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STATE OF OHIO
BUREAU OF WORKMEN'S COMPENSATION

Claim No. OD-104319

Claimant [redacted]

SPECIALIST'S REPORT

Address 1218 McIntosh Avenue, Akron, Ohio

Date of Examination July 10, 1959 Age 31 years

NOTE: Submit report under following headings: (1) History. (2) Complaints. (3) Examinations, including X-ray and laboratory work. (4) Discussion. (5) Opinion. Sign and mail THREE copies to the MEDICAL SECTION—Retain one for your file.

REPORT OF EXAMINATION TO BE RETURNED WITHIN TWO WEEKS FOLLOWING REQUEST.

HISTORY

The opinion rendered herein is based upon the examination of the record as provided to the undersigned in accordance with a letter of A.L. Kefauver, M.D., dated June 25. The claimant was not examined, nor was he requested to appear for examination, in as much as the lapse of time since the onset of his disability had been such as to render futile the application at this time of procedures which, at an earlier date, would probably have elicited precise information concerning the role played by occupational exposure to, and absorption of, lead in his illness and disability. Such conclusion as has been, or can be, arrived at at this time, must depend upon the interpretation of the facts set forth in the record, since these could not be clarified or extended by information gained from the claimant at this time.

Summary of Case

Briefly to restate the facts, Mr. [redacted] developed certain visual disturbances which were first interpreted by a physician as a toxic central optic neuritis in May 1957. Prior to this he had suffered from nervousness, dizziness and headaches and he continued to be disturbed by these symptoms, which appear to have been augmented somewhat by anxiety. The claimant's loss of vision progressed, and he was referred to various physicians and specialists, from time to time, over a period which came to an end, so far as the results of examinations have been recorded here, with the ophthalmologic examination of Dr. C. Wilbur Rucker of the Mayo Clinic on January 6, 1958, the neurological examination by Dr. N.P.Goldstein of the Mayo Clinic on January 9, 1958, and a general consideration of his problem from the viewpoint of causation by Dr. M.M. Hargraves of the Mayo Clinic, just after the examination of Drs. Rucker and Goldstein. Mr. [redacted] was seen early in his illness, and was followed up to and after his visit to the Mayo Clinic, by Dr. J.C. Damitz of Akron, a specialist in diseases of the eye, ear, nose and throat. The record does not show the precise degree of visual loss now (July 1959), although it is said in the report of an investigator (Helton 5-22-58) that all vision in both eyes has been lost, but the report of Dr. Rucker indicated that, in January of 1958, the visual acuity had been reduced to 1/60 in each eye, and that there were "large dense central scotomas". All in all, the records available are entirely satisfactory in demonstrating the clinical entity from which this claimant has suffered and is still suffering. He now has a very serious (perhaps total) loss of vision, in association with a bilateral optic neuritis of a type said by the ophthalmologists to be of toxic origin, without other evidence of disease of the central or peripheral nervous system that would explain (or help, by association, to explain) the optic involvement.

Discussion of the Case

There is no evidence in the record by which to establish reasonably or probably the cause of the injury to the optic nerves. One of the ophthalmologists strongly advised the patient against the further use of tobacco, obviously on the grounds that the effects of nicotine on the vascular supply to the optic nerves might well be the toxic mechanism. Multiple sclerosis was suggested as the basis for the optic lesions. Arsenic was thought of and evidently dismissed, but not on evidential grounds. Lead, almost inevitably, came under scrutiny, and a case was made vigorously by ~~two~~

Robert W. Kelsey M.D.
(Signature of Examiner)

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of the ophthalmologists (Drs. Kyriakides and Damitz) for the significance of the patient's occupational exposure to lead. Several points are noteworthy in connection with the hypothetical incrimination of lead in the etiology of Mr. [redacted] condition:-

(1) It does not appear that this factor was even taken seriously by anyone, including the physicians at the Cleveland Clinic or the Crile Veteran's Hospital, at the time of onset and development. It seems evident that the occupational aspect of the case, in relation to lead absorption, was not regarded as a sufficient justification for the analytical determinations of the lead content of the urine or blood, despite the fact that this has come to be the normal or expected procedure when it is suspected that lead is a factor. It is unlikely that this was merely ignored (our analytical facilities have been called upon by Cleveland Clinic physicians for years in suspected cases of lead intoxication); rather it was not believed that this patient had sustained a significant exposure to lead. The only indication in the record that either lead or arsenic was under serious consideration, initially, even for the purposes of diagnostic exclusion, is the communicatio of Dr. Hargraves of the Mayo Clinic, who reported the finding of 0.01 mg. of lead per liter of urine (approximate date January 9 or 10, 1958). The finding with respect to arsenic was not even reported, and it is evident that it was regarded as insignificant. This single instance of finding an insignificant concentration of lead (and arsenic) in the urine was not followed up with further analysis, and it is evident that the analysis was done merely as a part of a "fishing expedition". The result was not considered to be of any consequence, and even its negative value, as evidence, has been ignored by all of the physicians.

