July 16, 1971

J. F. Brennan, M.D.
National Lead Company
16th and Cleveland
Granite City, Illinois 62040

Dear Doctor Brennan:

I am sending herewith a report of the examination of Mr. [redacted] at the hands of the group of consultants, including myself, during the stay of Mr. [redacted] in the Cincinnati General Hospital.

As to his physical state and the interpretations of the findings, the report of Dr. Richard W. Vilter to me, includes the opinion of Dr. Charles Aring, head of the Neurology Department of the Hospital and Medical College. [redacted] was the subject of staff conferences and discussions in the Department of Internal Medicine, and in the Department of Neurology, and I feel sure that we have had the benefit of the judgment and experience of these men as to the nature of [redacted] present illness.

I have appended my own observations, the findings of the Kettering Laboratory, and my interpretation of [redacted] condition, in the report, of which two copies are enclosed.

I would like to emphasize to you, in this letter, that despite our best efforts, it is not possible at this time for us to arrive at a joint conclusion which, in any sense, is decisive. I have my own opinion about it, which I have recorded in the report, but I know full well that it is impossible to prove, at this time, that the absorption of lead by [redacted] was not the cause of the injury to his brain which has resulted in his present condition. His condition is unusual and atypical, so far as lead encephalopathy is concerned, but it also varies somewhat from the usual pattern of Multiple Sclerosis, which is, it seems, the most likely diagnosis of his disease, according to Dr. Aring. Dr. Aring is a very able and thoughtful neurologist. In fact, he is the ablest man whom I know in his field, and I have tremendous respect for his judgment.

I also enclose my bill for services. It is not a large bill, for my contribution to the work-up of this case was very modest. I attach the receipted bill for the work of the Kettering Laboratory, which I paid in order to include herewith all of the work for which I was responsible directly. I learned from Dr. Vilter that all of the expense associated with the work of the Cincinnati General Hospital, including its staff, is reported in a bill submitted by the Hospital. This was a surprise to me, but it is now the procedure, since the Cincinnati General Hospital is now the responsibility of the University of Cincinnati. An arrangement has been made and agreed upon by the Clinicians and the Administration of the Hospital with the full knowledge and consent of the Board of Directors of the University of Cincinnati, whereby these things are handled in a uniform manner in all instances. I have not even seen this bill, for I

have not been involved in this program since I am a member of the Hospital staff only by the full courtesy of the University, so far as access is concerned.

If our joint arrangements are lacking in any respect, I trust that you will tell me so, since I am directly concerned, professionally, with the responsibility of dealing with your requirements.

Cordially yours,

Robert A. Kehoe, M.D.
Professor Emeritus of
Occupational Medicine

RAK:wb

enclosures

DIAGNOSIS OF THE CONDITION OF MR. [redacted]

In consideration of all of the evidence obtained in the Kettering Laboratory, and of that obtained in the Cincinnati General Hospital, and of the interpretations and opinions expressed collectively by the physicians consulted, including the undersigned physician, it is my opinion that [redacted] is suffering, at the present time, from Multiple Sclerosis. This opinion cannot be proved, nor can the effects of the absorption of lead be summarily dismissed as entirely irrelevant to his condition at the present time, since there still remains, in his body, a quantity of lead (not determinable precisely) which is sufficient to elevate the lead content of his blood well above that which should obtain after two years of freedom from occupational exposure to lead, and which, at present, is sufficient to induce biochemical effects (not necessarily harmful) indicative of the active participation of lead in the metabolism of the body, with particular reference to interference with the production of hemoglobin in the blood. It is my opinion that this abnormal body burden of lead may well be a contributory factor in the degree of illness which is associated with damage to the central nervous system (brain especially) caused by the unknown factor (or factors) responsible for a somewhat typical form of Multiple Sclerosis.

Signed: Robert A. Kehoe

Robert A. Kehoe, M.D.

RAK:wb

July 16, 1971

UNIVERSITY OF CINCINNATI
COLLEGE OF MEDICINE

ADDRESS:
DEPARTMENT OF INTERNAL MEDICINE
CINCINNATI GENERAL HOSPITAL
CINCINNATI, OHIO 45229

July 9, 1971

Robert A. Kehoe, M. D.
Environmental Health
Kettering Laboratory

Re; [redacted] CGH Case # 528 109

Dear Dr. Kehoe:

Mr. [redacted] was admitted to the Cincinnati General Hospital on 6/15/71. He was referred here for evaluation of his neurologic status relative to lead exposure. He is a 42 year old black male with a 20 year work history at the National Lead Company in St. Louis working with lead batteries until four years ago when he had the on-set of crampy abdominal pains, headache and weakness. He was unable to keep up with his work, developed stammering speech and was found to have elevated lead levels at work. He was studied at the St. Louis V. A. Hospital where he has been undergoing treatment for three years. He has not worked for 2 years because of "weakness". At the present time he complains of headaches over the vertex of his skull lasting up to 2 days about once a week, instability of gait which he ascribes to his weak knees and poor coordination. He has had occasional dizziness and tinnitus. In the past he has had a duodenal ulcer and in the mid 60's was transfused with two units of blood, whether because of bleeding ulcer or of lead poisoning is not clear. His family history indicates no tendency to familial neurologic disease. Review of systems indicates only mild pedal edema for the last several years and some numbness of the palms of his hands.

