INTERPRETATION

[redacted]

The lead concentration in these tissues is well above normal levels, as would be expected from the history of exposure. The concentration in the brain is about four times the upper normal level. These findings, however, in themselves, do not justify the diagnosis of lead encephalopathy, since such results have been obtained repeatedly without relation to demonstrable clinical plumbism. On the other hand we have found lead concentrations of these magnitudes in association with well defined lead intoxication. The diagnosis, therefore, must be made on clinical grounds, the analytical results merely tending to demonstrate the absence or the occurrence of significant lead exposure. In the absence of evidence of significant lead exposure, the diagnosis of lead poisoning is untenable; when present, the evidence can only confirm a conclusion arrived at on clinical information.

Robert A. Kehoe, M. D.

I should be interested in having the clinical record.

Dr. Gibson
Dr. Evans
Dr. Ritterhoff

INFORMATION REQUIRED ON CASES FROM WHICH TISSUES ARE OBTAINED AT NECROPSY FOR KETTERING LABORATORY OF APPLIED PHYSIOLOGY

See the complete record of a contaminated case in hospital

Name [redacted]

Necropsy number 180068 Date of Necropsy 1-28-43

Hospital number N-43-44 Date of Death 1-27-43

Age 50 Sex ♂ Nutrition Fair Length of Body 160. cm.

Clinical Diagnosis Approximate Duration of Illness 20 DAYS.

HYPERTENSIVE ENCEPHALOPATHY, POSSIBLE LEAD ENCEPHALOPATHY
TERMINAL LEFT LOWER LOBAR PNEUMONIA.

Anatomical Diagnosis

1-8-43 ONSET OF STAGGERING – KNOWN HYPERTENSIVE FOR 1 YR.
1-16-43 PERSONALITY CHANGES & MENTAL CONFUSION & DYSPHASIA
1-25-43 EXPLORATORY BURR HOLES – NEG. FOR SUBDURAL.

Previous Medical History in Brief

Occupational History WORKED FOR EAGLE Picher LEAD CO.
31 YEARS c̄ HIST. OF EXPOSURE TO LEAD.

History or Evidence of Exposure to Lead, Arsenic, Mercury
31 YRS.

Pertinent Remarks NO CLIN. EVIDENCE OF LEAD POISONING.

TISSUES DESIRED

(Please obtain such of these as circumstances will permit, giving weights of entire organs when available. Weight of sample provided will be determined in Kettering Laboratory.)

Organ	Total Weight	Weight of Sample	Organ	Total Weight	Weight of Sample
✓ Lungs (R) —	515		Brain	1290 gms.	
(L) —	890				
✓ Heart —	415		Spinal Cord		
✓ Liver —	1625		Striated Muscle		
✓ Spleen —	190		Adipose tissue		
✓ Kidneys —	270		Long Bone		
Suprarenals	25 gms (1)		Rib		
Stomach and Intestines			Testicle		
Prostate	35 gms.		Skin		
Thyroid					
PANCREAS	100. gms.				

[redacted] — Age 50 years 20 days

1st floor — making soldering, lead pipes
molten lead area

These jobs then moved to basement

making bullets for King's mills
for period of several months

Became ill — 3 or 4 months ago to 1 year
ago — vomiting, headache
Dx'd as hypertensive encephalopathy

Took sick on 8th day of Jan
vomiting and headache
Doctor saw him on 9th
1 week later began to have as delirium
talking about family in England
feared losing his mind — spoke of it
to step son and neighbor
Talked constantly, became maniacal
and had to be restrained — moved
continuously — Dr Matuska would
not send me to hospital
Mrs. Todd, friend of E.P.
visited — went to see Dr.
Went to CGH on 19th
Died on 27th

KE 0016812

[redacted] [redacted] file in "Destruction - Men" file Autopsy - 1-28-43

180068 N-43-44 Clinical Summary

1-19-43. On 1-8-43, patient became dizzy and was seen to stagger. Since then the patient has had severe occipitalheadaches. Several hours after the onset of dizziness, he began to vomit. Vomiting continued for approximately three days. Patient complained of slight abdominal pain, and of double vision. Patient remained in bed until the time of admission. One week after the onset of his illness, he began to be irrational, and required physical restraint. Also suffered hallucinations and delusions. Food intake had been normal until three days before admission. Patient had been a known hypertensive for over one year. At this time, because of vomiting and bilateral abdominal pain, accompanied with abdominal swelling, he was studied in the clinic and numerous x-rays were taken at that time. Patient was not hospitalized, however.

