RE 0016749

May 11, 1943

Mr. E. V. Parker,
Deputy Commissioner,
U. S. Employees' Compensation Commission,
1261 Calvert Building,
Baltimore, Maryland.

Dear Mr. Parker:

I am very glad to reply as well as I can to the additional questions in your letter of May 4.

First, with reference to the significance of the stippling initially reported by Dr. Nofsinger and commented upon additionally in his subsequent letter to you, I scarcely think there is adequate evidence to enable us to interpret the precise significance of the stippling in this case. I would not wish to say dogmatically either that the stippling as reported by Dr. Nofsinger was the result of the hemorrhage or that it was not. Neither would I be willing to say that it was the result of lead absorption or that it was not. The presence or the quantity of stippling is not sufficiently predictable to justify a conclusion on this score, in my opinion. Therefore, I am left in the same position as I was before. I am not sure that the claimant had enough exposure to lead to be responsible for an attack of lead intoxication. Neither am I sure that he did not have significant exposure. Consequently, I should be disposed to assume that he did have, and therefore give him benefit of the doubt. In other words, I should not like to combat the judgment of a professional colleague in the absence of adequate evidence. This is not only a matter of professional courtesy, but more importantly a matter in which there is a reasonable doubt.

With reference to your second question, my conclusion that disability from lead absorption would not have been prolonged, is based on the fact that the claimant did not have any of the more serious manifestations of lead intoxication, namely, paralysis or cerebral lead intoxication. It is our general experience that lead intoxication, which is limited to gastro-enteric symptoms and hematologic changes, subsides regularly and fairly rapidly when further exposure to lead is brought to an end. The disability may occasionally last longer than eight weeks, but it is only rare that it does, and usually there are other factors active in the rare cases to prolong the disability. In this instance, I cannot find any good

reasons for believing that this man's hemorrhage was due to or was influenced by such lead absorption as may have occurred. Granting that there was lead absorption, I fail to see any mechanism whereby this would have precipitated hemorrhage from a duodenal ulcer. One can suppose all manner of things, and obviously, it is not reasonable to take the position that the ulcer could not have been influenced in any possible way by lead absorption. On the other hand, so far as I know, there is no clinical evidence to lead one to think seriously of any relationship between lead poisoning and hemorrhage from duodenal ulcer. In my opinion, the occurrence of the hemorrhage was a pure coincidence. (Obviously, there is always some cause behind such a hemorrhage, if it could be determined. I mean, therefore, that one could no more determine the mechanism in this instance than in any other case.) If by any chance you should come across any evidence that would lead you to believe that I am wrong in this conclusion, I should be glad if you would let me know, since I would not like to continue in an incorrect position if it could be shown to be such.

Very truly yours,

Robert A. Kehoe, M. D.

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