellent conditions of freedom from plant dusts that prevailed in the examining rooms, and thus could be used as a check on the samples obtained by the employees. For the latter, each employee was provided with a jug for the collection of his urine, and a jar for the collection of a single fecal evacuation. Instructions were given to the group as to the manner of collecting the samples, and when the containers were shipped from the laboratory, ready for use, a sheet of printed instructions was provided for each person. The containers were delivered to the homes of the employees, sealed in paper bags, and were to be used only during the hours away from the plant. The samples were collected promptly and arrived at the laboratory in good condition.

The data obtained on each employee were studied carefully as a clinical whole in search of evidence of lead absorption or lead intoxication. Specifically important items on the group were assembled in tabular form for comparison with similar data from other groups of subjects. The number of employees was too small for adequate statistical handling, and the calculation of mean values for most of the tabulated qualities was not justified.

The Results of the Investigation.

The initial inspection of the plant revealed several sources of potentially hazardous exposure. In decreasing order of importance these appeared to be, (1) the operation of tinning castings in the foundry with a 50-50 tin lead alloy, (2) the polishing of metal strips, and the sweeping, in the electroplating department, (3) the soldering operations in the service department, (4) the soldering operations in special repair work at the end of the assembly lines, (5) tank assembling (no local ventilation), (6) dismantling operations in the thermostat department, (7) removal and inspection operations at the exit from the core dips, (8) soldering operations on the less modern and less adequately ventilated assembly line. The most modern assembly line appeared to be adequately safeguarded, and except for possible local variations in ventilation, hazardous exposure seemed improbable. Likewise the area around the core dips appeared to be safe, except for solder on the floor nearby which suggested the need, perhaps, for improved housekeeping. In general, also, the thermostat and the heater departments gave little evidence of dangerous exposure. The magnitude of the exposure at any point was open to conjecture, but it was not believed to be of serious proportions (except in the foundry), an opinion which was confirmed by the examination of data of air analyses carried out by others.

The histories and the physical examinations on the employees failed completely to develop any evidence of significant lead absorption. The subjective symptoms of constipation, fleeting colic-like pains, vague intestinal disorders, morning anorexia, soreness, stiffness and weakness of muscles, and indefinite ill health, many of which are of common occurrence in certain hazardous lead trades, were conspicuously absent. Objectively one did not see a single set of clinical findings suggestive of serious lead absorption. Pallor was of infrequent occurrence among the men and not of the pronounced type seen in a "leaded" group of men. No suggestion of extensor weakness or of atrophy of the muscles of the upper extremities of any employee was found. No definite lead line was seen on the gums of any person, although there were two instances of faint discoloration of a very small gum area doubtfully suggestive of such deposits. Since pyorrhoea was present in the entire male group, with the exception of the edentulous employees, the failure of occurrence of a single definite lead line is strong evidence of the insignificance of the lead exposure.

The clinical examination of the urine, and the microscopic examination of the blood also failed to adduce any evidence of lead absorption. The data of the leucocyte counts are grouped in Table 4. The one definitely abnormal high result was obtained in a man with a low grade cystitis and a bladder fistula (Benzein). Tables 5, 6 and 7 show the other data on the blood, classified according to their frequencies of occurrence within the group. It is significant that a mild to severe reduction of the erythrocyte count was observed in more than half of the individuals in the group examined, but the lack of any specific relationship of these findings to absorption is indicated by the failure of occurrence of significant numbers of "stippled" erythrocytes. In most instances these anemias had their origin, in all probability, in the chronic infections of the nose and throat and the teeth of the employees. In one instance (Cleri) this explanation of the anemia may be doubted. In this employee (female) the erythrocyte count (2,900,000) and the hemoglobin determination (75%) justify further clinical study and treatment. The others are within the range associated with low grade infection and chronic toxemia.

The clinical findings, while insignificant in relation to lead absorption, were otherwise of interest and importance. Chronic rhinitis was an almost constant finding among the persons examined, and instead of the common mild congestion and edema seen among workmen in general, there was a glairy bogginess of the naso-pharyngeal mucosa which denoted the presence of some irritant. The examiner's early suspicion in this regard was confirmed by the discovery of several ulcers of the nasal septum and by two cases of complete perforation of the septum, presumably derived from the acid fumes associated with soldering.

