tion, no convincing evidence that it is of value in the treatment of lead poisoning.[6] Telfer[7] reported one case of lead poisoning treated with BAL. Although the worker was excreting normal amounts of lead after receiving intravenous injections of calcium gluconate for three days, the urinary lead rose to high levels after a series of dimercaprol injections.

We have been much interested in the suggestion that urinary coproporphyrins be used as an index of lead intoxication. Increased coproporphyrinuria was found during acute illness in all four of the cases presented in this paper and well before the acute episode in at least two of the cases. In all cases the urinary lead and coproporphyrin values showed relatively good correlation. It should also be mentioned that in all cases the sign of coproporphyrinuria was seen much earlier than the stippling of the red blood cells. In the case of S. O. there was a great increase in coproporphyrin excretion, accompanied by an absence of stippled red blood cells in the peripheral blood. On the fourth hospital day, stippled red blood cells were found in the bone marrow smear but not in the peripheral blood. On the sixth hospital day, stippled red blood cells appeared for the first time in the peripheral blood. From then on there was a gradual increase in the stippled cell count, while both urinary lead and urinary coproporphyrin values showed a downward trend. In the cases of C. A. and J. S. the coproporphyrin excretions were elevated well before the symptoms developed, and during this time stippled cell counts were normal. In the case of C. S., who had lead poisoning over a long period, both the excretion of coproporphyrin and the stippling of red cells were increased.

In postulating the mechanism of this increased coproporphyrinuria in plumbism, it appears from studying these four cases that it is entirely possible for the mechanism to vary in different cases. It is believed at this time that in those acute poisonings which develop over the relatively short period of several months and which are accompanied by anemia and increased icterus, the mechanism is due to the destruction of the red blood cells, which leads to a release of protoporphyrin, which is then converted into coproporphyrin in the liver and excreted as such in the urine.[8] Aub[1] and his associates have already described the mechanism of

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