WORKMEN'S COMPENSATION BOARD OF BRITISH COLUMBIA
707 WEST 37th AVENUE, VANCOUVER 13, BRITISH COLUMBIA. TELEPHONE 266-0211

July 16, 1968

Doctor Robert A. Kehoe
345 Resor Avenue
Cincinnati, Ohio
U.S.A.

Dear Doctor:

Re: Ferenc Haradi
Claim #67090880

Enclosed please find cheque number 9410-286480 in the amount of twenty dollars ($20.00) in payment of your account dated April 9, 1968 for services rendered this workman .

Yours truly,

J.D. Hornald
Medical Aid Division

JDH/dw
Enclosure

N13159

KE 0012471

WORKMEN'S COMPENSATION BOARD OF BRITISH COLUMBIA
707 WEST 37th AVENUE, VANCOUVER 13, BRITISH COLUMBIA. TELEPHONE 266-0211

May 10, 1968

Mr. Ferenc Naradi,
845 East 16th Avenue,
VANCOUVER 10, B. C.

Dear Sir:

Re: Your Claim No. 670908BO

Your claim has been reviewed by the Board of Review and Commissioners and it has been recommended that your claim now be accepted as a Board responsibility.

You should submit any accounts applicable to the condition for which you were treated on December 16, 1967. We should also like to have the date you first returned to employment after December 16, 1967 having this confirmed by your employers, and the dates of any subsequent lay offs should be included.

Yours truly,

D. A. Moffatt

D. A. MOFFATT,
CLAIMS DEPARTMENT

DAM/cn
UNIT # 5

c.c.: Metalex Limited
c.c.: Dr. C. W. M. Murdoch
c.c.: Dr. Robert A. Kehoe
c.c.: Dr. D. P. Jones
c.c.: Mr. Bell, Vancouver General Hospital Social Service
c.c.: Dr. Wm. S. Huckvale

N13159.01

KE 0012472

April 9, 1968

R. M. Hayes, M.B., M.R.C.P.
Medical Officer
Workmen's Compensation Board of
British Columbia
707 West 37th Avenue
Vancouver 13, British Columbia
Canada

Re: Ferenc MARADI - Claim 67090880

Dear Doctor Hayes:

I do not wonder that you and your associates are in some doubt as to the correctness of the diagnosis of lead poisoning in the case under consideration as noted above. I find myself in a somewhat similar situation, by virtue of certain contradictions in the findings of fact, as well as the conflicting opinions expressed in the records sent to me. I shall undertake first to state the facts as I view the record, then to set the contradictory views opposite each other, and to express some skepticism as to the validity of certain data, and finally to give you my opinion for what it may be worth on the background of the uncertainties.

1. There seems little doubt that this man has had a sufficiently prolonged period of employment in a hazardous lead plant to have absorbed, plausibly, a dangerous quantity of lead. There is a valid question as to what he was doing, and it is important to recognize that the different jobs in these plants yield very different degrees of severity of exposure. Nevertheless, I tend to accept the probable significance of his exposure (subject however to the analytical evidence of the extent of the exposure, of which more later).

2. The finding of a "lead line" of the gums seems to have been confirmed, but this has been given undue weight in the diagnosis. This line, when present and due to lead is indicative of more than normal absorption of lead, but does not mean that such absorption is of sufficient severity as to justify the diagnosis of lead poisoning. (I have seen a well defined lead line in its development when the concentration of lead in the blood did not exceed 50 micrograms per 100 grams of whole blood at any time during the development of the line.)

3. Of considerably greater significance are the clinical findings of apparent weakness of the extensor muscles of the hand and wrist. The latter was not "wristdrop" as referred to repeatedly in the record, but rather was apparent weakness. The latter varied, it seems, with the method of testing and the time of testing, and could hardly have been certainly significant. The evidence of electromyography, however, indicates the presence of muscular impairment, which was of such a type as to be compatible with the effects of the absorption of lead. I do not trust this information implicitly, but I am influenced by them.