The teeth of the employees were in deplorably bad condition with but few though very striking exceptions. One first suspected that exposure to acid fumes may have had some influence on the incidence and the severity of dental caries, which in the small group of men appeared to be unusually extensive. However this tentative idea was negatived by the examination of the female employees, who had the characteristic naso-pharyngeal irritation (one had a perforated septum),

KE 0003684

unusual number of persons in the high age brackets among the persons examined, and the data on the blood pressure have been shown in Table 2. There is a high degree of correlation between age and elevation of the blood pressure within the group, but there were instances of high blood pressure among relatively young individuals, a fact which is also in keeping with present knowledge of vascular disease. Persons with progres arterio-sclerosis must be regarded as something of a hazard in that cereb accidents may occur under stress and at inopportune times. In a trade involving lead exposure, such persons may become liabilities for other reasons. Unless it could be demonstrated that the lead exposure of the plant is of insignificant proportions, any disabling sequellae of arterio sclerosis, or of the clinical syndrome known as Bright's Disease, might be regarded in orthdox medical and compensation circles as an expression of the effects of prolonged lead absorption. It is not suggested that

ons in the upper age groups, or persons with clinical evidence of ular disease should not be employed, especially when they have served but who, without exception, had clean and essentially normal teeth. One, therefore, suspects that the bad oral conditions encountered were due largely to lack of personal care of the teeth on the part of the men. Indeed, the records show that the men as a group made little or no attempt to exercise the ordinary care of their teeth. Regardless of its cause, this situation is a potentially dangerous one in an industry, and particularly in one involving lead exposure, since it is likely to result in a relatively high incidence of illness, which in turn might be attributed erroneously to the effects of lead. It may well be that some provision will be required for the treatment of the oral sepsis, and for the inculcation of the principles of oral hygiene among the employees of the plant, as a part of the plant medical service. If so, precedent for such a course can be found in other industries.

Mention has been made of the presence of an

The results on the urine samples are shown in Table 9. It should be recalled that the small samples of approximately 100 cc were obtained under the supervision of the examiner in order to check the possibility of contamination in the collection of the large samples. The results on the small samples, when compared with those on the large samples, demonstrate that the large samples were collected with due care. The small samples yielded both slightly higher and slightly lower results than the large samples, and in six cases out of twenty-nine on whom both results were available, the two results were widely variant. The explanation of these variations has been provided through previous experimental observations. We have found that urinary samples taken from hour to hour vary considerably in their lead content, partly because of variation in the lead exposure of the occupation but largely because of variation in the intake and output of water. Scanty water output on the part of the kidneys produces a relatively high urinary lead concentration, while profuse water output results in the reverse. Those variations tend to be smoothed out through the collection of a large sample over a period of hours or days, and therefore such a sample portrays the status of the individual with respect to lead absorption with greater accuracy than does the small spot sample. Despite this greater usefulness of the large samples, it is apparent that the small samples yielded substantially the same general information as the large. In twenty-six cases the results on the two samples were in substantial agreement. Moreover the mean results on the two sets of samples of urine are not significantly different, statistically, and from the physiological viewpoint they have the same significance. When compared with results obtained by similar methods on normal persons without occupational lead exposure, they demonstrate conclusively the occurrence of occupational lead absorption. They do not exceed normal values by a wide margin, and they are not of the magnitude that we have found in groups of workmen in trades in which we have found clinical evidences of lead intoxication. For the sake of comparison, data on normal persons, and on groups of persons in a frankly hazardous lead trade are given in Table 10. It is of interest to note the relatively narrow range of variation in the results obtained in the case of normal persons, and the workmen in the plant under study (Table 9), as contrasted with those in the workmen of the definitely hazardous plant. The latter plant had a variety of hazards of different magnitude, and cases of intoxication were found among the more heavily exposed workmen, who also showed the higher rates of urinary lead excretion. The plant under study, on the contrary, had much the same type of hazard throughout, and there was little evidence of gross variation in the severity of the potential exposure. The analytical results are in satisfactory agreement with that observation.

The analytical results on the feces are given in Tables 11 and 12. Table 11 groups the results on the entire fecal samples while Table 12 shows them on the basis of the lead present per gram of fecal ash. The latter means of presenting the data while not wholly satisfactory from a physiological point of view, is an attempt to compare the individuals on some time or quantity basis. We have shown previously that the fecal output of lead of persons without occupational lead exposure is approximately 0.25 milligrams per day. On this basis the mean figure of 0.606 ± 0.073 for the group studied is definite evidende of abnormal lead intake. Expressed in terms of the ash, the normal fecal lead has been found to be about 0.08 milligrams per gram of ash. The value of 0.136 ± 0.013 is therefore significantly high, indicating also the occurrence of someshat abnormal exposures. An interpretation of the significance of the results on the feces is complicated by the two high results of 2.49 and 3.25 milligrams per sample. These results are patently abnormal to the group. Whether they represent an unusual exposure on the part of these two men on the day before the samples were obtained, or whether they are the result of unusually high lead content in the food for that particular day, cannot be determined. From the point of view of plant safety it is wiser to interpret them as due to exposure in the plant than to ignore them as abnormal and unrelated to occupational exposure. There is some justification for following the former course. If one examines the entire record of these two men (C. Salmons and Stanhope) it will be seen that both men showed relatively high results in small and large samples of urine, in evidence of relatively high lead exposure and absorption, although neither showed any clinical evidence of toxic lead absorption of even a doubtful variety. The urinary results, however, are not in keeping with any such daily exposure as is represented by two or three milligrams of lead per day in the feces. Therefore we must conclude that such an exposure was of infrequent occurrence for these men and for other men in this plant.

