

Willard Storage Battery Company

MAIN OFFICE AND FACTORY
246-286 E. 131ST ST.

Cleveland, Ohio

May 26, 1944

Robert Kehoe, M. D.
Kettering Laboratory
University of Cincinnati
Cincinnati, Ohio

Dear Doctor Kehoe:

I am writing you for a dual purpose; first, to present the clinical picture accompanying the specimens of George Toy and James Willie Floyd; and secondly, to present some observations of my own on some of these cases of lead absorption or poisoning.

[REDACTED] Urine Specimen sent on 5-12-44. (Blood specimen not obtainable) 53 year old colored man, hired on 8-31-43. This man started work in a lead department at that time and worked there until 4-19-44. On this later date he was absent, and remained absent for three weeks. On his return, he presented a note from an outside M. D., stating that he had lead poisoning and should not return to work in a lead department. The diagnosis was made on a blood smear, said to show elevated stipple count, and on clinical symptoms, chiefly fatigue. During his period of work here he had 7 stipple counts made as follows: 8-31-43 (hire) 0; 10-14-43: 0; 11-11-43: 1; 12-14-43: 0; 1-18-44: 1; 3-1-44: 0; 5-11-44: 0; There were no accompanying clinical symptoms or signs. His weight on hire was 137, and this weight fluctuated from 140 to 132, which was his weight upon returning after his absence, 5-11-44.

History: Essentially negative. Born in this country. No previous illnesses or operations. No history of exposure to lead or other toxic substances. Had worked as a chauffeur for several years before hire here.

Physical examination: Weight - 137, height- 5' 8". B.P. - 112/78. Head, negative, eyes: 20/20. Teeth - Upper plate, partial lower; tonsils, small; heart and lungs, negative. Abdomen showed no abnormality. External rings, large; reflexes, normal; serology, negative.

Present Status: Symptom-free, no complaints except mild fatigue. Working in a department where there is a little or no exposure to lead.

It would greatly facilitate future placing of this man if we had definite quantitative values for urine and/or blood lead



to substantiate our impression that he has had no absorption of any consequence. Should you feel that the information presented, together with your quantitative value would enable you to make a statement about the probability of recent lead ~~contact~~, I should appreciate it. //

[REDACTED]
Blood and urine specimens sent on 5-17-44.

This 24 year old, colored man was hired on 10-11-43. At that time he had a stipple count of 11 and a positive serology (under treatment.)

History: Negative except for the serology and treatment.

Physical examination: Weight - 175, height - 6' 1". Blood pressure 150/80; Head: negative; Eyes: 20/20 bilat.; Mouth: negative; teeth: good; No lead line. Tonsils: not diseased; heart and lungs: negative; abdomen: negative; reflexes: normal.

Course: He was placed in the Forming Department, where he worked until 12-20-43, when he was transferred to the transportation department, because of symptoms associated with a persistently high stipple count. Between 10-11-43 and 12-20-43 he had weekly counts as follows: 11, 6, 9, 14, 27 (12-13-43) and 22 (12-16-43). Associated with the past two counts he had muscle cramps in the left side of his chest, and some anorexia. During this time he was given Cal. Gluconate by mouth in doses of about 40 gr. daily.

At the time of his transfer he showed 19 stipple cells in his smear, he weighed 192 pounds, and had no symptoms except a slight head cold. Between 1-3-44 and 1-7-44, he complained of stomach cramps and sluggish bowels. Alkaline medication apparently helped him.. Smear on 1-7-44 showed 13 stipple cells. On 1-17-44 he complained of drowsiness, headache, metallic taste in mouth, abdominal pain, and at that time he showed a slight lead line about a lower incisor. His stipple count was 12 at the time. There were no significant physical findings except the slight lead line. - lead on incisor

There after the 11th. of March his smears showed decrease to a low of 3 stipple cells, with no symptoms at that time. On March 11th. he had some indefinite abdominal pain, persisting until the 17th., when he had definite abdominal pain, anorexia, metallic taste, with no physical findings. His smear showed 18 cells. On March 27th. he had the same symptoms, plus nausea, and at that time his smear showed 6 cells. Thereafter he had mild abdominal symptoms, especially cramping, anorexia, sluggishness of bowels, until the time of the taking of the specimens on 5-17-44. Repeated smears during this time showed 4 cells. From 5-3-44 he complained of weakness and fatigue in his hands, and general loss of strength. There was no loss of weight, with a fairly constant level maintained at about 193. He showed no lead line after the one occasion early in his course. He takes no medication except mineral oil for his constipation. He is working at present, doing the same work he has been doing since 12-20-43, presumably a non-lead job.

The record of the second patient, [REDACTED] represents a group of records I have studied, showing a puzzling, and possibly a valuable departure from the expected reaction. This group of patients has shown a common tendency toward an increase in symptoms, accompanying a fall in stipple count after removal from known lead exposure. In several such cases there occurred a latent (several weeks) increase in symptoms after removal from lead exposure, and this latent increase in symptoms was unaccompanied by an increase in stipple count. In these cases, lead determinations were diagnostic of lead poisoning. In at least one case there occurred thereafter a secondary rise in stipple count following the recovery from the symptoms.

As you have stated, little is known about the actual partition of lead in the body, and the entire metabolism of lead seems to be remarkably stable, and difficult or impossible to affect by means usually effective in altering metabolism of metals in the body. It has occurred to me that some agency may be influencing the partition of lead in the bodies of the cases I have been observing. I had thought it might be the simple chemical influence of mass action pushing an equation through. Lead entering the body might conceivably keep the stream of lead going into the blood stream and thence into whatever storage form or site available. The original insult to the bone marrow produces the flood of immature cells, but as lead continues in contact with the marrow the effect, in producing abnormal numbers of immature cells in the blood, becomes less, and the stipple count returns to normal. Thereafter, there is maintained a steady flow of lead into these storage places, but without any stimulation of the marrow, now conditioned to the presence of lead. However, should the flow of lead be stopped, there would appear to be a tendency for the reaction to return to the left, i.e., lead to return to the circulating form. However, in the absence of good excretory facilities, this circulating level reaches symptom or toxic state. Some hypothesis, eh?

Anyway, there seems to be some influence at work and it would be interesting to follow it through. I would be much interested in any suggestions you might throw at me, either in the way of explaining this situation or toward the direction of further study.

Very truly yours,

WILLARD STORAGE BATTERY COMPANY

James J. Badal, M.D.

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