4. The urinary coproporphyrin is said to have been appreciably elevated (3+). If this was true, it is an important item to add to the other features of the case. I do not trust this analytical finding as the product of a non-quantitative test, but if it was done by a knowledgeable and experienced person, it has important

The contradictions seem to me to be as follows:

1. One clinician has regarded the so-called "wrist drop", i.e., the extensor weakness, as insignificant, from the aspect of a neurological lesion.

2. The analytical result of "0.56 micrograms of lead per 100 cc"of whole blood (only one is reported) if correct, is incompatible with the diagnosis of lead poisoning provided it was obtained during or in virtually immediate relationship to the exposure to lead that is credited with responsibility for excessive absorption of lead.

(I do not accept the interpretation, given in the record, of the significance of the urinary findings, with respect to lead prior to or during the therapy with "versenate" /which I take to have been disodium calcium versenate/. That this man had absorbed abnormal quantities of lead in the course of his work is reasonably certain, but there is no reason from these findings to conclude that his absorption was in the range of amounts which are compatible with lead intoxication.)

3. "Stippling" of the erythrocytes has no certain relationship to lead poisoning as a diagnostic test. When it is due to lead, and is increased beyond normal limits, it signifies abnormal absorption of lead, but not necessarily, lead poisoning.

Conclusion:

If I were certain of the accuracy of the result obtained by the analysis of a sample of blood obtained presumably at the height of this man's occupational exposure to lead, (there appears to be only one such result), I would not be able to arrive at a diagnosis of lead poisoning, for I have never observed the onset of lead intoxication in association with such a level of lead concentration in the blood

However, the several items of evidence which favor the diagnosis of lead poisoning, when combined, make it impossible for me to offset them with the one analytical result on the blood. (The analytical error of the best of the analytical procedures and of the best of technical performances, is of the order of $\pm$ 10 percent. It is often considerably greater in relatively inexperienced hands. I would need to have knowledge of the analytical precision in this instance if I were to bank on it.)

I cannot, therefore, either arrive at the diagnosis of lead poisoning, nor exclude it. Accordingly, I should be disposed to give the benefit of the doubt to the claimant in this instance, not so much because of the benefit to be derived from compensation, as from an unwillingness to subject him to the continuation of his employment under the hazardous conditions which might lead to irreversible disability (palsy).

I regret that I find myself in this position. I am sure that if this man had been studied properly at the time of the onset of his apparent illness (with particular reference to the verification of the analytical findings), a firm diagnosis could have been arrived at. With the lapse of time and the application of chelation therapy, it is doubtful that a definitive conclusion could now be reached.

Sincerely yours,

RAK:wp

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

April 9, 1968

To: Workmen's Compensation Board of
British Columbia
707 West 37th Avenue
Vancouver 13, British Columbia
Canada

Attention: R.M. Hayes, M.B., M.R.C.P.

For: Reimbursement to Robert A. Kehoe, M.D.
For the Review and Recommendations in regard to
Ferenc MARADI - Claim 67090880

. . . . . $20.00

March 25, 1968

R. M. Hayes, M.B., M.R.C.P.
Medical Officer
Workmen's Compensation Board
of British Columbia
707 West 37th Avenue
Vancouver 13, British Columbia

Re: Ferenc MARADI
Claim 67090880

Dear Doctor Hayes:

This is an acknowledgment of your letter of March 18, 1968 to Doctor Robert A. Kehoe. He is presently on a business trip abroad and will not be back in his office until early April.

I shall, of course, bring your communication to his attention upon his return, and I am certain you will be hearing from him sometime in April.

Very truly yours,

(Mrs.) Winifred Peaslee
Secretary to
Robert A. Kehoe, M.D.

wp

WORKMEN'S COMPENSATION BOARD OF BRITISH COLUMBIA
707 WEST 37th AVENUE, VANCOUVER 13, BRITISH COLUMBIA. TELEPHONE 266-0211

March 18, 1968.

Dr. Robert A. Kehoe,
The Kettering Laboratory,
College of Medicine,
University of Cincinnati,
Cincinnati 19, Ohio.