From the combined data on the urine and feces, it is quite clear that the employees in this plant have been exposed to lead over and above that which was ingested with their food and drink. It is also evident, especially from the urinary results, that the occupational lead exposure was not so great as that occurring in many other and recognizedly hazardous lead trades. From the lack of clinical evidence of lead intoxication it is apparent that the lead absorption incident to employment in this plant was not generally sufficient to produce frank cases of lead poisoning. It is doubtful if lead poisoning may be expected to develop under conditions of absorption such as those represented by the mean urinary lead output of the group of workmen.

On the other hand there were individual cases among the group with a uniformly high lead content of blood, urine and feces. Moreover it is conceivable that the subjects selected for study did not represent the complete range of exposure and of excretory reaction to exposure; i.e. that there may have been employees with somewhat greater exposure than the greatest represented in the group selected for study. In any case the individuals who portray the effects of maximal exposure among our group are of most significance, since these persons, if any, would be expected to show borderline evidences of intoxication. Actually, there is only one person in the group (Scott), in whose case there was such a combination of clinical and analytical findings as to suggest the existence of incipient or mild plumbism. Certain clinical findings in this man could be interpreted as deriving from lead absorption. The low erythrocyte count and the low hemoglobin, with stippling to the extent of 16 per 50 fields, when combined with a dubious lead line, slightly hyperactive reflexes, tremor of the hands and a slight tendency to constipation are, at least, suggestive. The urinary lead concentration of 0.17 mg. per liter in the spot sample was confirmed in its significance by the finding of 0.15- mg. per liter in the large sample, this being the highest result obtained in the latter group of samples. This man, in our opinion, should be kept under observation, not so closely as to induce anxiety on his part, but sufficiently closely to make possible the detection of any trend in the state of his health and well being.

<u>Discussion and Recommendations</u>

The comments made in the presentation of the data yield a basis for interpretation, to which little need be added. However, certain general conclusions which are justified by our experience should be added. From our previous studies we have formed the opinion that a maintained urinary lead excretion of approximately 0.20 mg. per liter or more denotes a dangerous degree of lead absorption, and that under conditions in which such results are found frequently or regularly, in a group of workmen, lead poisoning of moderate to severe degree will occur. Persons excreting lead to the extent of 0.15 mg. per liter, or thereabouts, under conditions of regular exposure are in a doubtful group, with relation to the probability of their becoming poisoned, but up to the present we have reason to believe that intoxication is likely to be mild and fleeting among such persons, if indeed it occurs at all. Therefore the mean figure of 0.079 mg. per liter of urine in the group studied denotes, we believe, a substantially satisfactory state, and one in which plumbism is scarcely to be expected. The questions which remain to be answered, with respect to this plant, and its employees, are as follows: (a) Did the survey cover the plant population in such a way as to disclose the results of the greatest lead exposure in this plant? (b) Are the few high results obtained characteristic of individuals who have had some unusual magnitude of exposure, or do they represent the chance occurrence of a temporary high lead output on the part of individuals who usually excrete lead at lower levels.

No adequate answer can be given to these two questions from the available data. Observations would have to be made on a much greater number of employees to yield the answer promptly. On the other hand, the answer can be obtained by collecting further data over a period of time. It is recommended, therefore, that doubtful or suspected cases of plumbism, and cases presenting symptomatic and physical findings that might mistakenly be attributed to lead absorption be studied carefully as they come to the attention of the plant physician. In such instances the result of a complete physical examination should be checked by the analysis of suitable samples of blood and excreta for decision as to the safety of the plant. If certain factors which influence the lead exposure at various times and under various conditions of operation have been missed by the present survey, (which admittedly is not wholly adequate), such factors will almost certainly come to light, and can be dealt with as the circumstances require. Meanwhile, obviously, continued attention must be paid to matters of good housekeeping, to the maintenance of ventilating equipment, and especially, to the possibility that the exposure may be modified for better or worse through the introduction of new operations and procedures, within the plant. The combined data obtained from careful supervision of plant activities, from careful medical supervision, and from the analytical study of unusual cases will yield valuable information from both a scientific and practical point of view.

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TABLE I

Distribution of Workmen According to Age

Age in Years	Number of Persons
20 - 24	(1) 3
25 - 29	(1) 2
30 - 34	3
35 - 39	(2) 6
40 - 44	8
45 - 49	(1) 4
50 - 54	0
55 - 59	3
60 - 64	6
65 - 69	0
70 - 74	1
Total	36

Numbers in parentheses refer to female workers and are included in the total under the respective rubric.

