had headache or abdominal pain. Many of his persistent subjective complaints probably stemmed from the medicolegal aspects of this case, which had not been settled.

Considerable laboratory work was done, and the data are listed in table 3.

The urinary studies done are shown in chart 4, which gives data on lead, coproporphyrin and therapy. It may be said that C. S. was absorbing, storing and excreting sufficient lead to substantiate the diagnosis of chronic lead intoxication. The correlation of the findings, the complaints and the treatment is not clear.

COMMENT

In three of the cases presented here there was a rather striking relief of symptoms coincident with sodium citrate therapy.

In the case of J. S. (case 1) it seems that renal lead excretion was suppressed by sodium citrate therapy. Perhaps this is the storage effect of sodium citrate suggested by Smith.[4] Perhaps this effect is produced by changes in acid-base pattern as evidenced by the temporary rise in carbon dioxide–combining power which occurred when the sodium citrate therapy was reinstituted. Conclusions may not be drawn from the data at hand, but we think that these suggestions merit further investigation. It is interesting to note that urinary lead increased during the period in which sodium citrate therapy was omitted. Urinary lead also increased after the initial fall occurring at the time therapy was reinstituted.

There is no convincing evidence in the data here presented that the excretion of lead was hastened in cases 1, 2 and 4 by the sodium citrate therapy. Both the prolonged abnormal urinary lead concentration and the slow rise of the hemoglobin to adequate levels we take as evidence that there was persistence of abnormal amounts of lead in these patients.

In case 3 the data presented and the clinical history give reasonable evidence that the sodium citrate therapy was useful in hastening lead excretion and prompt relief of symptoms. Although this man's exposure was relatively short, it was intense, and it is certain that considerable lead was stored in his lungs and absorbed from there into his circulation for some time after his exposure ceased. The prolonging of sodium citrate therapy for a matter of weeks after this man's initial acute lead poisoning was controlled perhaps enabled the remaining lead to be handled in a manner to prevent exacerbation of symptoms. Since S. O. was not allowed to return to work involving lead exposure, his lead intoxication might well have followed a benign clinical course without medication.

The problem of C. S. (case 4) is complicated owing to his long-continued intake of lead at his job and at home. As in case 3, this patient, if free from lead exposure, might have excreted comparable amounts of lead without the use of sodium citrate. However, there can be no doubt that the control of his symptoms—and in the case of C. S. this was important—was achieved by sufficiently large oral doses of sodium citrate.

The use of dimercaprol (BAL) in the case of C. S. is hard to evaluate, because the patient continued to take in small doses of lead from his drinking water supply after leaving the hospital. We were led to review the available literature and present the following summarizing remarks.

Although dimercaprol is most effective in the treatment of poisoning caused by certain heavy metals, such as arsenic, gold and antimony, there is, with one excep-