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FORTY-ONE EASTERN PARKWAY
(COPLEY PLAZA)
BROOKLYN, NEW YORK

April 23rd, 1934.

Dr.Robert A. Kehoe,
College of Medicine,
University of Cincinnati,
Cincinnati, Ohio.

My dear Dr. Kehoe:

I want to express my deepest appreciation of your letter of April 19th and also of the series of articles which I received today. I have become familiar with your work on plumbism and I am very much pleased to have all your articles in my library.

Your experience with plumbism has been much greater than mine and I know that when you make the statement that "patients will de-lead themselves down to the normal level when left alone", it is made after considerable study and experimentation. I should like you to know just how it happened that I became interested in the de-leading treatment.

Some three or four years ago several patients with chronic plumbism had been receiving compensation under the Workmen's Compensation Act because of protracted manifestations associated with their industrial accident. The physical examination in most of these individuals was essentially negative. There was a slight secondary anemia in an occasional patient and furthermore, the patients were able to furnish proof that traces of lead were found in the urine. At that time it was not generally known that small traces of lead are present in individuals not exposed to lead (as conclusively shown by your recent studies in Mexico) and so litigation and compensation continued in these industrial accident cases. The insurance companies were confronted with the problem of proving that the worker was no longer suffering from lead poisoning. You fully realize and in fact you have stated that the clinical symptoms are the most important aid in the diagnosis. These individuals had continuous symptoms and thus the burden of proof was thrown upon the company to either prove or disprove the question as to whether or not these people had lead poisoning.

When I started the de-leading treatment in 1931, I was familiar with your writings of 1926 and 1929 in which you spoke of the excretion of lead by normal persons. In these cases of chronic plumbism,

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I believed that no danger could follow the de-leading treatment as advocated by Aub and his co-workers. It seemed reasonable to assume that if we could prove by the de-leading treatment that abnormal amounts of lead were not excreted in the urine such individuals did not have abnormal amounts stored in the skeletal system and many of their symptoms were alleged and not real. All of these patients were adults and at no time was the de-leading treatment instituted in the acute phase.

We treated approximately fourteen individuals to date, some of whom had absorbed a great deal of lead and under treatment excreted an abnormal amount of lead. To date, I have not seen any untoward symptoms nor have I at any time observed an acute toxic episode during the course of treatment. I agree with you that in elevating the lead concentration in the blood there may be a deposition of the metal in the central nervous system. The absence of any symptoms in the central nervous system does not preclude the fact that lead may be deposited there in the course of the treatment. However, I have not observed any clinical evidences suggestive of either peripheral or central nervous system involvement.

During the past year I have been using the high phosphorus, low calcium diet in the de-leading treatment. I have spoken with Dr. Shelling of Johns Hopkins (who recommended the high phosphorus diet) and I believe that his experimental work is of significance. Since his publication of his experimental work on rats he has had the opportunity of treating patients with the high phosphorus low calcium diet and his results have been satisfactory.

You appreciate the fact that the problem which confronted me when these industrial cases were sent to me was one which required that I furnish proof of either the absence or the presence of lead poisoning. If under de-leading treatment large amounts of lead were found in the urine (and all these cases had continuous subjective symptoms), it was reasonable to state that the protracted manifestations could be due to lead excretion. If, on the other hand, continued symptoms were not supported by the laboratory findings (i.e. abnormal amounts of lead in the urine, anemia, stippling and abnormal amounts of lead in the stool), it was reasonable to assume that lead was not a factor in the production of symptoms. And so it was that I became interested in the de-leading treatment.

Some criteria must be obtained and established in order to honestly arrive at some conclusion regarding the compensability in relation to this problem. There are of course other phases of this entire work which require further study and investigation. I should be glad to co-operate with you in any work regarding the treatment of these workmen which you think will help in establishing criteria to determine the termination of disability. This you realize is a major problem in which we are all concerned.

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At the Jewish Hospital of Brooklyn where I have done this work, a spectrograph is now being installed. We are going to study the content of various tissues obtained at autopsy and examine the excreta of many normal individuals. We hope to continue the investigations and add to the many interesting and important contributions you have made on this subject.

I am looking forward to seeing you in Cleveland.

With kindest regards, I am

Cordially yours,

Irving Gray

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