TABLE 2

Distribution of Workmen

According to Systolic Blood Pressure

Systolic Blood Pressure in mm. Hg.		Number of Persons
100 - 109		3
110 - 119		9
120 - 129		5
130 - 139		6
140 - 149	*	7 (31)(36)(38)(46)(56) (56)(62)
150 - 159	*	1 (49)
160 - 169	*	1 (60)
170 - 179	*	1 (42)
180 - 189	*	1 (63)
190 - 199	*	1 (62)
Total		35

* Clinically abnormal blood pressure levels.

Numbers in parentheses represent the ages of the persons within the limits of the rubric.

TABLE 3

Distribution of Workmen

According to Duration of Exposure

Period of Exposure in Years	Number of Persons
1 - 2.9	6
3 - 4.9	(2) 6
5 - 6.9	(1) 4
7 - 8.9	(1) 4
9 - 10.9	7
11 - 12.9	3
13 - 14.9	1
15 - 16.9	1
17 - 18.9	
19 - 20.9	(1) 3
21 - 22.9	
23 - 24.9	1
Total	36

Numbers in parentheses refer to female workers and are included in the total under the respective rubric.

TABLE 4

Distribution of Workmen

According to Leucocyte Count

Leucocytes per cu. m.m.	Number of Persons
4000 - 4999	4
5000 - 5999	8
6000 - 6999	5
7000 - 7999	7
8000 - 8999	7
9000 - 9999	3
10000 - 10999	1
11000 - 11999	
12000 - 12999	
13000 - 13999	1
Total	36

TABLE 5

Distribution of Workmen

According to Erythrocyte Count

Erythrocytes in Thousands per cu.m.m.		Number of Persons	
2750 - 2999	*	(1)	1
3000 - 3249	*		
3250 - 3499	*		
3500 - 3749	*		
3750 - 3999	*	(1)	7
4000 - 4249	*		6
4250 - 4499	*	(2)	8
4500 - 4749		(1)	9
4750 - 4999			1
5000 - 5249			2
5250 - 5499			
5500 - 5749			2
Total			36

* Significantly low values

Numbers in parentheses refer to female workers and are included in the total under the respective rubric.

TABLE 6

Distribution of Workmen

According to Hemoglobin of Blood

Hb. in Per Cent	Number of Persons
70 - 74	* (1) 1
75 - 79	* 1
80 - 84	(1) 3
85 - 89	(1) 4
90 - 94	(2) 14
95 - 99	8
100 - 104	5
105 - 109	
Total	36

* Significantly Low Values

Numbers in parentheses refer to female workers and are included in the total under the respective rubric.

KE 0003696

TABLE 7

Distribution of Workmen

According to Stippling of Erythrocytes

Stippled Erythrocytes per 50 Fields	Number of Persons
0 - 1	21
2 - 3	6
4 - 5	0
6 - 7	2
8 - 9	1
10 - 11	1
12 - 13	0
14 - 15	* 2
16 - 17	2
18 -	1
Total	36

* Values which may be but are not necessarily associated with abnormal absorption of lead.

Table 8

Distribution of Workmen

According to Lead in Blood

Mgs. Pb per 100 grams	Number of Persons
0.0 - 0.019	1
0.020 - 0.039	4
0.040 - 0.059	5
0.060 - 0.079	4
0.080 - 0.099	1
Total	15 *

Mean	0.05 ± 0.004
Standard Deviation	± 0.022

* 11 of these were obtained at the time of our examinations, 4 were obtained at other times by the plant physician.

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TABLE 9

Distribution of Workmen

According to Lead in Urine

Mgs Pb per Liter of Urine	Frequency: In Small Samples	Frequency: In Large Samples
0.0 - 0.019	4	0
0.020 - 0.039	6	5
0.040 - 0.059	8	6
0.060 - 0.079	7	11
0.080 - 0.099	3	5
0.100 - 0.119	6	2
0.120 - 0.139	3	4
0.140 - 0.159		3
0.160 - 0.179	1	0
Total	38 *	36
Mean	0.068 ± 0.004	0.079 ± 0.004
Standard Deviation	± 0.04	± 0.04

* 29 of these were obtained at the time of our examinations. 9 were obtained at other times by the plant physician.

