might be metabolized by the liver, leaving the lead residue to be excreted in the bile and thus in the feces. They demonstrated a rather sharp decline in the blood lead levels with this treatment in a series of 15 cases. In another publication, Kety and Letonoff [3] showed that in a series of patients treated with sodium citrate there was a rise in lead excretion when the total of the urinary and the fecal lead was considered. The most marked rise was in the fecal lead. The longest period during which the lead excretion was followed in these cases was 11 days.

Smith,[4] in his discussion of the treatment of lead poisoning, listed sodium citrate as a storage agent, but said that it had only a transitory effect in the one case reported.

With the above considerations in mind, we thought that it would be worth while to report our experience in treating four patients for lead poisoning with sodium citrate as the main component of therapy.

REPORT OF CASES

CASE 1.—J. S., a 24 year old battery factory employee, was admitted to the West Roxbury Veterans Administration Hospital on Oct. 17, 1949 because of abdominal pain of two days' duration. During the three years previous to admission he had been working in the battery

TABLE 1.—*Urinary Lead Concentrations—Case 1, Patient J. S.*

Date	Mg. Lead per Liter of Urine	Mg. Lead per Liter of Urine Adjusted to Sp. Gr. 1.024
11/19/46	0.12	0.12
2/ 5/47	0.12	0.10
3/12/48	0.29	0.26
9/ 7/49	0.31	0.41
10/17/49 *	0.18	0.48

* On this date the patient was hospitalized.

factory as a caster and paster of the battery plates. On this job, he came in contact with molten lead and with lead oxide paste. Over the two years previous to admission he had short bouts of "stomach ache" occurring two times weekly to once monthly and lasting two hours at the most. Two days before admission he had the onset of a dull ache low in the anterior part of the chest. The pain shifted to the abdomen and changed in character to a sharp colicky pain recurring every five to 15 minutes. On the day before admission he had the onset of vomiting without diarrhea and vomited frequently from that time to the time of admission.

Urinary lead concentrations had been determined during his period of service at the battery factory. These are shown in table 1.

Physical examination revealed a rather thin but adequately nourished white man in acute distress with severe abdominal pain. His temperature was 97.4 F.; pulse rate, 56; blood pressure, 118/70. There was no scleral icterus. The teeth were in fair repair, and there was no lead line. Heart and lungs were normal. The abdomen was diffusely tender, without spasm. The tenderness was maximum in the right lower quadrant of the abdomen. There was questionable rebound tenderness directed toward the left upper quadrant of the abdomen. Liver, kidneys and spleen were not felt. Peristaltic sounds were decreased in frequency. There was no muscle weakness, and there were no peripheral sensory changes. The abdominal reflexes were decreased.

Laboratory Data.—The white blood cell count was 9,400, with polymorphonuclear granulocytes 71 per cent. Hemoglobin amounted to 10.5 Gm. per 100 cc. Red blood cells showed

3. Kety, S. S., and Letonoff, T. V.: Treatment of Lead Poisoning with Sodium Citrate, Proc. Soc. Exper. Biol. & Med. **46**:476-477, 1941.

4. Smith, F. L.: Effect of Therapeutic Agents in the Treatment of Lead Poisoning, Pennsylvania Department of Labor and Industry, Harrisburg, Pa., June 1941, 17 pp.