5804

The Toxicity of the Products of Pancreatic and Gastric Digestion.*

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The most widely accepted theory regarding the etiology and pathogenesis of acute pancreatitis may be briefly stated as follows: A premature or intra-pancreatic activation of the trypsinogen of pancreatic juice is caused by the entrance of bile or duodenal juice into the pancreatic ducts or as a result of trauma or infection. It is then further assumed that the active trypsin so produced digests away the gland, that the end products of this tryptic digestion of pancreas are toxic, that they are absorbed into the blood stream and occasion a fatal toxemia. Ellis and Dragstedt[1] found that the uncontaminated liver of normal dogs contains bacteria in a very high percentage of cases and that the toxemia resulting from liver autolysis *in vivo* is dependent upon the activity of these bacteria in the presence of necrotic liver. By a similar technique to be reported in detail elsewhere we have found that the normal uncontaminated pancreas of healthy adult dogs also regularly contains bacteria and that the fatal toxemia resulting from the intraperitoneal autolysis of pancreas is likewise in large measure dependent upon the activity of these bacteria in the presence of necrotic pancreas. In this connection it has occurred to us that a reinvestigation of the toxicity of gastric and pancreatic juice and of the end products of the disintegration of body tissue produced by these juices in the strict absence of bacterial activity would be desirable. The present experiments were designed to determine the toxicity of the end products of gastric and pancreatic digestion.

Gastric juice was obtained from a Pavlow pouch and fresh active pancreatic juice from a specially devised fistula, previously described.[2] These were sterilized by passage through a Berkefeld filter (N). Carefully weighed samples of dog pancreas, liver, and muscle were then autoclaved at 15 lbs. for 15 minutes. Proportionate amounts of the sterile juices were added to each and digestion allowed to proceed at 37°C. for 66 hours. A control experi-

* This work has been conducted under a grant from the Douglas Smith Foundation for Medical Research of the University of Chicago.

1 Ellis, J. C., and Dragstedt, L. R., *Arch. Surg.*, 1930, **20**, 8.

2 Dragstedt, L. R., Montgomery, M. L., and Ellis, J. C., PROC. SOC. EXP. BIOL. AND MED., 1930, **28**, 109.

ment was carried out with fresh uncontaminated, but not autoclaved, tissues with equivalent amounts of the digestive juices and one series with Ringer's solution. All controls showed profuse bacterial growth while the others remained sterile. The toxicity of equivalent portions of the digestion products in each case was determined by intraperitoneal injection in mice. The results show that the products of the aseptic gastric and pancreatic digestion of such organs as pancreas, liver, and muscle are relatively non-toxic as compared with similar products complicated by the growth and activity of the bacteria commonly found in these tissues. These *in vitro* experiments confirm the previous observations with respect to the *in vivo* autolysis of liver and pancreas that the toxemia in each case is dependent more upon the products of bacterial activity than upon the substances of simple, non-bacterial digestion or autolysis.

5805

Effect of Celiac Ganglionectomy on Sugar Tolerance of Dogs.

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Since the literature indicates that the output of insulin is in part regulated by the vagus and sympathetic nerves,[1] that a sympathectomized cat is hypersensitive to insulin,[2] and that ligature of portions of the pancreas increases the sugar tolerance of dogs[3] (de Takats) it was considered important to determine the effect of removal of the celiac ganglia on the sugar tolerance.

The intravenous sugar tolerance of 3 dogs has been determined by the method of Woodyatt, Sansum and Wilder.[4] These dogs were trained to lie quietly. Blood sugar determinations were made before, during and at intervals up to 4 hours after the injection of sugar. Urine was collected under toluol. After determining the intravenous sugar tolerance, the celiac ganglion was removed and

[1] Britton, *Am. J. Physiol.*, 1925, **74**, 291. LaBarre, *et al., Compt. rend. Soc. Biol. Belge*, 1927, **96**, 193, 421, 708, 710; 1927, **97**, 917, 1048, 1406. Clark, *J. Physiol.*, 1925, **59**, 466; 1926, **61**, 576. Macleod, *Lancet*, 1930, **219**, 512.

[2] Dworkin, *Am. J. Physiol.*, 1931, **98**, 467.

[3] de Takats, *et al., Arch. Surg.*, 1929, **19**, 771; 1930, **20**, 866.

[4] Woodyatt, Sansum and Wilder, *J. Am. Med. Assn.*, 1915, **65**, 2067.