TABLE 10

Distribution of Groups of Persons with Variable Lead Exposure
According to Concentration of Lead in Urine

Mgs.Pb per Liter of Urine	Frequency: Daily Samples from Two Normal Persons Studied Under Laboratory Conditions	Frequency: Persons Employed in an Occupation with no Lead Exposure	Frequency: Persons Employed in a Hazardous Lead Trade
0.00 - 0.049	196	17	
0.05 - 0.099	2	6	6
0.10 - 0.149			9
0.15 - 0.199			10
0.20 - 0.249			8
.25 - 0.299			3
0.30 - 0.349			2
0.35 and over			3
Total	198	23	41
Mean	0.018 ± 0.004	0.037 ± 0.004	0.201 ± 0.013
Standard Deviation	± 0.009	± 0.025	± 0.121

TABLE 11

Distribution of Workmen

According to Lead in Feces

Mgs Pb per Sample of Feces	Number of Persons
0.00 - 0.149	4
0.15 - 0.299	8
0.30 - 0.449	8
0.45 - 0.599	4
0.60 - 0.749	2
0.75 - 0.899	2
0.90 - 1.049	3
1.14	1
1.23	1
2.49	1
3.25	1
Total	35

Mean	0.606 ± 0.073 *
Standard Deviation	± 0.640

* By the omission of the last two results (2.49 and 3.25) which appear not to belong in this set of data, the mean becomes 0.470 ± 0.038, with an S. D. of ± 0.318.

TABLE 12

Distribution of Workmen

According to Lead Per Gram of Fecal Ash

Mgs.Pb per Gram of Ash	Number of Persons
0.00 - 0.049	3
0.05 - 0.099	15
0.10 - 0.149	8
0.15 - 0.199	2
0.20 - 0.249	4
0.25	1
0.44	1
0.63	1
Total	35

Mean	0.136 ± 0.013 *
Standard Deviation	± 0.116

* By the omission of the one last result (0.63) which appears definitely abnormal to this universe of data, the mean becomes 0.122 ± 0.010, with an S.D. of ± 0.083.

Key to Symbols and Abbreviations in Large Table

1. Age is recorded to the nearest integer
2. Marital status - M, married; S, single
3. Years in this plant does not refer to present occupation, and is not necessarily related to a single type or severity of exposure.
4. Habits of sleeping - G, good; F, fair.
5. Constipation - O, none; ± slight or doubtful; + definite history; ++ persistent.
7. General health - - unsatisfactory; ± questionable; + satisfactory;
8. Fatigue - O none; ± occasional; + definite complaint.
9. Headache - O none; ± occasional; + frequent complaint.
10. Appetite - G, good; F, fair.
11. Nocturia - O none; ± occasional; + frequently or regularly.
14. Nutrition - - poorly nourished; ± fair state of nutrition; + good nutrition; ++ obese.
15. Musculature - - poorly developed; ± fair development; + well developed; ++ heavily muscled.
18. Blood Pressure - A dash or period separates systolic and diastolic
19. A.R., acute rhinitis. C. R., chronic rhinitis; Turb. Ed., edema of turbinates; T.Hyp. hypertrophied turbinates; I.T., inferior turbinate; S.D.L. - S.D.R., septum deviated to left or right; Par. ob. partial obstruction; Ph., pharyngitis; O, normal.
20. Ca. caries; edent. edentulous; Part.Dent., Partial denture; dev. devitalized, ging. gingivitis.
21. Pyorrhoea - O none; ± slight and localized; + present generally; ++ severe, +++ very severe.
23. Extremities - Healed fr., healed fracture; V.V. varicose veins; Derm. dermatitis.
26. Tremor - O none; ± inconstant; + definite.

6.Recent Wgt.Loss	0	0	0
7.General Health	+	+	+
8.Fatigue	0	0	0
9.Headache	0	+ Eyes	0
10.Appetite	G	G	G
11.Nocturia	0	0	0
12.Height(inches)	73	69	66
13.Weight	175.0	152.0	180.0
14.Nutrition	+	+	++
15.Musculature	+	+	+
16.Pulse	84	72	80
17.Temperature	98.8	99	98.6
18.Blood Pressure	144.90	136.72	190-100
19.Nose and Throat	C.R.	0 Turb.Ed.	C.R.
20.Teeth	Ca	Sl.Gin. 5 out	Worn
21.Pyorrhea	+	0	+
22.Lead Line	0	0	0
23.Extremities	Healed Fr.V.V.	ok	Small Varice
24.Abdominal Reflexes			
Upper	0	0	0
Trunk	0	0	0
Lower	0	0	0
25.Dynamometer R	120	95	90
L	120	95	90
emor	0	0	
Acidity	0	+	+
ds	8.15	7.95	9.00
29.RBC -T sands	4,520	5,000	5,160
30.Haemog in Dare	90	95	95
31.Stippling (50 F)	2	1	0
32.Polys - Percent	69.5	48.0	52.5
33.Band F rms	3.5	2.0	3.5
34.Lympho ytes	18.5	40.5	36.5
35.Mono.	5.5	7.5	6.5
36.Eosin.	2.5	1.0	0.5
37.Baso.	0.5 Myl.	1.0	0.5
38.Mgs.Pb/100 gms.Blood	0.065	0.045	0.09 (conte
39.Mgs Pb/Liter (Spot)Urine	0.07	0.12	0.11
40.Mgs Pb/Liter(Large)Urine	0.06	0.10-	0.03-
41.Mgs Pb-Sample Feces	0.11	0.62	0.79
42.Mgs Pb-Gm.Ash Feces	0.06-	0.11	0.23