Re: [redacted] Claim 67090880

Dear Doctor Kehoe:

Lead absorption and lead intoxication continue to present problems to this Board. In the past you were good enough to assess the evidence on a claim and make recommendations. We are having considerable debate and difference of opinion in this case and would again request your assistance.

The claimant, above-named, is 35 years of age and has been employed with a metal salvage company for 19 months. Housekeeping in the plant leaves much to be desired and we have received and accepted several claims from employees of this company.

I am enclosing copies of multiple consultants reports which will give you all of the clinical and laboratory findings.

Our Industrial Hygiene Department wrote a memo to this file on March 12, 1968, which states as follows:

> "This workman's urinary lead excretions have been well below the suggested safe limit of 200 micrograms per litre since mid 1966, with one exception in January 1967 when his urinary lead level was 320 micrograms per litre. The lead excretion during treatment (7 grams, 5 grams and 5 grams) was not significantly higher than that of unexposed persons on the same treatment.
>
> Multiple cases of lead poisoning amongst other workmen in this plant, the presence of urinary coproporphyrins and basophillic strippling support a diagnosis of excessive lead absorption by this workman.
>
> Limited symptoms consisting of a finger and wrist weakness do not support this diagnosis and other possible causes of this should be considered."

The present position of the Medical Department is that this man has absorbed considerable lead but his symptomatology and laboratory reports do not support a diagnosis of lead intoxication.

pensation Board
sh Columbia

Dr. Robert A. Kehoe -- 2. March 18, 1968.

Re: [redacted] - Claim 67090880

We will await your opinion with considerable interest and of course will expect your account for the usual consultation fee.

Sincerely yours,

R. M. Hayes, M.B., M.R.C.P.
Medical Officer

RMH:lm
encls.

KE 0012478

6709080

DR. D. P. JONES
750 WEST BROADWAY
VANCOUVER 9, B.C. 27 December, 1967.

Dr. G.M.M. Murdoch,
905 - 750 West Broadway,
VANCOUVER 9, B.C.

Dear Dr. Murdoch:

Re: [redacted]

[redacted] told me that for the last nineteen months he has beenemployed in operating a furnace to melt lead batteries in company with about a dozen other men. About four weeks ago he noticed weakness in extending the fingers of his right hand (he being right-handed) and two weeks ago the left hand was similarly affected. There is weakness also in extending the wrists. He does not complain of any pain or numbness and he has no trouble with the legs. His appetite has been poor recently but there is no history of abdominal pain. He gives a past history of limb pains during a five month period in 1958 when he was employed as a miner in Hungary. Otherwise his health has been good in the past and he comes from a healthy family.

On examination he looked rather pale. There was a well marked blue line along the gum edges, especially in the upper jaw. The optic disks and other cranial nerves were normal. In the arms there was no wasting. At rest the hands were slightly flexed at the wrists and the fingers more so. Voluntary dorsiflexion of the wrists could be done to a slight extent but not of the fingers whereas the flexor muscles were strong. He was unable to abduct the fingers but could do so a little when they were extended passively. Power in proximal muscles was normal, sensation was intact and the tendon jerks were normal and symmetrical. In the lower limbs power, co-ordination, sensation and reflexes were normal. The superficial abdominal reflexes were present and the plantar responses flexor.

I agree that he has lead palsy and I think it would be best if you admitted him to hospital now for chelation. My experience with chronic lead poisoning is meagre but according to the books the eventual prognosis is good. He will need to find a different kind of job afterwards.

I should be most interested to see him again in hospital.

Yours sincerely,

DPJ/e

N13159.06

KE 0012479

DR. D. P. JONES
750 WEST BROADWAY
VANCOUVER 9, B.C.

4 January, 1960.

221

67090880

39 C'

Dr. G.H.H. Harbord,
905 - 750 West Broadway,
VANCOUVER, B.C.

