this control period more lead and less coproporphyrin were excreted. After the relationship between the intake of sodium citrate with a low calcium diet and the levels of urinary lead and coproporphyrin with accompanying abdominal pain and headache had been demonstrated, the patient was discharged on citrate therapy. The jaundice and the lead line gradually disappeared. It was necessary to administer ferrous sulfate in doses of 0.26 Gm. three times a day to restore the hemoglobin to an adequate level. The patient returned to work not involving lead exposure and has remained entirely well. The gradual loss of lead from the body by excretion is indicated in the chart of urinary lead levels (chart 3). In this case, whatever the mechanism, the administration of soduim citrate accompanying a low calcium intake appeared to be of real merit.

CASE 4.—C. S., a 45 year old white man of Polish descent, worked as a pyrometer man in a steel plant for the eight years prior to his hospitalization. As part of his job, he supervised baths of molten lead through which steel wire passed for annealing. From the amount of lead in the air (ranging from 0.3 to 2.3 mg. of lead per 10 cubic meters) and the amount of lead in the urine of C. S. and his fellow workers (0.12 to 0.38 mg. of lead per liter) it is certain that

TABLE 3.—*Laboratory Data—Case 4, Patient C. S.*

	Urine	
7/26/49	Color: Clear yellow Reaction: 6.0 Specific gravity: 1.015 Albumin: 0 Sugar: 0 Bile: 0 Rare WBC No casts, no RBC	
7/27/49	0.17 mg. lead per liter 0.40 mg. lead per liter adjusted to sp. gr. 1.024	
	Blood	
7/25/49	RBC: 3.27 million WBC: 11,200 Hgb: 10.5 Gm. Polymorphonuclears: 75% Large lymphocytes: 3% Small lymphocytes: 20% Monocytes: 0 Eosinophils: 0% Basophils: 2% Platelets: adequate RBC hypochromic, anisocytic; 3.7% with basophilic stippling; rare normoblast	
	Chemistry	
7/26/49	Calcium (serum)	9.2 mg. per 100 cc.
	Phosphorus (serum)	3.5 mg. per 100 cc.
	Alkaline phosphatase (serum)	4.3 Bodansky units per 100 cc.
	Total protein (serum)	7.7 Gm. per 100 cc.
	Albumin/globulin ratio	2.0
	Albumin (serum)	5.09 Gm. per 100 cc.
	Globulin (serum)	2.61 Gm. per 100 cc.
	Nonprotein nitrogen (blood)	39 mg. per 100 cc.
7/29/49	Chlorides (as chloride ion, serum or plasma)	106 mEq./L.

this patient absorbed lead during this job exposure. Of the group studied, 15 men doing comparable work, C. S. was the only one who developed symptoms.

His past medical history revealed childhood scarlet fever without known sequelae. In 1944 (five years before admission) C. S. was treated for brucellosis with several courses of sulfadiazine. He had drunk milk drawn from one of his own cows that had aborted. He complained of fever of one month's duration, numbness in the head, generalized bone and joint pain. His Brucella agglutination titer was 1 : 135. After two months he was apparently well.

Two years before admission C. S. began to suffer from pain in the lower right quadrant of the abdomen, extending into the groin. One physician suspected appendicitis, but appendectomy was not performed. The patient was admitted to another hospital after a period of increasing abdominal pain, colicky in character, and vomiting. A diagnosis of lead poisoning was made, and the patient responded well to calcium administered by vein and by mouth. However, he continued to complain of weakness of muscles and did not return to work. Because his abdominal pain returned, plus headache, as well as persistent weakness, he returned 10 months later to this same hospital for eight days, during which he was treated with dimercaprol injection U. S. P. (BAL in oil; 2,3-dimercaptopropanol in oil), with considerable symptomatic relief. When he returned home, he felt improved but did not go back to work at the steel mill.

His family physician gave C. S. calcium by vein and by mouth for four months after he left the hospital. After an interval of reasonably good health, the patient again complained of