KE 0003704

Table 13

Name	Moran	C. Salmons	Petty	Wick
Number	4	5	6	7
1.	38	56	38	36
2.	M	M	M	M
3.	10	25	10	12
4.	G	G	G	G
5.	0	0	0	0
6.	0	+ Extractions	0	0
7.	±	+	+	±
8.	0	0	0	0
9.	0	0	0	+ Freq.Irreg.
10.	F	G	F	G
11.	0	+	0	0
12.	-	67	68	-
13.	-	158.0	146.0	-
14.	±	+	+	+
15.	+	+	+	+
16.	68	72	72	64
17.	99	97.2	97.8	98.6
18.	116-74	148-68	114.70	118-70
19.	S.d.R. - A.R.	A.R. - C.R.	C.R.- I.T.	T.Hyp.-R. Ph.
20.	Encrusted-Eroded	Edent.	Sl.Ging.Good	Many Ext.
21.	+	0	0	±
22.	0	0	0	0
23.	Small Varices	Small varices	0	0 Formerly pair
24.	0	0	0	0
	0	0	0	0
	0	0	0	0
25.	90	100	100	100
	90	100	100	100
26.	Lids	0	0	0
27.	+	+	+	0
28.	8.75	10.5	6.85	5.10
29.	4,990	4,500	5,730	4,180
30.	100	95	102	93
31.	1	0	1	7
32.	59.0	87.0	64.0	58.0
33.	-	2.5	4.5	3.5
34.	31.0	7.5	20.0	32.5
35.	7.0	3.0	8.5	2.0
36.	3.0	-	1.5	2.5
37.	-	-	1.0	1.5
38.	0.05	0.05	0.07	0.06
39.	0.12	0.01	0.07	0.07
40.	0.12	0.08-	0.14-	0.10-
41.	2.49	0.40	0.26	0.40
42.	0.44-	0.10-	0.06-	0.06

6.	0	0	0	0
7.	+	+	+	+
8.	0	0	0	0
9.	0	0	0	+
10.	G	G	F	G
11.	+	+	+	0
12.	66	66	62	66
13.	181.5	143.5	148.0	131.0
14.	++	+	+	+
15.	+	+	+	+
16.	76	92	84	68
17.	98.2	98.4	98.8	98.4
18.	138-84	136-70	130-78	126-
19.	Cong.	S.d.1. Spur C.R.	Dusty spur C.R.	A.R
20.	Ca.	Bad - 1 Pivot	Edent.	Bad - Ca
21.	+	++	0	+
22.	0	0	0	0
23.	0	Varices	Varices	Derm.Scar -
24.	Sl.Hyp.	Sl.Hyp.	0	0
	0	0	0	0
	Sl.Hyp.	0	0	0
25.	90	80	80	80
	90	70	80	80
26.	Eyelids +	0	0	0
27.	+	+	+	+
28.	7.00	7.9	8.65	8.6
29.	4,310	4,050	4,540	3,89
30.	95	90	100	92
31.	1	1	0	15
32.	66.5	64.5	57.0	62.0
33.	1.0	5.0	4.5	1.5
34.	23.0	25.0	28.0	26.0
35.	8.5	3.5	5.5	2.5
36.	1.0	1.5	4.00	5.5
37.	-	0.5	1.00	2.5
38.	0.035	0.03	0.055	-
39.	0.02	0.05	0.03	0.0
40.	0.09	0.05-	0.03-	0.0
41.	0.36	0.07	0.35	0.1
42.	0.09-	0.07	0.08-	0.0

KE 0003706

Table 13

Name	Fritton	Landry		Jepson
Number	12	13	14	15
1.	22	43	41	31
2.	M	M	M	M
3.	4	10	17	3
4.	G	G	G	G
5.	0	$\frac{+}{0}$	0	0
6.	0	0	0	0
7.	+	+	+	+
8.	0	$\frac{+}{0}$	0	0
9.	+ Sinus	0	0	0
10.	G	G	G	G
11.	0	0	0	0
12.	-	65	68	57
13.	-	131.0	168.5	188
14.	+	+	+	++
15.	+	+	+	±
16.	76	52	76	100
17.	99.2	97.4	98.4	99
18.	-	126-72	124-70	142-82
19.	A.R.	Spur C.R.	C.R. -S.d.R.	Spur - C.R.
20.	Fair-Part.Dent.	Edent.	Good	Bad - Almost Edent.
21.	+	0	+	+
22.	0	0	0	0
23.	Injured Toes	0	0	0
24.	0	0	0	+++
	0	0	0	0
	0	0	0	+++
25.	100	100	120	120
	100	100	100	100
26.	+ Eyelids	+ Eyelids	0	+
27.	0	+	+	+
28.	6.65	5.65	5.40	7.60
29.	4,610	4,060	3,700	4,330
30.	92	90	94	96
31.	1	1	1	3
32.	45.0	33.0	44.0	46.5
33.	1.0	4.5	8.5	7.5
34.	46.0	56.5	36.5	37.5
35.	4.0	3.5	5.5	6.0
36.	2.0	2.0	5.0	2.5
37.	2.0	0.5	0.5	-
38.	-	0.04		
39.	0.04	0.02	0.07	0.05
40.	0.05-	0.07	0.09-	0.02+
41.	0.15	0.16	0.23	0.07
42.	0.04-	0.09	0.08+	0.12-

