John T. Lewis and Bros. Co.,
2545 Aramingo Avenue, Philadelphia,
Pennsylvania

May 21, 1947

Dr. Kenneth A. Koerber,
Plant Physician,
John T. Lewis and Brothers Company,
2545 Aramingo Avenue,
Philadelphia, Pennsylvania.

Dear Dr. Koerber:

I send you herewith my best interpretation of the analytical results obtained by us in the case of Emery Robinson, as well as a general opinion on the case. These are in triplicate as you requested.

In order to leave argument out of the opinion, I call your attention herein to what may be regarded as incorrect statements or interpretations in your last letter. Our results as reported on January 8, 1947 showed a definitely abnormal lead concentration in the blood, as so indicated in the report forwarded with my letter of January 13, 1947. The urinary lead concentration was at the upper level of normal values, but was regarded dubiously because of the size of the sample.

Our second set of analyses, reported on March 1, 1947 and transmitted with my letter of March 4, 1947, showed somewhat higher but not contradictory values in the blood and an even lower value for the urine.

In neither instance was the urinary lead concentration beyond the limits of normal variation for samples of small volume, nor were they in any way remarkable in view of the volume of sample submitted. The status of this man, however, with respect to lead exposure, was shown not by the analyses of the urine but by those on the blood. The blood results confirm each other quite adequately, and demonstrate beyond any reasonable doubt that Robinson's lead exposure during the months immediately preceding the taking of these samples was significant.

The Superintendent's statement does not show that he had no lead exposure. On the contrary, it specifically refers to work which could have, and clearly did actually result in lead exposure and absorption. Moreover, the lead concentration in the blood in both instances is such that symptomatology from lead absorption could have existed.

The result of this situation is that the diagnosis must be made on purely clinical grounds. I agree with your diagnosis and that of

Dr. Hollander, that this man is suffering from gout and hypertension. I see no reason for supposing that he has lead poisoning in addition. His present symptomatology and the present hematological findings of anemia without stippling or abnormal numbers of other young forms, fail to indicate the existence of lead poisoning. Therefore, the evidence of lead exposure and absorption is, at present, incidental, from the diagnostic viewpoint. As to the relationship of lead absorption to gout, the evidence is of poor quality, and unsatisfactory. The British clinicians, especially, considered lead absorption to be a precipitating factor in gout, but their evidence lay purely in the coincidental occurrence of the two phenomena of gouty diathesis or the onset of gout, with lead exposure in the lead trades. Gout was and is common in England and the relationship, in my opinion, was fortuitous.

I trust that this clarifies my opinion in this case. What your Philadelphia colleagues fail to recognize is that they and no one else can make the diagnosis of lead poisoning by analytical or any other than clinical means. Lead exposure and lead absorption are not synonymous with lead intoxication. They confuse the issue by not sticking to clinical considerations.

Cordially yours,

Robert A. Kehoe, M. D.

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Interpretation
of
Analytical Results and Clinical Syndrome

Case of [redacted]

This man was exposed to appreciable quantities of lead in the course of his occupation in the period immediately prior to December 31, 1947 and thereafter. The exposure was not highly hazardous, but it led to his absorption of potentially dangerous quantities of lead, as shown by the lead concentrations in the blood on two occasions: - namely 0.085 mg. Pb per 100 grams of whole blood as reported by us on January 8, 1947, and 0.13 mg. Pb per 100 grams of whole blood as reported by us on March 1, 1947. In our opinion, these results, taken with the relatively low (normal range) lead concentration in the urine on both occasions, strongly suggests that Robinson's lead exposure was of a low but significant order of magnitude over a long period of time (years). The occupational history indicates that this was the case.

A diagnosis of lead poisoning is not justified by the symptomatology and the clinical facts, including the hematological findings. A diagnosis of gout is tenable and reasonable, thereby making it wholly unnecessary to explain any of the symptomatology on the highly dubious basis of atypical plumbism. An etiologic relationship between lead absorption and the onset of gout is, I should say, purely speculative.

Robert A. Kehoe, M. D.

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