COPY

August 7, 1951

Randolph K. Byers, M. D.
The Children's Medical Center
300 Longwood Avenue
Boston 15, Massachusetts

Re: [redacted]
Lawrence, Massachusetts

Dear Dr. Byers:

A visit was made to the home of [redacted] and Mrs. [redacted] interviewed.

The history given by Mrs. [redacted] was that [redacted], now four years of age, began to chew on the window sills and on his crib at the age of 2½ years. In addition to this chewing habit, he was known to eat dirt and chew on objects when allowed to play outdoors. However, this pica stopped at the age of 3, and for the past year he has not to their knowledge been chewing on any objects. Approximately 4 to 6 weeks ago, he was discovered playing about a hole in the plaster in the hallway leading to their apartment. The [redacted] live in a very old house, and the plaster on the wall crumbles quite easily. Little [redacted] would sit by the wall and pick at the plaster. Although he was discovered playing with the plaster, he was never actually seen eating it.

Mrs. [redacted] gave me the following history of his illness. On July 6, he experienced an upset stomach with nausea, vomiting and diarrhea. The family physician gave him Coca Cola syrup and penicillin, and in two days he was free of all symptoms and apparently well. On the 8th of July while sleeping on his bed, he rolled and fell from the bed, landing on the floor and striking his head. One hour later his mother noted that his left eye began to "cross." On the 9th, the crossing of the left eye became more marked, and he began having visual hallucinations of snakes, grass growing on his bed, and cuts upon his hands. He became very restless and failed to recognize his parents. On the 10th, he was admitted to the Lawrence General Hospital for X-rays of the skull and observation. The X-rays were negative, but his hallucinations and restlessness increased. The diagnosis of lead encephalopathy was contemplated after Mrs. [redacted] told them of the past history of paint chewing. On the 12th, BAL therapy was initiated. Shortly thereafter, the boy began to have convulsions. He had several more convulsions on the 13th, and was transferred to the Massachusetts General Hospital. He was hospitalized one week and returned home on the 20th.

At the present time [redacted] is, to quote his mother, "like a baby again." His language is incoherent, he must be fed, and he has lost control of his toilet habits. He has just begun to recognize his parents.

Samples of the window-sill paint and wall plaster of the hall were collected and sent to Miss Taylor at the Children's Hospital. The crib was stained and lacquered, so a sample from the surface was not collected. The water supply is carried by brass pipes, and no lead pipes could be seen.

I found this to be a very interesting case, and if you have the time,

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would appreciate hearing your views on the following points:

(1) If the paint on the wall plaster does not contain lead, and in view of the absence of pica for one year, what would be the logical explanation for this boy having such a high urinary lead?

(2) Do you believe that BAL therapy might have acted as a de-leading agent and precipitated this episode of lead encephalopathy.

(3) What emphasis would you put on his fall, which seemed to initiate his illness?

(4) What were the results of the later urinary lead and coproporphyrin values, and if a spinal tap was performed, was the spinal fluid analyzed for coproporphyrin?

Again let me say this was a most interesting case, and I enjoyed very much working up the field study.

Sincerely yours,

Clarence C. Maloof, M. D.

CCM:BL
CC: Mr. Bowditch