6.	0	0	0	0
7.	+	+	+	+
8.	0	0	0	0
9.	0	0	0	0
0.	G	G	G	G
1.	+	0	0	+
2.	70	65	68	70
3.	156	160.75	209	143.5
4.	+	+	++	+
5.	+	+	+	+
6.	76	108	52	64
7.	98.2	99.0	98.8	98.6
8.	148-98	180-98	110-80	146-96
9.	S.d.R. - C.R.	S.d.R. - C.R.	C.R. Dust	0
0.	Bad-Ca-Almost Edent.	Bad-Almost Edent.	fair	Edent.
1.	+	++	+	0
2.	0	0	0	0
3.	0	Leg Varices Large	0	Psoriasis Arm
4.	++	+++	0	0
	0	0	0	0
	0	+++	0	0
.	80	80	120	80
	80	80	100	90
.	+	++	+	0
.	+	+	+	0
.	5.50	6.75	5.75	5.70
.	4,550	4,600	3,960	3,800
.	94	90	93	82
.	8	0	0	0
.	58.0	47.0	53.0	47.5
.	3.0	1.5	3.5	-
.	26.0	43.0	37.0	41.0
.	6.0	7.5	1.0	9.0
.	7.0	1.0	3.5	-
.	-	-	2.0	1.5
.	-	-	-	-
.	0.07	0.01	0.08	0.10
.	0.06 +	0.04-	0.12	0.04
.	-	1.14	0.93	0.66
.	-	0.12	0.25+	0.09+

KE 0003708

Table 13

Name	Stanhope	Webb	Buhr	Benzein
Number	20	21	22	23
1.	42	44	36	38
2.	S	M	M	M
3.	5	12	2	8
4.	G	G	G	G
5.	0	0	0	++
6.	0	0	+ Dieting	0
7.	+	+	+	Numerous operati Lr.Int.
8.	0	0	0	0
9.	0	0	0	+
10.	G	G	G	G
11.	0	0	0	+
12.	58.5	52.5	63	66.75
13.	219.5	153.5	287.0	229.75
14.	++	+	+	++
15.	+	+	+	+
16.	112	76	104	84
17.	99.2	99.0	99.2	99.4
18.	170-100	126.90	142-100	146-100
19.	A.R. - Crusts	A.R. - C.R.-S.d.r.	S.d.r. - A.R.	Perf.Septum
20.	Fair	Ca. Bad	Ca. Bad	Edent.
21.	+	+	+	0
22.	0	0	0	0
23.	0	0	0	Bad varices
24.	0	++	0	0
	0	0	0	0
	0	0	0	0
25.	90	80	120	100
	80	80	140	100
26.	0	0	0	0
27.	+	+	+	Not Tested
28.	7.80	6.15	7.15	13.95
29.	4,340	4,260	4,450	4,770
30.	98	88	100	100
31.	2	1	14	0
32.	64.5	54.5	54.5	71.0
33.	2.0	6.0	3.0	2.0
34.	27.5	28.5	39.0	21.0
35.	4.5	7.0	3.0	4.5
36.	1.0	2.5	0.5	1.5
37.	0.5	1.5		
38.				
39.	0.11	0.07	0.10	0.11
40.	0.14	0.10+	0.07+	0.08-
41.	3.25	0.51	0.97	0.37
42.	0.63-	0.04-	0.23	0.09+

5.	0	0	+	+ Sl.
6.	0	0	0	0
7.	+	+	+	+
8.	0	0	0	0
9.	0	0	±	0
10.	G	G	G	G
11.	0	±	0	+
12.	66.75	68	67.5	66.25
13.	149.5	173	122.25	133.0
14.	+	+	±	+
15.	+	±	Gen.Sen.Atr.	±
16.	76	88	72	72
17.	99.0	98.6	98.6	99.4
18.	138-80	164-92	114-72	108.68
19.	0	Ulcers	Phar.Varices	S.d.r. Injury
20.	Bad	Poor. Ca. Worn	Poor - Almost Edent.	Fair
21.	+	+	+	+
22.	?±	0	?±	0
23.	Bunion-Rt.Gr.Toe	Small Varices	Bursitis-Left wrist	0
24.	+++	+++	+++	0
	0	0	0	0
	0	++	+++	0
25.	90	120	80	100
	100	100	Very weak ?	130
26.	+	+	+	+
27.	+	+	+	0
28.	9.80	5.05	8.25	6.55
29.	3,890	4,030	4,130	4,770
30.	94	95	75	93
31.	7	10	16	2
32.	60.5	72.0	63.5	62.0
33.	7.5	3.0	2.0	10.0
34.	23.5	24.0	20.5	20.0
35.	7.5	-	7.0	8.0
36.	1.0	0.5	7.0	-
37.	-	0.5	-	-
38.				
39.	0.05	0.09	0.17	0.03
40.	0.08	0.06+	0.15-	0.05-
41.	0.34	0.21	0.39	0.90
42.	0.13-	0.08-	0.07	0.21