Patient had pneumonia in 1927. Review of symptoms revealed that the patient had worn glasses for the last six or seven months because of diminution of acuity of vision. Nocturia 1 x. Occupational history revealed that he had worked at the Eagle Pitcher Lead Co. for approximately 30 years.

Examination revealed a well developed, well nourished, poorly oriented, 50 year old white male with some blurring of his speech. Pupils were small and reacted poorly. E.O.M. normal. The retinal fundi were not well examined, because of lack of cooperation. However, it was thought that there was some edema of the left nerve with graying of the fundi and some A.V. nicking. There was a coarse horizontal nystagmus. B.P. 195/110. No lead line on gums. Neurological examination negative except for bilateral plantar reversals.

Laboratory Work:
Urea N. 65 mgm. %, Wassermann and Gold curve negative.
B.U.N. 16 mgm.%.
Routine laboratory studies revealed no anemia and no stippling of red cells. Urine contained albumen and numerous WBC. Lumbar puncture yielded clear, slightly yellow fluid under initial pressure of 140 with ++++ Pandy, 2 WBC and protein of 95 mg. %.

On 1-21-43 patient developed urinary retention and a retention catheter was inserted. He had been running a daily temperature elevation between 100 and 102, since 1-20-43. X-rays of skull on 1-23-43 showed a few areas of hyperostosis in frontal region, but were otherwise negative.

Patient was transferred to surgery following negative exploratory bi-occipital and bi-subtemporal burr holes under local anesthesia. B.P. on day following was 140/90.

1-26-43. Patient noted to be much worse, dyspneic, with slight cyanosis. He was less dyspneic than previously with slight spastic twitching. T. 103.8 (rectal), pulse 132, B.P. 120/70. He roused to mutter unintelligibly when disturbed.

1-27-43. Patient's condition was progressively downhill and he expired at 3:35 P. M.

Pat case file

UNIVERSITY OF CINCINNATI
[INTERDEPARTMENTAL CORRESPONDENCE SHEET]

To Doctor Robert Kehoe
Kettering Laboratory
College of Medicine

From Doctor Charles D. Aring
Neurology Offices
Cincinnati General Hospital

Date February 26, 1943

Dear Bob,

Slides on [redacted] have not come through yet. I'll send a copy of our record as soon as it is complete, probably in about a week.

As ever,

Chas.

KE 0016814

[redacted]

Hypertensitive encephalopathy -- Possible lead encepapholopathy

31-year exposure to lead -- Eagle ~~Brand~~ Picher Lead Company - Bradbury

Necropsy 1-28-43 -- Drs. Gibbs, Evans, and Ritterhoff

Analysis Number	Tissue	Weight Grams	Mg. Pb Found	Mg. Pb/100 g.
43A-245	Lung	30.7	0.03	0.098
43A-246	Heart	23.5	0.004	0.017
43A-247	Kidney	23.5	0.040	0.170
43A-248	Spleen	32.6	0.130	0.400
43A-249	Liver	27.0	0.32	1.18
43A-250	Brain	41.5	0.20	0.48
43A-251	Pectoral Muscle	28.0	0.07	0.25
43A-252	Pancreas	34.0	0.03	0.088
43A-253	Clavicle	7.5	1.14	15.20
43A-254	Rib	7.5	1.22	16.25
43A-255	Suprarenals	3.8	0.004	0.105

Samples Received: 1-28-43

Samples Reported: 2-2-43