Dear Dr. Harbord:

Re: [redacted]

Dr. Ruckvale telephoned me today saying that he thought [redacted] symptoms were hysterical in origin and not due to lead poisoning. I had considered the possibility of a functional disorder myself but eventually discarded it on the grounds that there was too much positive evidence. I am sure that Dr. Ruckvale's opinion is one to follow in this case but nevertheless I am still worried about what would happen if an intoxication were missed and I would favour asking the Board's permission for hospital admission for a few days. If the condition is hysterical no harm would have been done by a short admission. It is unfortunate that I have not experience enough to make a firm diagnosis on present evidence but if he does go in I should be glad of the chance to see him again.

Yours sincerely,

DPJ/e
c.c. Dr. W.L. Ruckvale
c.c. Workmen's Compensation Board
Claim No. 67090880.

KE 0012481 N13159.08

Dear Dr. [illegible]: [redacted]

[illegible]. C. [illegible]. no. 670[illegible]

The above noted consulted me on 2 Jan.68 at your request regarding the possibility of his symptoms and signs being due to lead poisoning.

History: He [illegible] worked [illegible] variety of jobs at [illegible] Ltd. for the past sixteen months. [illegible] is primarily engaged [illegible] a variety of scrap (chiefly old batteries) into metallic lead.

In [illegible] of 1967 [illegible]'s hand[illegible] started to shake. The [illegible] lasted about one [illegible]. Following this he couldn't extend the [illegible] and [illegible] fingers of his right hand. After another [illegible] all the fingers of his right hand [illegible] affected; in another week the fingers of his left hand could not be [illegible] fully.

Past Ill[illegible]: [illegible] as a child.

[illegible] condition [illegible] working [illegible] a coal mine in Hungary [illegible] forced him to seek employment elsewhere. [illegible] couldn't determine the nature of this illness he attributes to the coldness in the mine.

Physical [illegible]: [illegible] positive findings of the [illegible] on [illegible]:

1. [illegible] reference to [illegible]

[illegible]

KE 0012482

[illegible] tooth.

III. The [illegible] flexed position of his forearm, on request he couldn't or didn't extend his fingers, but in doing the pointing test, with eyes closed, his index fingers were fully extended.

It is [illegible] to say [illegible] that neurologically I [illegible] nothing positive. [illegible] the examination of Mr. [illegible] [illegible] sterile.

Laboratory [illegible]: [illegible] recent blood lead test [illegible] 56 [illegible] [illegible] of [illegible] blood. [illegible] Commission [illegible] a [illegible] record of [illegible] of urine [illegible] lead from December 1956 to October 1957 inclusive. Only one of these is consistent with a diagnosis of lead poisoning that of Jan [illegible] 57 at [illegible] micrograms per liter.

Conclusions: I [illegible] believe this man's symptoms or signs are due to [illegible] of lead. He doesn't present [illegible] symptoms [illegible] either acute or chronic lead poisoning. Of [illegible] the only one to [illegible] [illegible] in the [illegible] [illegible] his teeth. [illegible] this [illegible] for [illegible] [illegible] it is more [illegible] [illegible] it is rare for it to appear [illegible] [illegible]. The symptoms referable to his [illegible] [illegible] attributed to lead [illegible] [illegible] there is no sign of [illegible]

[redacted]

[illegible] in any of the muscles supporting the wrist joints. (The effect of inorganic lead is mainly on muscles) Lastly the laboratory tests dont support a diagnosis of lead poisoning. In few diseases have laboratory procedures been worked out with the precision by which lead can be detected in body fluids; without positive laboratory evidence it is almost impossible to make a diagnosis of lead intoxication.

I am sorry I cant be of more help in this case.

Yours truly,

W. B. Hackbarth

copy:
Dr. W. F. Jones
I. C. B.

COPY

-75 REV.63

VANCOUVER GENERAL HOSPITAL

CONSULTATION

REQUISITION

HP B-4

Mr. [redacted] 66-29-17

DR. D. P. JONES

DEPARTMENT REQUESTED PHYSICAL MEDICINE CONSULTANT W. ST.J. BUCKLE

DIAGNOSIS ? Lead Neuropathy. ? Hysteria

RELEVANT DATA

DATE 12th January 68 19 REQUESTED BY D. P. JONES

DO NOT WRITE IN THIS SPACE

REPORT

12th January 1968 DATE

FINDINGS I saw this patient this morning for electrodiagnostic assessment of bilateral wrist drop, diagnosed alternatively as lead poisoning or hysteria. He had been working melting lead batteries until a month ago and has had progressive wrist drop starting on the right six weeks ago. Clinically this is genuine and not hysterical.

