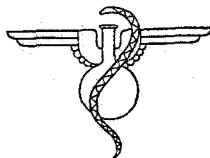


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AT LABORATORIES RESEARCH DIVISION
NORTH CHICAGO
ILLINOIS



ROBERT D. COCHILL
Director of Research
MARLIN T. LEFFLER
Associate Director of Research
RICHARD K. RICHARDS
Associate Director of Research

March 14, 1952

Robert A. Kehoe, M.D.
The Kettering Laboratory
University of Cincinnati
College of Medicine
Eden Avenue
Cincinnati 19, Ohio

Dear Dr. Kehoe:

Please let me thank you for your kind letter of March 10th in reply to my earlier inquiry regarding the treatment of lead poisoning.

I fully understand that you are very busy with the various important projects which you have under investigation, and there is no need to apologize for the delay in your answer.

I wonder if I still may impose upon you one time in asking for a clarification of your statements regarding the failure of the administration of various agents, like ammonium chloride, potassium iodide, potassium phosphate, citrate, etc. to produce an increased lead excretion. I have reviewed several recent articles which give tabulated data showing an increase of the lead excretion in the urine under such management.

Do you feel that the amount excreted in excess to that prior to this treatment is insignificant in relation to the lead still present in the body? This would be about the explanation which I would infer from your letter, but I would prefer your personal comment if you have time to do so.

Thank you again for your very helpful advice on this matter, and I will be grateful indeed to hear from you.

Sincerely yours,

R. K. Richards, M.D.

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March 10, 1952

Dr. R. K. Richards
Abbott Laboratories Research Division
North Chicago, Illinois

Dear Doctor Richards:

I am slow in responding to your letter of February 4 because of a rather heavy burden of matters to be dealt with lately.

I have read over my letter of February 5, 1949 relative to the therapy of lead poisoning and I find little in it to change or to add. I'd like to correct the impression that seems to have been made on your mind that I think deleading inadvisable. That is not the point of my comments. I did fear the procedure recommended by Aub and his associates when I had some faith in the possibility of "mobilizing" lead, and I still have some reservations about the use of BAL in lead poisoning, especially since the reports of Vigliani et al: British J. Ind. Med. 8:218 (1951). The latter has reported the recurrence of acute symptomatology in certain patients treated with BAL, and although this effect does not agree with our observations it is not to be taken lightly. However, our evidence with respect to the effects of calcium deprivation and the administration of NH_4Cl , KI and Na Citrate, as well as the effects of inducing changes in the phosphorous metabolism, have demonstrated clearly that nothing of significance occurs to the lead metabolism under these conditions. These data have not been published partly for the lack of time on my part to get them together and partly for the reason that the clinical observations on these cases were made by member of our clinical staff whom I've thought should get together and write the articles. Henry Ryder has been up to his ears in other experimental work with another group, while the two other clinicians involved have been preoccupied with other matters. It seems that I shall have to do what needs to be done toward the publication, but I've simply been unable to get to it because of the constant burden of keeping current work going without an excessive lag. Essentially what has been done in the study of this problem has been accomplished by two methods. In those I have studied, the intake of lead in food and drink, and the output of lead in the feces and urine have been followed with great care during an initial period, during a period of intense therapy, and then during a subsequent period. During each of these periods the concentration of lead in the blood has been determined repeatedly. All of the results have been within the expected and demonstrated limits of physiological variation, uninfluenced by specific therapy, except in the case of BAL therapy. Later, Henry

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Dr. R. K. Richards - (2) - March 10, 1952

Ryder, Karl Kitzmiller and John Lauer varied the method with my approval, by following only the blood and urine before, during and after therapy. Ryder also studied the partition of lead in the blood as between cells and plasma.

The only other problem not touched upon in my letter is that of tetraethyl lead poisoning, which is a very different matter, involving acute cerebral intoxication without exception. As this disease is unique, so is its therapy, which at present is a matter of controlling the acute manifestations of cerebral excitation and chemical irritation so as to prevent death from exhaustion. I regret that I cannot supply you with the data in support of my statements, but it would take too much work at this time.

Cordially yours,

Robert A. Kehoe, M. D.

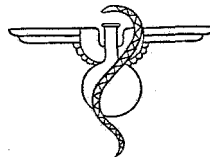
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P. S. Perhaps in the foregoing discussion I have not made my primary point clear, which is - that the "mobilization" of lead is a spontaneous physiologic phenomenon which is uninfluenced by so called "de leading" procedures. "De-leading," in other words, does not occur, except at its own slow rate. Therefore, it need not be feared, but by the same token it accomplishes nothing. The matter of BAL therapy is something else, in that it seems to have little influence on lead in the bones.

R.A.K.

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PARKE-DAVIS LABORATORIES RESEARCH DIVISION



NORTH CHICAGO
ILLINOIS

ROBERT D. COGHILL
Director of Research
HARLIN T. LEFFLER
Associate Director of Research
RICHARD K. RICHARDS
Associate Director of Research

February 4, 1952

Dr. Robert A. Kehoe
Kettering Laboratory of Applied Physiology
Eden Avenue
Cincinnati, Ohio

Dear Dr. Kehoe:

About three years ago you very kindly sent me reprints on some of your work on lead intoxication and also made some additional comments in response to my request.

I am now in the process of revising a chapter which I have written for Kaiser's textbook entitled "Therapeutics in Internal Medicine". A part of this chapter concerns acute lead poisoning.

I wonder if any importance additions have been made to the therapy of acute lead intoxication. Last time you expressed the opinion that any attempt to delead a person is probably rather inadvisable. If you would be good enough to comment again on your attitude in this respect and give me recent data on the treatment of acute lead poisoning, it would enormously strengthen the value of this chapter.

I can assure you of my gratitude for your very helpful cooperation.

Respectfully yours,

R. K. Richards, M.D.

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