On physical examination all vital signs were normal and his blood pressure was 130/90. He was a large, middle-aged black man looking his stated age and in no distress. His head was normocephalic. His skin showed some dark brown maculopapular areas on the forearm. His conjunctivae were slightly pale, all eye movements were normal. The ocular fundi showed mild arteriolar narrowing. Ears, nose and throat were normal. The tongue was well papillated and the throat normal. There was no lead line visible although he did have a mild degree of gingivitis. The neck veins were not distended, the thyroid was not enlarged, there was no significant lymphadenopathy. The lungs were clear to percussion and auscultation, there are no rales. His heart was not enlarged and there were no murmurs or gallops. All peripheral pulses were easily palpable. The abdomen showed no evidence of organ enlargement and no masses. There were no hernia and the testes were normal. Rectal examination was negative, the prostate was not enlarged, tender or nodular. There was no edema or tenderness of the extremities, the joints were essentially normal. On testing motor power, the extensors of the right lower extremity seemed weaker than the left, and there was generalized but mild weakness especially in the grip of both hands. A neurological consultant found that he was awake, alert, and oriented, recent memory was fair to good, information of recent events was relatively poor. He was unable to recall his complete address. General information was good. Speech was disarthric, there was no aphasia, cranial nerves

were as follows: visual fields were full; the discs were slightly pale; there was no venous congestion; the pupils were 3 mm and reacted to light and accommodation. There was bilateral fine horizontal nystagmus which was not persistent. Extra ocular movements were full. There was no facial weakness, hearing was good, gag reflex was normal, tongue protruded in the midline.

There was decreased strength in the hands and in the bicepts but this appeared to be volitional, otherwise no weakness of extensor muscles of the hands or legs could be found. There was a fine tremor on extension of the hands and forearms and some titubation of the head. There was increased tone in the legs. Sensation was generally intact with some decrease in the right great toe and foot, position sense was intact, cortical sensation intact. There was marked dysmetria on finger to finger and finger to nose test and rapid alternating movements of the hands seemed to be abnormal. His gait was good. He walked well on his toes and heels, though he was maderately unsteady and seemed to be weak. All deep reflexes were present, equal and active. There was a positive snout reflex and negative Hoffman's. It was the opinion of this consultant that there were cerebellar signs including dysarthria, nystagmus and dysmetria, and possibly pyramidal tract signs with some increase in the deep tendon reflexes.

The patient was seen at Neurology Grand Rounds after having been transferred to the Neurology Service for special studies. He was presented to Doctors Aring, Olinger, Schneider and Staff with Dr. Biehl moderating and Dr. Dotson giving the history. His examination at this time showed ptosis bilaterally but no nystagmus. There was slowness and slurring of speech, "asthenic like" speech, as described by Dr. Biehl. He had tremulous hands and dysmetria on finger to nose and on heel-knee-shin test. His reflexes were brisk. It was the opinion of Dr. Aring and the Neurological Staff that none of them had seen this syndrome associated with lead intoxication, though all agreed that he had had lead poisoning in the past and still had significant lead in his tissues. They felt that lead encephalopathy could not be ruled out however but that multiple sclerosis should be seriously considered. The gamma globulin test in the spinal fluid could not be performed anywhere around this Medical Center. This is positive in about 70% of patients with multiple sclerosis.

Because of the possibility that myasthenia gravis might be responsible for the ptosis and general asthenia, a tensilon test was performed which was negative. Dr. Park Biehl of the Neurology Department wrote a dissenting opinion. It was his feeling that lead could probably produce a chronic asthenic process such as this. He did not favor myasthenia gravis and would not favor multiple sclerosis either.

Dr. Robert Kehoe

Re: [redacted] # 528 109

The following laboratory procedures were performed: Urinalysis - negative; Prothrombin 73%; Uric Acid 7.2 mg%; Urine Culture - sterile after 24 hours; Watson Schwartz Test - porphobilinogen - negative; serum iron 89 mcg%; serum iron binding capacity 288 mcg %; BUN 16; CO_2 20; chloride 100; blood sugar 114; white count 11,800; red count 4,370,000; hemoglobin 11.7 gms; hematocrit 37%; MCV 82; MCH 26; MCHC 31; polys 56; STABS 1; lymphs 37; Monocytes 2; EOS 4; platelets adequate. No stipple cells seen. Red cells normocytic and normochromic. Cerebral spinal fluid was under normal pressure, crystal clear, no cells, quantitative protein 52 mg%; gold curve negative, brain scan was within normal limits as was the electro-encephalogram. X-rays of the skull and skelatal survey were negative. X-ray of the chest was normal. X-ray of both hands showed an old deformity of the left first metacarpal bone. Upper GI series showed mild deformity of the duodenal bulb in an otherwise remarkable upper GI series. Electromyography and nerve conduction studies showed no abnormalities in conduction of nerves of the right upper extremities. There were abnormal responses of the tibial nerve and low amplitude of perineal nerves. There was evidence of long standing distally distributed denervation speaking in favor of old polyneuropathy of lead intoxication. I understand from a telephone report that you sent me that there is still a significant amount of lead in [redacted]

My opinion based on all of the evidence derived furing Mr. Latchison's stay at the Cincinnati General Hospital is as follows. He has had lead exposure over many years and has had symptoms of lead poisoning in the past. He has nerve conduction studies suggesting old neuropathy probably due to this etiology but this is of very mild degree. None of our Neurology Staff have seen the exact encephlopathic picture which [redacted] presents in association with lead poisoning; however, it is impossible to completely eliminate lead as the causative factor though it seems that multiple sclerosis is more likely. The mild degree of anemia is probably the result of lead poisoning.

Very sincerely yours,

Richard W. Vilter

Richard W. Vilter, M. D.
Director
Department of Internal Medicine

cc: Dr. Charles Aring
Dr. Don Nelson
Dr. Elliot Familant

KE 0065066