REVIEW OF RECORD AND REPORT

Claim No. C. D. 30323

[redacted] Dec'd. August 19, 1943

The undersigned reviewer took part in a clinical-pathological conference with staff physicians at the Cincinnati General Hospital and the staff of the Laboratory of Neuropathology of that institution in early March 1943. At that time the entire history of the case, the clinical record, the necropsy findings, the analytical data, and the report on the gross and microscopic study of the brain were considered in detail. The microscopic slides were examined carefully by the reviewer and a number of questions were raised for discussion. The reviewer was not convinced that the microscopic evidence presented was sufficient to establish the diagnosis of lead encephalopathy beyond cavil. However, it was not possible to set up another diagnosis that could be substantiated, and the concensus of opinion of the conference, with which the reviewer concurred, was that the diagnosis of lead encephalopathy was justified for both clinical and medico-legal purposes.

A review of the record as submitted has given no reason for altering this conclusion. It cannot be said that the diagnosis of lead encephalopathy has been established without question. It is possible that the primary clinical condition was hypertensive encephalopathy, and that death resulted from the hemorrhage. However, the hemorrhage is regarded by the neurologists and the neuropathologists as incidental in this case, and that conclusion cannot be proved erroneous.

Against the diagnosis of lead encephalopathy is the opinion of Dr. Matuska who regarded the condition as primarily hypertensive,

and considered that this employee had never had symptoms or signs suggestive of plumbism. There is also the opinion of the Assistant General Manager of the Eagle-Picher Lead Company to the effect that the deceased had not been seriously exposed to lead in his work. The latter opinion, however honestly held, is not to be taken seriously in view of the analytical findings on the tissues of the deceased. These analyses demonstrate that the lead exposure had been of significant proportions, and the lead concentration in the liver, spleen and muscle prove that the exposure had continued to be significant up to a time shortly before death. The skeleton contained fifteen to sixteen times the concentration of lead found in the bones of unexposed persons. In fact, while the lead concentrations demonstrated in these tissues do not prove the existence of illness due to lead, nor do they reveal the cause of death, they do, nevertheless, fit into the range of concentrations found in unquestioned fatal cases of lead poisoning and they definitely establish the hazardous character of the occupational lead exposure.

The clinical information in the record, with respect to hematological abnormalities is inadequate, and the existence of other clinical symptoms of plumbism cannot be established. The latter is often the case when the cerebral manifestations are as severe as they were in this case. The clinical course of the disease was compatible with the diagnosis of diffuse encephalopathy from any cause. Considering all the available information, the differential diagnosis lies between hypertensive encephalopathy and lead encephalopathy. Either of these may have been the primary cause of the fatal illness. The combination of the analytical result on the brain, which is in the range associated with the occurrence of lead encephalopathy, together with the micro-

scopic pathology of the brain, which the neuropathologist regards as typical of the effects of lead absorption, tips the scales in favor of lead encephalopathy.

To exclude the diagnosis of lead encephalopathy one would have to prove that hypertensive encephalopathy was the cause of death. This, in the opinion of the reviewer, cannot reasonably be done in the face of the evidence. Therefore, the benefit of such doubt as may still exist should be given to the claimant in this case.

Signed _____
Robert A. Kehoe, M. D.

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