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April 1, 1930.

Dr. Robert A. Kehoe,
Associate Professor of Physiology,
University of Cincinnati,
Cincinnati, Ohio.

Dear Dr. Kehoe:

I have read with no little interest your reprint "On the Diagnosis and Treatment of Lead Poisoning" from the Cincinnati Journal of Medicine, March, 1930, which you were kind enough to send me under date of March 17th. I have also taken occasion to abstract the material at some length for the American Journal of Public Health.

This is the best thing on lead poisoning I have come across for some time. I presume you sent it to me for any observations or comments which might occur to me. The data submitted in this reprint, while brief, appear to bear out your conclusions well and I presume your more extended article, when you have it ready for publication, will answer most of the queries which might arise.

One of the first things which I note is that the Committee on Lead Poisoning (A.P.H.A.) may have to reconsider its definition of Lead Intoxication to meet more exactly your conclusions as to actual evidence of intoxication (mentioned in the Reprint on p.6, 2nd column, center paragraph). I have no doubt that you are right in your conclusions that progressive changes in the blood findings in the nature of a diminution of hemoglobin mean actual intoxication with or without the presence of associated subjective symptoms. (I am excepting those instances, which you call attention to, in which blood changes are due to other causes). There would, of course, be no question where subjective symptoms are present. Whether it is practical from a compensation standpoint to recognize lead intoxication without subjective symptoms is quite a question. As a guide to the industrial physician who is watching Lead Absorption there can be no question of the value of the progressive changes which you emphasized in blood findings, and perhaps other physical findings, as a guide to action. Under the Committee's definition of Lead Absorption and its explanation, I believe all workers so grouped should be under medical supervision, hence, should have their medical supervision fees paid by compensation agencies, - compensation itself being limited to disablement only, for the present.

I mention this because there is a tendency abroad not only to pay medical fees when there is no lost time from work but to pay an agreed upon pro rata for early stages of occupational diseases even when there is no immediate disablement. So far as I know, we have not come to this conception anywhere in this country as yet. In certain afflictions like silicosis in which an X-ray diagnosis of a first stage means a certain amount of irreparable damage and an increasing likelihood of pneumonia, or tuberculosis, there may be undoubted justification for such action. As there seems to be

no evidence that an early stage of lead intoxication may result in irreparable damage, however, the conditions are not the same. Therefore, my suggestion is to let the Committee's definition for Lead Intoxication stand as it is, "When absorbed lead causes subjective symptoms with objective findings". This will greatly simplify compensation procedures despite the fact that the definition may not be critically correct.

I am very much interested in what you say about the probable negative effects of lead upon the blood vessels. I have recently abstracted an article by Legge who analyzes some 10,000 cases of lead poisoning which occurred during his experience as Chief Medical Officer of Factories, England and Wales (1901-1927), who comes to the opposite conclusions, if I have interpreted him correctly. H.G. Wells, Chicago, once told me that he thought the arterio-sclerosis of alcoholism was more than likely due to lead poisoning acquired from containers. Practically all the older literature supports the arterio-sclerotic point of view. However, some years ago, Sir Thomas Oliver and, I think also Goadby, suggested that the sclerotic kidney of painters was due rather to turpentine than to lead. I understand that Aub would agree with your point of view. - I doubt whether the question can be settled by animal experimentation, using the ordinary laboratory animals which, I understand, are not so apt to show arterio-sclerosis as man. Perhaps the monkey might prove satisfactory for the purpose. At all odds, there is considerable controversy here, and the subject would be well worth a careful research.

In the Reprint, page 7, 2nd column, end of the 2nd paragraph, I am wondering if the phrase "detectable quantity of blood" should not read "detectable quantity of lead"? (Italics are mine).

I recently have had occasion to look up the subject of latent lead poisoning, reported practically altogether in older literature and have come to the conclusion that there is "no such animal". Practically all of the cases reported had a history of definite lead intoxication during the time of exposure or symptoms of lead intoxication occurred within a few weeks, and, at most, within a few months, following lead exposure. Thus, these cases should more properly be called relapsing, or, at the most, delayed lead poisoning. Even here, I have grave doubts whether subsequent exposures to lead were carefully ruled out. I am supported in this view by the fact that no physicians of the State of Ohio have ever reported a long deferred case of lead poisoning to the State departments so far as I have been able to find out and I have rather carefully watched this feature for the past 17 years in connection with some 2,200 cases of lead poisoning reported to the State Department of Health since May, 1913.

While you have not discussed symptomatology in this paper it would be interesting to know the symptoms associated with the case "J.G.", referred to in Table IX, as completing the picture.

Your finding (page 8, column 2, 1st paragraph) that there is no evidence that lead is fixed in any tissue of the body with the exception of the central nervous system, apparently refutes Aub's findings. Also, it should be made clear in this connection that the distribution of lead as shown in Table IX and perhaps in some of the other tables presented, does not represent fixed lead.

This discussion on page 9 is not very clear to me, particularly the discussion in the 2nd column. In connection with Table XII, is it meant that lead in its normal distribution in the tissues is found in such large relatively quantities in the central nervous system? I was under the impression that the central nervous system was rather spared in early lead intoxication, but I can see that when lead once reached it, it may become fixed, as you state. In this connection have you any evidence as to how it is fixed and whether there is any difinite pathology present? Can the lead be identified in tissue combination? Oliver discusses such pathology, but not anything characteristic.

I am certainly impressed with the rational of your plan of treatment as it appears logical from the biological, i.e., physiological, point of view, also, it agrees with general medical experience in treating lead poisoning in the past.

The above represents my immediate reflections on the several new points or controversial points which you have developed in your very praiseworthy efforts. I know you are very busy and am not expecting you to take the trouble of replying post haste, but should be glad to have your views sometimes when we meet again. I shall also be very pleased to be remembered with reprints of your published articles whenever you have them for distribution.

Yours very sincerely,

Emery R. Hayhurst

Emery R. Hayhurst, M.D.,
Professor of Hygiene.

72-29