

May 1, 1948

Department of Pathology,
Cincinnati General Hospital.

Subject: Analysis of Tissues - [REDACTED]

I enclose herewith two copies of a report of our analyses of the tissues of one Oliver Smith. One copy of this, presumably, should be forwarded for inclusion in the hospital record of this case.

Any examination of the data shows clearly that this man absorbed abnormal quantities of lead in significant amounts, during some considerable period prior to the date of his death. The evidence for this may be found principally in the high concentration of lead in the rib. It is quite unusual to find concentrations in excess of 2 mgs., or at most 3 mgs. per hundred grams of fresh bone in any part of the skeleton of individuals who have not been exposed to unusual quantities of lead. The lead concentration here, therefore, is at least twice that ordinarily found. The lead concentration of the liver is also high but not remarkably so, in relation to the lead content of the bone. The concentration of lead in the skeleton is often found to be several times that found in this case, among persons with comparable histories of occupational exposure. Therefore, it may be assumed, as the history indicates, that this man lost rather sizable quantities of lead from his body following the termination of his occupational lead exposure in 1947. We have never seen lead intoxication in an individual whose period of lead exposure had been terminated some months before, and therefore I cannot credit this man's lead absorption, as demonstrated at necropsy, as having any important significance in relation to his terminal illness. One might feel, perhaps, that there might be a stronger case for some contributory effect of lead in this illness, if the lead concentrations in the soft tissues had been found to be higher. Actually these concentrations are in keeping with the moderate increase in the lead concentration in the skeleton over that normally found.

Cordially yours,

Robert A. Kenoe, M. D.

RAK ef

Enc.

KE 0005054

N 7441

Material: Tissues

Source: [REDACTED]

History: Worked with lead for 15 years. Employed by Eagle-Picher Lead Company until August, 1948. Had lead colic attack 10 years ago. Had abdominal cramps and headache about 6 months ago.

Diagnosis: Malignant hypertension; arteriosclerotic and hypertensive cardiovascular disease; acute pulmonary edema

Submitted: 2-26-48 by the Department of Pathology, Medical College, University of Cincinnati, Hospital #235394, Necropsy #N-48-114

Required: Lead Determinations

<u>Analysis Number</u>	<u>Description</u>	<u>Grams Sample</u>	<u>Mg. Pb Found in Sample</u>	<u>Mg. Pb in 100 g. Sample</u>
48A-1874	Lungs	22.0	0.011	0.05
48A-1875	Heart	52.0	0.023+	0.045
48A-1876	Liver	26.5	0.165	0.66
48A-1877	Spleen	60.5	0.09+	0.15
48A-1878	Kidneys	52.2	0.057	0.09
48A-1879	Stomach	41.0	0.041	0.10
48A-1880	Small Intestine	18.5	0.028+	0.16
48A-1881	Prostate	10.0	0.01	0.10
48A-1882	Large Intestine	54.0	0.70	1.30
48A-1883	Adrenals	9.2	0.005	0.055
48A-1884	Muscle	36.3	0.012+	0.035
48A-1885	Rib Bone	26.5	1.85	7.00

Reported: 4-27-48

KF 0005055

N 7441.01

Name

KE 0005056

 - Additional Information
Obtained from hospital history.

This 41-year-old colored male was admitted Feb. 25, 1948 and died the same day. On admission he complained of having had a sudden attack of dizziness followed by vomiting and nausea while at work, 5 days previously. At that time he left work and went home. That evening he had cramping pain over entire abdomen, most marked over lower abdomen. His nausea and cramps continued until the day of admission. The day before admission the patient, while in bed, had a sudden attack of shortness of breath, accompanied by a cough producing frothy blood-stained sputum.

Patient was employed by the Eagle Picher Lead Company for 15 years until August 1947. From then until 5 days before admission he was employed by the Lackner Sign Company. He suffered an attack of abdominal cramps and vomiting 10 years ago which was diagnosed as lead intoxication. He was seen 6 months ago, by Dr. Matuska, because he complained of headache and abdominal cramps.

He was told that his blood pressure was elevated 10 years ago after an Army examination and again in April 1947 when he was examined following an attack of flu.

During his final year he lost 25 lb. in weight (155-130). He had been short of breath on exertion for 6 months. He had had no edema or palpitation. He was orthopneic only during his final week.

The positive findings on Physical Examination were:

T 98.6, P 112, R 30, BP 270/160

well developed, fairly well nourished

dyspneic, orthopneic, coughing

skin warm, moist

mucous membranes pale

discs blurred, arteriolar spasm (?) of retinal vessels

no lead line

venous pressure slightly raised clinically

moist rales over entire chest

heart enlarged to left. Sinus rhythm. A₂ tympanitic and greater than P₂. No murmurs.

Abdomen not tender; no rigidity. Spleen and liver not felt. Small umbilical hernia.

N 7441.03

no edema

neurological normal

The patient's B.U.N. was 200 and his Kahn was negative. No other laboratory work was completed.

The patient's course in hospital lasted about 3 hours. He was given Ammophyllin 7-1/2 gr. i.v. at sometime during this period. No record was made of the result. He was also given morphine gr. 1/6 and atropine gr. 1/150 by hypo. Five minutes after this medication patient died, apparently a cardiac death.

The diagnoses contained in the hospital history are as they appear on the attached information sheet. The two house officers who have written comments in the chart apparently felt that lead intoxication, although its presence could not be ruled out, was contributing little to the present illness.

I quote:

Dr. Seebohm: "Plumbism may be playing a part in this picture but I doubt it."

Dr. P. Edlin: "Plumbism likely present but not contributing to P.I."

Dr. Ian MacLachlan