August 12, 1960

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Avon 1-2568

Dr. H. B. McWhorter
Second National Bank Building
Ashland, Kentucky

Dear Doctor McWhorter:

In response to your letter about your patient, I find it advisable first to comment on the diagnostic problem as it relates to lead. To begin at the beginning of this problem, this man's exposure might conceivably be sufficient to cause lead intoxication for two reasons; (1) he may be swallowing appreciable quantities of lead because of contaminated hands which contaminate his food, beverages, tobacco and the like; and (2) he may be inhaling droplets of liquid containing white lead (if such droplets are being dispersed mechanically in the air). However, he is not absorbing lead through his skin, for this does not occur under the conditions which you describe.

Second, the extent of his absorption can be determined with certainty by a precise analytical determination of the lead in his blood; to a less satisfactory extent this can be done by analysis of the urine, but only if samples of urine are collected with extreme care to avoid contamination. I have no idea how or where the analyses to which you refer were made, but unless you have incontrovertible evidence of the absolute reliability of the results obtained by the laboratory in question, I advise you not to be unduly influenced by them. I suspect their invalidity because of your statement (perhaps derived from the laboratory) that the normal concentration in the blood ranges from 0 to 100 micrograms per 100 grams. This is not true, for there is no such thing as a negative result, if the proper method of analysis is carried out properly, and, more importantly, normal results never exceed 60 micrograms and rarely exceed 50 micrograms. Concentrations up to 75 micrograms are not associated with the occurrence of lead intoxication, while anything above 80 is indicative of a degree of lead absorption which is, in itself, dangerous, and is often associated with lead intoxication.

The values obtained in the urine - whether the sample is small or large - are much less certainly significant, because of the much wider range of variability of the urinary excretory rate. The results obtained on a 24-hour sample of urine will not be as definitive as they will be on a sample of blood.

Because of what I have said above, I advise you to send us duplicate samples of blood and a spot sample of urine in containers which we shall supply. I do not suggest by this advice that there is no other reliable laboratory for this purpose. I know, however, that such laboratories are very few, and I know of none in your area. There may be one about which I do not know. Nevertheless, this is one of the most troublesome bits of analytical work that is required in relation to diagnosis, and your diagnostic problem can probably be solved by correct analytical data and by their proper interpretation. Since we are responsible for the initiation of this diagnostic approach, we do all we can to assist our colleagues.

If you want this done, let me know, and I'll send you the necessary containers. The work will be done at its cost to us - for the two samples of blood, analysed by two separate methods, we charge as though they were one, and we make the same charge for the urine, the total being $12.50 for each, or $25.00. When the work is done for a patient who cannot afford the charge, or for whom no one else is in position to pay, no charge is made, or only such charge as the physician in the case directs.

Now, as to the possible influence of lead intoxication on the endocrine glands, in specific relation to myxedema, we have not the slightest basis, so far as is known, for any such suspicion. There has been a tendency toward the belief that the clinical manifestations and the sequelae of lead intoxication are many and varied. My experience in thirty years of investigation of this disease and its physiologic and pathologic backgrounds is to the contrary. The application of fairly rigid criteria to the diagnosis of the disease has resulted in simplifying and clarifying the clinical types. There is a constancy of pattern - or actually, patterns, since there are three fairly definite types - which renders very dubious indeed the inclusion of bizarre effects within the clinical syndrome.

I suggest that we first determine whether or not your patient has absorbed enough lead to be dangerous. If he has, your diagnostic problem is a bit complex, perhaps, but if he has not, it is a much simpler situation, although not necessarily one of better outlook.

Cordially yours,

Robert A. Kehoe, M.D.

RAK ef

H. B. McWHORTER, M. D.
SECOND NATIONAL BANK
ASHLAND, KENTUCKY

CARDIOVASCULAR DISEASE
INTERNAL MEDICINE

BY APPOINTMENT
EAST 5-2665

August 9, 1960

Kettering Laboratory
Cincinnati, Ohio

Dear Sirs:

I would appreciate your assistance in a very interesting problem. I have a 43 year old man who began working 1 year ago with a mixture of liquid white lead which was used as a jet to cool the bit of a steel drill-press. He states that his hands would be wet with this solution and that there frequently would be a fine mist about his machine. After a 6 months period he first noted edema of the hands, feet, and eyes, associated with progressive fatigue. He also has noted slight constipation, thickening of his speech, and drowsiness. He has had no neurological symptoms whatsoever. He was laid off work May 8, 1960, however his symptoms have remained stable or perhaps slightly progressed.

I first saw this man several weeks ago at which time he presented a classical clinical picture of myxedema with a P.B.I. of 2.2 mcg % and serum cholesterol of 342 mg %, as well as a mild normochromic anemia and lymphocytosis. In addition he had a suggestive lead-line at the gum margin of his remaining 5-6 incisors which were quite carious with pyorrhea. There was a moderate basophilic stippling of his red blood cells as well. A blood lead was 68.5 mcgs % by the Dithizone method, the normal being 0-100 mcgs %. A quantitative 24 hour urine lead is being obtained at the present time.

Unquestionably this man has had exposure to lead but has no evidence of neurological, hepatic, or renal disease. His entire clinical picture and symptom complex appear to be best explained by myxedema. I am wondering whether this is primary myxedema or possibly might be secondary to toxic effects of lead. I am unable to find any information or reported cases of this in the literature or standard references. I know of your interest in Toxicology and would appreciate any information you have on possible toxic effects of lead on the thyroid or pituitary glands.

Sincerely yours,

H. B. McWhorter, M. D.

HBMcW/rjdp

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