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The destruction of strophanthin in the animal organism.

By **ROBERT A. HATCHER** and **HAROLD C. BAILEY.**

[*From the Laboratory of Pharmacology of Cornell University Medical College.*]

Strophanthin is toxic in much smaller doses when injected into a vein or subcutaneously than when given by the mouth.

Dilute solutions injected slowly or at intervals are approximately as toxic as stronger solutions injected at once.

Strophanthin is not readily destroyed by the liver; no difference could be observed in the amounts required to cause death from slow injections of dilute solutions into the superior mesenteric, and into the femoral vein.

Peptic and activated pancreatic digests destroy only small amounts of the poison, not enough to account for more than a small part of the difference in toxicity by the mouth. Bile exerts little influence.

Ten times that amount of strophanthin which is fatal by the vein may disappear from the dog's intestine in an hour and a half without proving fatal, the portion remaining in the lumen of the intestine retains its toxicity, but death may result promptly (in thirty-eight minutes) from the introduction into the duodenum of an amount but slightly exceeding that necessary to cause death after oral administration.

The poison may be destroyed in part during its passage through the walls of the intestine, but since part of even a very small dose is absorbed unchanged, it seems fair to conclude that certain cells alone are concerned in the destruction while others permit it to pass through.

The cat and dog react to similar doses, the rabbit will stand somewhat larger doses administered subcutaneously and vastly larger doses by the mouth; this suggests that histological differences may furnish a clue to the cells concerned in the destruction.

One milligram of an active strophanthin given by mouth, per kg. of cat, has proved fatal, while a man has been given about as much (one hundred and fifty milligrams total) on two days.

The toxicity of strophanthin by the mouth can be varied at will to a considerable degree for the cat.

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On the nature of the socalled glycogenolytic fibers in the greater splanchnic nerves.

By **J. J. R. MACLEOD.**

[*From the Physiological Laboratory, Western Reserve University.*]

The hyperglycæmia which invariably results from interference with pulmonic ventilation, produced either by constriction of the air passages or by inadequacy of the respiratory movements, has led to the question whether or not the hyperglycæmia following stimulation of the great splanchnic nerves may not also be due to an asphyxial condition induced locally in the liver. Such a local asphyxia of the hepatic lobule might be the result of diminished blood supply caused by constriction of the blood vessels in Glisson's capsule, or by the diminution of portal blood supply following constriction of the splanchnic vessels.

The question, therefore, presents itself as to whether stimulation of the great splanchnic nerve causes hyperglycogenolysis because secretory nerve fibers influencing the production or activity of the glycogenolytic ferment in the liver are contained therein, or because of a local disturbance in the blood supply of the liver following stimulation of vaso-constrictor fibers.

In the following communication a preliminary report is offered of several experiments devised to throw some light on these problems.

1. All the tissues running to the hilus of the liver except the portal vein were cut between peripherally and centrally placed ligatures. As much as possible of the outer coat of the portal vein was also removed. By these three operations all the hepatic nerves running from the cœliac plexus to the liver were severed. Stimulation of the great splanchnic nerve was found to cause no increase in the sugar content of the blood although the usual marked vaso-constriction of the splanchnic vessels occurred.

2. Clamping the portal vein for periods of about a minute at