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.

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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.

the tolerance was determined at intervals during a period of from 5 to 10 months.

The intravenous blood sugar tolerance of the 3 dogs before operation was 1.9, 1.8, and 1.8 gm. of glucose per kilo per hour, respectively. The sugar tolerance now 5 to 10 months after removal of the celiac ganglia is 2.8, 3.4, and 3.8 gm., respectively. The sugar tolerance was increased immediately after the operation. The first determination was made at one week. In one dog the tolerance had increased from 1.8 gm. to 3.8 gm. in one week; in a second, the maximum tolerance of 2.8 gm. was not reached until one month; in the third, the tolerance was not determined until 5 months after the operation, when the tolerance was 2.8 gm.

It is to be noted that the normal tolerance values for the dog found by us are higher than those of Woodyatt, Sansum and Wilder. This may be due to the fact that we used a 5% solution of glucose, instead of a 10 to 50% solution, and in the period of one hour injected a volume sufficient to give the desired dosage per kilo per hour. We used Pfanstiehl glucose for intravenous use, and our dogs were not excited during the injection.

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The Titration Curve of Gastric Mucin.

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Fogelson[1] has prepared a "gastric mucin" from the gastric mucosa of hogs which has a relatively high combining power for free hydrochloric acid and which has proved to be effective in relieving the symptoms of peptic ulcer when administered in adequate doses. Kim and Ivy[2] have found that the administration of "gastric mucin" prevents the development of duodenal ulcers in biliary fistula dogs. Since Fogelson did not claim that his "gastric mucin" was a pure product, since with his method of preparation, it is very likely that peptone and other products of incomplete protein digestion are present which may account for some of the acid-combining

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[1] Fogelson, S. J., *J. Am. Med. Assn.*, 1931, **96**, 673.

[2] Kim, M. S., and Ivy, A. C., *J. Am. Med. Assn.*, in press.