

cats in histamin shock. Histamin also causes agglutination of human, avian, guinea pig, dog and cat red blood corpuscles "in vitro," which is consistent with the formation of thrombi and emboli "in vivo."

The following agents, caramel (50 per cent.), cane sugar, casein, calcium lactate, lutein extract, horse serum, colloidal gold (sensitive), colloidal gold (protected) and colloidal sulphur (saturated), did not cause anaphylactoid symptoms. Pulmonary thrombi were not demonstrable after cane sugar, sodium chloride, casein, calcium lactate, horse serum and colloidal gold (sensitive).

Cane sugar and sodium chloride (hypertonic solutions) have been advocated and used for the prevention and treatment of anaphylactoid symptoms. The rationale of this is not understood, but is alleged to be concerned with alterations in the physical-chemical properties of the colloids of the blood. Accordingly, attempts were made to treat anaphylactoid symptoms produced by kaolin, histamin, acacia, and beef serum by preliminary injections of 50 per cent. cane sugar and of 10 per cent. sodium chloride. However, the results obtained were uniformly negative as to prevention of symptoms and thrombi.

Intraperitoneal injections were made with the following agents, allowing at least one hour after administration for development of symptoms; agar sol-gel, toxified agar, horse serum, beef serum, lung extract, histamin, colloidal gold (sensitive), colloidal sulphur (saturated), Congo red, glacial acetic acid and copper sulphate. With the exception of copper sulphate, the results were uniformly negative. The intraperitoneal injection of copper sulphate in the same dosage as was used intravenously, caused anaphylactoid symptoms and death in 40 minutes. Pulmonary thrombi were definitely present after intraperitoneal injection of histamin only.

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Urinary excretion of salicyl after the administration of salicylate and salicyl esters.

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Using sodium salicylate as a control, the excretion of methyl

salicylate, acetylsalicylic acid and salicylosalicylic acid (diplosal) was compared in each of three human individuals. Small doses of 0.5 gm. to 1 gm. were used. The mean total excretion of sodium salicylate was 80 per cent., which agrees with the results obtained with very large doses (12 gms.) previously reported. On the other hand, the mean total excretion of salicyl after the administration of the salicyl esters was distinctly less, namely, 60 per cent. Special treatment of the urines for detection and estimation of the undecomposed esters gave contradictory results. Unchanged esters appeared to be present to a small extent only in some urines, absent in other urines. Ethereal extractives of the urines after the administration of methyl salicylate possessed a fruity odor, indicating the presence of the unchanged ester. Larger doses of the esters may give more conclusive evidences along this line. Since all urines were collected until excretion of salicyl was completed, the salicyl unaccounted for appears to have been destroyed, and this is confirmative of previous results with large doses of sodium salicylate. The mean duration of excretion of sodium salicylate and the esters was practically the same, namely, about 48 hours; only the methyl salicylate showing a tendency to somewhat more prolonged excretion (55 hours).

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The effect of the administration of salvarsan in combination with various colloid substances on its toxicity.

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In some unpublished articles it has been shown that the ill effects following salvarsan administration may be divided into two types; first, an immediate reaction from which the animal dies suddenly in a convulsive seizure as a result of embolism of its agglutinated red cells, and second, a late death occurring in from two days to as many weeks, which is the result of degenerative lesions in the kidneys and liver. As the former has been