

February 7, 1944

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Mr. F. E. Wormser,
Lead Industries Association,
420 Lexington Avenue,
New York City 17.

Dear Mr. Wormser:

I have had time only recently to consider your letter of January 19, and the article by Byers and Boyd in the American Journal of Diseases of Children. Having been out of the country for several months, I could not get around to answering it sooner.

I fear that you will be disappointed by my answer, for I am disposed to agree with the conclusions arrived at by the authors, and to believe that their evidence, if not entirely adequate, is worthy of very serious consideration. Perhaps my own experience prejudices me in favor of the acceptance of their findings, for I have seen cases of serious mental retardation in children that have recovered from lead poisoning of the encephalopathic type, and among my records is one case of permanent feeble mindedness which I attribute to a well defined episode of lead encephalopathy in an infant. The child is now fourteen or fifteen years of age.

The article must be considered on two bases, - (1) evidence of the occurrence of lead poisoning, and (2) evidence of mental impairment. For my part I am willing, - perhaps too willing - to accept the conclusions of the authors as to the second point. It must be pointed out that the contribution of Elizabeth E. Lord, Ph.D. was made on this aspect of the problem, and not on the question of lead exposure or lead poisoning. I should not take issue with the propriety or competence of her work in this field, although I am frank to say that I have no knowledge of her abilities. It is true that the authors do some rather amateurish theorizing on the subject of the behavior of lead in the human organism in the first part of their article, and that they discuss the criteria of the diagnosis of lead poisoning in a somewhat less than well-informed manner. On the other hand, it appears that they did not make the diagnoses in these cases but rather accepted the diagnoses made as a matter of record in the Childrens Hospital, and carried out their follow-up studies on cases that would ordinarily be accepted as lead poisoning in children. I agree that the diagnoses may have

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been in error in some instances, perhaps even in many, for I know how difficult this problem is in the case of children. In the final analysis it is frequently impossible to be sure on ordinary clinical grounds, and often a series of analyses of the blood and urine are required to prove or disprove the significance of the lead exposure. On the other hand, the criteria used here, as indicated in the Summary of the Data, are orthodox.

You quarrel with the statement about chewing paint and say that the manufacturers don't use it on cribs and toys. That may well be true. My experience leads me to accept it as such. However, the householder repaints these articles, and often with lead-containing paints. Please note that the article makes point of the fact that the children chewed paint "off cribs, window sills or furniture," and also refers to the statement of parents that they had repainted cribs. I'm afraid it will do you no good to try to combat the significance of the history of chewing articles in relation to the problem of lead poisoning in children. The most significant feature of the history of exposure in an overwhelming proportion of the cases of lead poisoning in children is just that fact. "Pica" is at the bottom of most of these cases, and unfortunately the environment of small children is not sufficiently free of lead for their safety. Have you seen the data on lead poisoning in children in Queensland? These cases were largely due to chewing the paint off the railings of the porches on which the children played.

You also object to the significance of the X-ray evidence of lead absorption. I'm afraid you are not on good ground. The work of Park and Vogt on this point is much too extensive and good to be dismissed so easily. This sign does not exist in adults, for there is no line to be photographed in the case of the adult, unless perhaps he absorbs much lead while a broken bone is mending. However, in the case of the child it is a very useful presumptive sign. Although the dense line at the epiphyseal edge of the rapidly growing and calcifying long bone of the child is not specific for lead, (this should always be checked by blood or urinary lead analysis) it is associated with the absorption of abnormal quantities of lead in many instances, and therefore it may not be ignored. Fairhall's work (he is not a physician, either) was mainly on the bones of dogs, which may or may not have been young and growing. He does not mention this point, and I am not sure he was even aware of its significance. However, he did not say that no line was visible in the bones of children exposed to lead. He only said that there is considerable doubt that any line seen in human or other bones is due to the shadow cast by lead itself at that point. I am not prepared to argue the matter of the lead concentration in the region of most rapid growth in the bones of children, for I have no analytical data on that aspect of the problem. I am prepared to say that the occurrence of a dense

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narrow band at the epiphyseal line in the long bones of a child is a warning that had better be carefully investigated, for although it is by no means certain, the likelihood is that this child has been swallowing or inhaling considerable quantities of lead in the very recent past. I advise you to examine the articles of Park and of Vogt with considerable care before you make too broad an interpretation and application of Lawrence Fairhall's observations.

The statements in the case records that "the urine was normal," do not refer to normal lead concentrations, but rather to the lack of ordinary abnormalities such as albuminuria, hematuria, and the like. You must remember that the use of the urinary and blood lead concentrations as evidence of the extent of the recent lead absorption of an individual was not popular in Boston even after these procedures had come to be accepted as diagnostic measures elsewhere. It is not surprising therefore, that lead analyses on the blood or urine were not carried out. In this connection it is enlightening to note (page 475) that the authors refer to the recently published method of analysis of Fairhall and Keenan as offering some hope that the lead in urine may eventually be found to have some clinical significance.

You must not take too seriously the reported absence of stippling and lead lines in many of these cases. The blood findings on children are somewhat more erratic than they are in adults, and then the large proportion of doctors do not know how to examine a blood smear to find out if stippling is present. I speak here from long experience with the results of this examination in hospitals. We never take them seriously unless we know who made the examination. As for lead lines, they are rarely seen even in the most severely poisoned children, for the reason that children seldom have chronic gingivitis.

Finally, from the purely clinical viewpoint, the symptomatology of most of these cases was such as to justify the strong suspicion that lead poisoning existed. Any attempt to discredit the diagnoses in most instances would not be worth the effort, since only detailed evidence could be used to do this. No such evidence is available. I quite agree that the reasoning in some instances was "confusing."

From all this you may gather that I think that the only thing that can be done about this paper and this concept, is to investigate the problem further, so as to settle the facts. What has been said is suggestive, and I think there is more than a grain of truth in it. The situation may not be as black, however, as it has been painted.

I apologize for the length of this letter, but I could not explain

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my point of view without considerable discussion.

I'm glad you liked the report of the A.P.H.A. Committee. I, also, think it is good.

Cordially yours,

Robert A. Kehoe, M. D.

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