In intensity duration curve plotted from the right extensor carpi radialis showed a slightly sluggish response to the 100 millisecond stimulus at 1.9 milli-amps, rising to 7 milli-amps at 1 millisecond, and 22 milli-amps at 0.1 millisecond.

The electromyograph from the same muscle showed a train of fibrillation potentials at rest. On volition there was a sparse pattern of slightly low voltage motor unit action potentials, with a tendency to polyphasicity.

Conduction times were also recorded from the median and ulnar nerves at 51 and 55 metres per second respectively.

This patient shows definite electrical evidence of partial denervation, though of minor degree, supporting the diagnosis of lead neuropathy. The conduction velocities measured in the clinically unaffected median and ulnar nerves are within normal limits, though only just in the former.

15th January 1968

I saw this patient again today, after discussion with Dr. Jones, for electromyography of other muscles. The extensor pollicis longus shows profuse fibrillation and many positive potentials at rest, with a discreet pattern of small diphasic motor unit action potentials on volition. The abductor pollicis brevis is silent at rest, with a complete pattern of normal motor unit action potentials on volition.

N13159.09

Conduction times in the right median nerve were extended up to [illegible] point and showed a conduction velocity in the upper segment of 53 metres per second, again being 51 metres per second in the forearm.

Repetitive stimulation of the median nerve produced an evoked action potential of 12 milli-volts from the abductor pollicis brevis. This did not decline on rates of stimulation up to 25 per second.

There is absolutely no doubt that the wrist drop is organic, due to (radial) nerve damage, and not hysterical, as has been alleged.

[illegible] Meckler, M.Phys.Med.
Director of Physical Medicine

c.c.: Dr. D. T. Jones
Dr. Murdoch
Dr. [illegible] Mukovala
M.M.S. (2)✓

5 REV.63

VANCOUVER GENERAL HOSPITAL

CONSULTATION

REQUISITION

COPY

67080882 RP 3-4

Dr. 64-27-19

[redacted]

Dr. D. A. Jones

DEPARTMENT REQUESTED PHYSICAL MEDICINE CONSULTANT Dr. A. W.J. MEHLER

DIAGNOSIS Lead poisoning.

RELEVANT DATA

DATE 23rd January 68 19 REQUEST OF Dr. D. A. Jones

REPORT

29th January, 1968 DATE

FINDINGS I saw this patient for the third time this morning, after he had started Versine therapy for lead intoxication, with wrist drop from a peripheral neuropathy. Clinically he showed some improvement in extensor power in the thumb, right more than left.

Conduction times were repeated from the right median nerve at 4 milliseconds at the wrist and 8.9 milliseconds at the elbow, with an intervening distance of 255 mm., giving a conduction velocity of 53 metres per second in the forearm. In the right ulnar nerve the times were 3 milliseconds at the wrist, 10.7 milliseconds in the upper arm, with an intervening distance of 233 mm., giving a conduction velocity of 56 metres per second.

The electromyograph from the extensor pollicis longus, extensor digitorum communis and the brachioradialis all showed profuse fibrillation and many positive potentials at rest. All these muscles and the right triceps, which was silent at rest, showed alteration in the motor unit action potentials, which were more polyphasic and of lower voltage than normal, with a reduced summation pattern, particularly in the extensor digitorum communis and to a less extent in the extensor pollicis longus, being least in the triceps.

This patient shows, if anything, a slight improvement in nerve conduction, but still has many denervation potentials and a marked alteration in motor units. Repetitive stimulation of the right radial nerve failed to show any end-plate block in the extensor digitorum communis.

