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STATE OF OHIO

DEPARTMENT OF HEALTH
COLUMBUS

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November 20, 1935

SUBJECT:

Dr. Robert A. Kehoe,
Laboratory of Applied Physiology,
College of Medicine,
University of Cincinnati,
Cincinnati, Ohio.

RE: [redacted]
Cincinnati, Ohio.

Dear Dr. Kehoe:

Thank you for your letter of November 18th in regard to the above case. Weighing all of the circumstances, I had concluded to approve it in our records as one of lead poisoning. Cutside of the history of possible exposure, etc., this was based upon Dr. Compton's report of supplementary evidence showing an onset with metallic taste, loss of appetite, constipation, weakness of extremities, severe pains in abdomen, also an extensive dermatitis of chest and arms - the dermatitis explained as due to sulphuric acid particles acquired from dried, pasty grids from his operation of running a slitting machine. Also objective findings of circumoral pallor, blue line in gums, W.B.C. count 6,800, blood pressure 104/60, pulse rate 58, temperature 97.6, gripping power of right hand very weak and left hand weak, slight tremor and incoordination, hyperaction of patellar reflexes, doughy-like masses in abdomen without rigidity of external abdominal muscles, and a slight loss in weight (160 to 155 lbs.), plus a whole gamut of subjective symptoms usually attributed to this form of intoxication. Dr. Compton's diagnosis was acute lead poisoning. The patient had had no previous attacks of lead poisoning.

It is true that Dr. Compton reported an absence of stippling, R.B.C. count 5,050,000, hemoglobin 104%, with good condition of teeth and gums, and no evidence of wrist drop, visual, auditory, mental, nutritional, or nephritic phenomena. All of these, of course, might be absent in a given case of lead poisoning. On the other hand, regarding possible involvement of the central nervous system, I think I am right in recalling that a rash and a low white count may occur in meningeal cases, likewise the absence of fever and the presence of a slow pulse and relatively low blood pressure. However, the usual characteristic findings of meningitis seem to be absent in this case. There remains the possibility of some true cerebral involvement, as you say, of either (low grade) infection, tumor or otherwise. No doubt the opinions of an ophthalmologist and a neurologist should also be secured. I hope these measures can be carried out. Will be much interested in your ultimate conclusions.

After some three or fours years of results following our supplementary inquiry form regarding lead poisoning, I have about come to the conclusion that far too much dependence is being placed upon laboratory reports in diagnosis of this condition. In fact, I am almost convinced that the condition cannot be diagnosed by ordinary laboratory methods (blood and urine). This, of course, does not refer to lead absorption, but to establishing the diagnosis of lead poisoning. I am seeing constantly cases turned down for compensation by our Industrial Commission because laboratory evidence of lead poisoning has not been established. You will appreciate that a large percentage of these cases are seen by physicians after the victim has ceased exposure, and oftentimes lead poisoning is not suspected until the case has become "chronic". Would like to have your views upon this point?

With all best regards,

Sincerely yours,

Emery R. Hayhurst

Emery R. Hayhurst, M.D.,
Chief, Division of Hygiene:
Consultant, Occupational Diseases.

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