KE 0003710

Table 13

Name	E.S.Woods	Dean	E.A.Woods	Campbell
Number	28	29	30	31
1.	34	45	43	35
2.	M	M	M	M
3.	10	10	10	12
4.	Fair	G	G	G
5.	±	0	0	0
6.	0	0	0	±
7.	±	+	+ till lately	±
8.	±	0	0	0
9.	0	0	0	0
10.	G	G	G	G
11.	0	0	0	0
12.	67.0	67.75	67.25	65.25
13.	135.0	141.50	130.25	138.25
14.	±	+	±	+
15.	±	+	±	+
16.	74	72	70	80
17.	98.4	98.4	-	98.4
18.	110.72	120-78	118-64	130-70
19.	C.R. Ulcers	R.I.T. - Crusts	S.d.R.Crusts R.I.T.	Crusts C.R.C.T.
20.	Edent.	Bad - Mostly gone	Very bad Ca. Mostly gone	Little Ca. Very bad
21.	0	+	++	+++
22.	0	0	0	0
23.	Complains of weakness. Not obs.	Amputated phalanges Slight varices	0	0
24.	0	0	+++	++
	0	0	0	0
	0	0	+	0
25.	90	90	90	120
	90	100	90	100
26.	0	0	0	0
27.	0	0	+	+
28.	7.50	8.50	4.75	8.25
29.	4,180	3,990	4,370	5,660
30.	80	95	87	85
31.	0	17	2	18 very coarse
32.	51.0	61.0	41.0	43.5
33.	1.0	4.00	2.0	2.0
34.	38.0	29.00	43.0	47.5
35.	7.5	5.5	7.5	4.5
36	1.5	-	6.0	2.0
37. Myl.	0.5	0.5	0.5	0.5
38.				
39	0.12	0.11	-	-
40.	0.11-	0.13+	0.14+	0.08-
41.	0.77	0.33	0.29	0.53
42.	0.20+	0.05-	0.11+	0.11+

KE 0003711

(9)

Table 13

Name	Miss Blundy	Miss Meyer	Mrs. Aegan	Mrs. Slowinski	Mrs. Clerf
Number	32	33	34	35	36
1.	49	25	24	24	33
2.	S	S	Widow	S	M
3.	19	4	5	4.5	8
4.	G	G	Not rested	G	F Fr.Dreams
5.	0	0	++	0	++
6.	0	0	0	0	0
7.	+	+	+	+	+
8.	0	0	+	0	0
9.	0	0	+	±	±
10.	G	G	F	G	G
11.	0	±	0	0	0
12.					
13.					
14.	++	+	+	+	+
15.	+	+	+	+	+
16.	84	84	72	64	74
17.	98.8	98.0	98.0	98.0	98
18.	150-88	104-78	100-60	116-80	110-68
19.	Perf.Septum Crusts	S.d.r. Rh.	S.d.r. Par.Ob. Cr.-C.T.	0	Sinusitis L.I.T.
20.	Excellent	Good.1 or 2 out.	Fair Pivot	1 dev. Good	Many large fillings
21.	0	±	0	0	±
22.	0	0	0	0	0
23.	Burn Scars - Sl.var.	0	0	0	0
24.	0	+	0	0	0
	0	0	0	0	0
	0	+	0	0	0
25.	70	60	50	90	60
	60	70	60	80	60
26.	±	0	±	0	0
27.					
28.	4.35	9.05	4.00	4.15	5.05
29.	4,420	4,260	3,830	4,560	2,900
30.	90	90	85	85	70
31.	0	0	3	0	0
32.	54.0	50.0	46.0	45.5	38.0
33.	1.0	4.5	4.0	3.0	4.5
34.	27.0	36.0	38.5	46.0	51.5
35.	15.5	7.0	8.0	3.5	4.5
36.	1.5	2.5	3.5	2.0	1.5
37.	1.0				
38.					
39.					
40.	0.06+	0.03+	0.07-	0.07-	0.04+
41.	0.45	0.22	0.55	0.09	1.23
42.	0.10+	0.05+	0.17+	0.10	0.19-

KE 0003712