(2) In the foregoing connection, while the finding of a low normal concentration of lead in one sample of urine (volume not stated) has little diagnostic usefulness, requiring as it does confirmation in other ways, and especially by the determination of the concentration of lead in the blood, in relation to the lead content of the body, it cannot be ignored utterly. The onset of this man's illness appears to have been in the spring (April or May) of 1957. If he had lead poisoning at this time of sufficient severity to give rise to encephalopathy (in retrospect), in association with a distructive lesion of the optic nerves, he must have been subjected to severe (not borderline) exposure to lead, and he must have absorbed a large quantity of lead. (Neither encephalopathy nor neuropathy is known to have occurred except in consequence of the absorption of unusually large quantities of lead. These are the most severe forms of saturnism known, and they are growing rare because industrial exposure to lead, while common, occurs to a less severe degree than it did formerly. It is necessary to establish that an unusual degree of absorption has actually occurred in order to substantiate either of these diagnoses.) If Mr. Austin had large quantiti of lead in his body in the spring of 1957, he would not have eliminated them so completely from his body by January 1958 as to have low normal levels of lead concentration in his blood and urine. Relatively high levels persist for much longer periods of time. The blood was not examined, and the one piece of (inadequate) evidence available to us is the low normal concentratio of lead in the urine. As such, it suggests, without proof, that Mr. Austin did not have abnormal quantities of lead in his body. If anyone had really believed that lead was the responsible agent, this was the time to establish the facts by further investigation. This was not done, and in view of the eminence of the physicians who examined Mr. Austin, during the period when this might well have been done, it seems highly improbable that they considered lead to be the likely cause of his tragic condition.

(3) Such evidence (inadequate) as is available supports the probability that lead was not the toxic agent in this instance.

(a) There is no plausible history of occupational exposure to lead. The work in which this man engaged could, perhaps, have provided

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(b) As further evidence (not necessarily conclusive but of some weight) that Mr. [redacted] did not absorb lead from any source in large or even significant quantities, in the failure, on the part of the physician to find, at anytime, any of the fairly characteristic chemical or microscopic hematological changes. Repeated tests for porphyrimuria were negative, as were also tests for basophilic stippling of the erythrocytes

(c) The analytical evidence (urine), such as it is, supports the opinion that Mr. Austin did not absorb abnormal quantities of lead from any source. There is little likelihood, indeed, that he absorbed the large quantities necessary to produce encephalopathy or optic neuropathy.

Opinion

It is the considered opinion of this reviewer that there is no evidence on which to ascribe the optic neuritis of the claimant to the effects of lead absorption; that such an interpretation of the illness of this patient is altogether speculative, and can be understood only on the basis of the desire of his physicians, after the fact, to find some econo basis for assuaging his misfortune.

There is no evidence worthy of consideration that this man absorbed any significant quantity of lead in the course of his work or otherwise. (It cannot be said, with certainty, that he did not.)

There is no clinical evidence that would support the opinion that this man suffered from lead poisoning at any time.

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a significant degree of lead absorption under unusual circumstances, especially in the case of an individual who had unusually careless habits in the handling of food, tobacco, and in other matters of personal conduct with respect to the opportunities for swallowing lead. The probabilities, in terms of the incidence of lead poisoning among men who are engaged in his type of work, are entirely negligible. Even the milder forms of lead poisoning are rare indeed under the conditions of work described. (Incide ally, the expressed opinions of Dr. Kyriakides with respect to the possible role of tetraethyl lead in this situation are entirely erroneous. The ordinary handling of leaded gasoline is not even a lead hazard. The absorption of tetraethyl lead through the skin following contact with the leaded gasoline does not occur, while the vapors given off from gasoline in open containers of leaded gasoline contain only traces of tetraethyl lead. No case of lead poisoning has ever been seen in connection with the use of leaded gasoline in the manner described. The references to tetra-ethyl lead poisoning in British literature, referred to by Dr. Kyriakides relate only to cases which have arisen in the manufacture of tetraethyl lead and in connection with the peculiar situation of exposure to tetraeth lead that occurs in the cleaning and repairing of large storage tanks. Ther is no instance of a case of lead poisoning from the ordinary handling and use of leaded gasoline. The doctor is simply mistaken in his interpretat of the literature of this subject. Moreover, in well over 100 recorded c of tetraethyl lead poisoning that have occurred in the U.S.A. and abroad no case of peripheral neuritis or of optic neuritis or atrophy has occurr It is a peculiar and highly interesting fact that no organic (destructive lesions of the central or peripheral nervous system have been described in connection with tetraethyl lead poisoning in man.)

Optic neuritis due to lead is a severe manifestation of lead poisoning and is associated only with the absorption of large quantities of lead; it is also associated with some of the other clinical manifestations of extensive lead absorption, such as characteristic digestive disturbances, typical neuromuscular abnormalities and hematologic changes of more or less characteristic type. The failure in the appearance of any of these necessary diagnostic criteria, tends to eliminate lead from consideration in this case, and leaves the case, as described at the Veterens Hospital in Cleveland as one "of unknown etiology".

Signed: Robert A. Kehoe

Robert A. Kehoe, M.D.