Memorandum on Case of [redacted]

The clinical record in this case does not give convincing evidence on which to base the diagnosis of lead encephalopathy, for two principal reasons; -

(1) There were no asscciated clinical evidences of lead intoxication in the form of anemia or gastro-enteric disturbances. The meager microscopic data on the blood are hardly adequate to exclude the possibility that there were blood changes of a type seen in plumbism, but the fact remains that evidence of the existence of such changes is lacking. Apparently there was no anorexia, constipation or colic at the onset. The colic might well be absent,but the lack of any apparent gastro-enteric symptomatology is less readily explained. (The history obtained may not be entirely reliable.)

(2) The existence of hypertension prior to the onset of the present illness provides another possible explanation for the cerebral symptomatology. (It is not clear from the record just how it is known that hypertensive disease existed. Presumably this was established in the Out Patient Dispensary, but examination of earlier records would be required to ascertain whether or not the previous illness may be explained in terms of lead intoxication.)

On the other hand, the diagnosis of lead encephalopathy has not been excluded by the clinical and post mortem findings that have been recorded. The onset and course in this case fit into the pattern of lead encephalopathy, with headache, dizziness, and a gradually developing state of mental confusion, hallucinations and mania, while the analytical data on the tissues are compatible

with the existence of an acute cerebral toxemia due to lead. (They do not prove the existence of such toxemia, since approximately corresponding lead concentrations may be found in the absence of symptoms. Normal lead concentrations in the brain range from 0.01 to 0.09 milligrams per 100 grams, in our experience; elevated concentrations without signs of cerebral or other lead intoxication range up to 0.35 milligrams per 100 grams in several cases on which we have adequate data.) In a series of cases of encephalopathy due to absorption of tetraethyl lead, the lead concentrations in the brain ranged from 0.46 to 1.65 milligrams per 100 grams, while in encephalopathy due to inorganic lead in the case of children, these concentrations ranged from 0.24 to 0.58 milligrams per 100 grams. Neither series of these cases is wholly comparable to the one under discussion, for a variety of reasons, and our data on lead encephalopathy due to industrial exposure to inorganic lead compounds is meager and somewhat questionable because of the rarity of cases of this type and the usually controversial nature of the diagnosis. However, there is adequate evidence of the existence of relatively severe and recent lead exposure in the analytical results on the tissues of the man. (Cf. data on liver, spleen, muscle and bones), and therefore, some adequate explanation of the cerebral symptoms must be given, if the possible role of lead is to be eliminated in this case. It is fair to say that the medico-legal evidence in support of a diagnosis of lead encepahlopathy in this case is quite strong, and would justify a decision in favor of a claimant for compensation, on the theory that the claimant is entitled to the benefit of the doubt, unless there is unrecorded

and unreported information in support of another diagnosis. The further examination of the brain may yield information of the latter type. From the medico-legal viewpoint it is unfortunate that somewhat greater effort was not made to confirm or exclude the relationship of lead to this illness before death occurred.

Robert A. Kehoe, M. D.

February 27, 1943

February 27, 1943

Dr. Edward McGrath,
Cincinnati General Hospital,
Cincinnati, Ohio.

Dear Dr. McGrath:

I am sending you herewith a memorandum on the case of [redacted] deceased, for such use as it may be to you. I am frankly dubious of the part that lead played in this man's illness and death, but an examination of the record does not provide a very adequate basis for differential diagnosis, so far as I can see. We are left, therefore, with certain very concrete evidence in favor of the diagnosis of lead encephalopathy, without much evidence to the contrary. I talked with Dr. Aring, who I believe, is convinced that this was not lead encephalopathy, and who believes that the examination of the brain may reveal the background of the condition. In justice to this man's wife, I think this should be done as soon as possible and that we should probably withhold any opinion or further action until it has been done.

I do not presume to put my opinion up against Aring's in the diagnosis of mental states, and I repeat that in this instance the diagnosis must be made on clinical grounds. I shall be very much interested in the conclusions eventually arrived at and in the reasons which are back of them. I should appreciate it very much if you would keep me informed.

Sincerely yours,

Robert A. Kehoe, M. D.

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February 27, 1943

Dr. Charles Aring,
Cincinnati General Hospital,
Cincinnati, Ohio.

Dear Charlie:

I am sending you a copy of a memorandum on the case of [redacted]. For a number of reasons, I am very much interested in the outcome of this case. This problem arises not too infrequently in industry, and decisions in favor of the claimant have been rendered in a number of instances on much more tenuous evidence than is available here. I hope you will find it possible to study brain in this case promptly, in justice to this man's widow, but for quite different reasons, I shall be very much interested in what you find, and in your opinion. The diagnosis here must be made on clinical grounds, but I do want to point out to you that lead encephalopathy among adults in industry is not unknown. It was much more common at one time than it is at present, because of the greater exposures to lead that once occurred in industry. Nevertheless, there are still situations in which severe lead exposure can be encountered. The plant involved in this instance provides a number of such exposures, and although I do not know the details of this man's work and environment, it is quite clear from the analytical data that he had a comparatively severe lead exposure. For this reason, it is scarcely valid, I think, to give too serious weight to the rarity of this condition. It is rare only because we have made it rare, but we have not eliminated it.

Sincerely yours,

Robert A. Kehoe, M. D.

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