acute rheumatism, it should be administered in very large doses. This, I believe, is in keeping with clinical experience. Furthermore, since the source of glycocoll is probably the metabolism of proteins, which is a limited process, the more rapidly the salicylic is administered the greater the amount of uncombined salicylic acid present in the blood. This suggests that in the treatment of acute rheumatism one should give large doses at short intervals, at least for a time.

Conclusions.

I. Salicyluric acid is an inactive substance.

II. The feeding of gelatine does not increase the proportion of salicyluric acid in urine. This has an important bearing on the theory that some amino acids are disseminated in the intestinal mucosa.

III. Glycocoll, given hypodermically, is an antidote to poisoning by salicylic acid.

IV. The maximum production of salicyluric acid in a man of average weight is obtained with the exhibition of somewhat less than 60 grains of salicylic acid a day. Beyond this amount the larger the dose of salicylic acid the more free salicylic acid in urine.

Glycocoll as a Defensive Agent Against Poisoning by Cholic Acid.

Cholic acid is present in both bile acids—glycocholic and taurocholic. In glycocholic it is combined with glycocoll, and in taurocholic with taurin. The union is, no doubt, similar to that of glycocoll with benzoic acid in hippuric acid, and of glycocoll with salicylic in salicyluric acid.

Glycocoll + cholic acid = glycocholic acid + water.
Taurin + " " = taurocholic " + "

In the light of the present paper the bile acids may therefore be looked upon as conjugated cholic acids, and following the general rule one should expect to find the conjugated less poisonous than the free.

I have attempted to obtain data on this subject in the following experiments:

Experiment 1.—Rabbit weighing 1.7 kilos was given subcutaneously 1 gram of cholic acid neutralized with caustic soda and dissolved in water. The rabbit recovered.