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March 24, 1934

SUBJECT:

Dr. Robert A. Kehoe,
Laboratory of Applied Physiology,
University of Cincinnati,
Cincinnati, Ohio.

Dear Dr. Kehoe:

I have read with considerable interest the reprint you recently sent me on "The Behavior of Lead in the Animal Organism. III. Colloidal Lead Compounds", taken from the Journal of Laboratory and Clinical Medicine for November, 1933.

I am interested in what you have found in this and others of your studies to the effect that lead is gradually eliminated from the body from which I judge that such a condition as Delayed or Latent Lead Poisoning is practically non-existent. If you will remember, I discussed this in a paragraph in connection with our former APHA Committee's Report on Lead Poisoning and contained in the Year Book of 1930-1931, page 171. Just prior to that time I had had occasion to look up most of the original literature in connection with the cases which Dr. Alice Hamilton has cited as bearing out the evidence of the possibility of an acute attack of lead poisoning's occurring some years subsequent to the last definite exposure to lead. The evidence was susceptible to other interpretations in almost every instance so that I am still convinced that the apex point of the evidence of lead poisoning occurs at the time of or within a few weeks at most of the last definite exposure. Thus, if any symptoms are to appear at all, they must appear at that time or before and not later. Through certain changes of the metabolism in the body, it seems probable that such symptoms might subside and possibly appear later, but I doubt whether such could be the case after an interval of a year or so. It seems to me your studies throw some light upon this disputed point. At least with nearly 3,000 cases of lead poisoning reported in Ohio in the last twenty-one years, there is no record of a clear case of Delayed or Latent Lead Poisoning as defined in the 1930 Report of our APHA Committee, pages 6 and 23. We have had some cases of Super-Added Lead Poisoning and have had a considerable number of cases of ultimate disability, oftentimes with death, following years after in cases of reported (and compensated) lead poisoning and due to cardiovascular renal disease - usually gradual kidney blockage and hypertension. In this connection I am wondering if you have seen the thesis by L. J. Jarvis Nye, M.B., Ch.M., Brisbane, Australia, entitled "Chronic Nephritis and Lead Poisoning", Angus & Robertson,× 1933, 145 pages? This has to do especially with lead poisoning among children

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in Queensland and has aimed to complete studies begun by federal investigators two years previously.

It is some surprise too to note that tertiary and secondary phosphates of lead are chemically and physiologically inert and that some colloidal form is more probable as the toxic form. This agrees with Sir Thomas Oliver and older investigators who spoke of lead albuminate or lead globulin-forms as the most probable in the blood stream.

In your last paragraph, first sentence, would the bones be considered living tissues? I presume, of course, they are, and that therefore lead compounds are not stored in them in any inert form?

This is fine work you are doing and I hope you may be permitted to continue until most of the controversial questions regarding lead poisoning have at least been given a good overhauling if not solved.

I have noted recently ~~that~~ a mention or two of rapid methods of detecting lead in feces and urine. Do you take any stock in these? As you know, we are discouraging the usual laboratory reports on such examinations at the present time as more misleading than virtuous, - still we should like to see some ready methods perfected which could be accepted with confidence in physicians' reports.

Yours sincerely,

Emery R. Hayhurst

Emery R. Hayhurst, M.D.,
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