DO NOT WRITE IN THIS SPACE

COPY

DR. D. P. JONES
750 WEST BROADWAY
VANCOUVER 9, B.C.

V.T.H. Unit No. 66-29-19
DISCHARGE SUMMARY

[redacted]

...ed to hospital for investigation of presumed lead poisoning as ...es. He had been seen prior to admission (on January 2nd) by a ...rted no positive neurological findings, and who did not find any symptom to suggest lead poisoning.

Investigations prior to admission:

Dec. 20/67 — blood urea 34 mgs%.
PSP: 67% excretion in two hours.
Urine: large amount of coproporphyrin.
Blood lead 56 [illegible] per 100 ccs.

Preliminary hospital tests:

Urine: Coproporphyrin +3, otherwise normal.

Blood: Haemoglobin 11.3 g%
+2 microcytosis
Stippling present
Lead 44 [illegible] per 100 ccs.

Spinal Fluid: Protein 30 mgs%, Kolmer negative, pressure 185 mm.

Effect of Chelation:

The patient was given three courses of versenate treatment: January 16 - 19 (7G),
January 23 - 28 (5G),
February 2 - 7 (5G).

Urinary lead excretion ([illegible] per litre) during the treatment period was as follows:

Jan. 16	90)		Feb. 1	80	
17	1700)		2	100)	
18	2800)	Versenate	3	900)	
19	1800)		4	1000)	Versenate
20	1400		6	390)	
21	420		7	560)	
22	210		8	90	
23	250)		9	110	
24	100)		11	90	
25	1400)	Versenate	12	-	Urinary porphyrins neg.
26	800)				
27	620)				
28	360)				
29	190	Urinary porphyrins negative			
30	160				
31	120				

N13159.11

[illegible] 0013187

[redacted] DISCHARGE SUMMARY (continued)

Other laboratory investigations were as follows:

Electromyography: This was performed by Dr. Buckler on January 12th, 15th, 23rd and 29th. It showed clear evidence of partial denervation of the affected muscles.

Serial photographs of the gums: I think these showed improvement in the lead line.

Kidney function: Blood urea 24 mg% and 22 mg%.
PSP (Strong Lab.) 73% of normal.
Blood creatinine 1.12 mg%.

Course in hospital:

Physiotherapy and occupational therapy led to slight improvement but it is anticipated that recovery will take place only slowly. The patient will attend for further treatment as an outpatient.

Evidence upon which a diagnosis of lead neuropathy is made:

1. History of possible exposure and the patient's statement that fellow-workers in the plant have been affected.
2. A typical clinical picture of weakness affecting the extensors of wrists and fingers, right more than left in a right-handed man.
3. Incontrovertible EMG evidence of denervation, thus disposing of a suggestion that the condition is hysterical.
4. Blue line on the gums.
5. Anaemia.
6. Basophilic stippling.
7. Renal involvement.
8. Excess coproporphyrinuria, disappearing with treatment (Med. J. Austr. Feb. 25, 1967, Br. J. Industr. Med. 1967:24.203).
9. Massive lead excretion after versene treatment (Soc. Med. Hop. Paris 1966:117.427).

DISCUSSION:

This man was shown at neurology ward rounds and one (only) speaker discounted the diagnosis of lead poisoning - on the grounds that blood and urine lead levels had been mostly normal. There is evidence in the literature that examination of a single specimen of urine is meaningless (Brit. J. Indusr. Med. 1966:23.263) and it is reported (Lancet Jan. 22, 1966, Med. J. Austr. Feb. 25, 1967) that blood and urine examinations for lead do not provide infallible evidence: rather, the results of these tests are to be considered not in isolation but as components in a range of tests. The Lancet editorial states "In spite of the new diagnostic aids, however, lead poisoning remains firmly a clinical diagnosis, and it is for the physician to decide, on the basis of clinical and laboratory data, when a man is being poisoned by lead".

Diagnosis on discharge: Lead neuropathy.

This patient is to be shown on Grand Rounds on Thursday, March 7th, 1968.

D.P. Jones, M.D.

DPJ/e

Feb. 19/68.