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Perfusion studies on pancreas and liver.

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Because of the rich anastomotic supply to the pancreas, duodenum and liver, an anatomical study was undertaken with the view of determining what arteries go directly to each of these organs, so that a more exact knowledge of the direction taken by any perfused fluids could be established. A modification of Gross's^{1, 2, 3} method of injecting the coronary vessels of the heart was used, and the tissues cleared by the Spalteholz method. A description of the method of preparation of these specimens will follow in a later paper. Proper temperature and pressure controls were used in order to approximate normal conditions in living organism.

X-ray photographs reveal that the hepatic artery coming from the coeliac axis gives off the right gastric and the large superior pancreatico-duodenal. This latter artery supplies the neck and body of the pancreas and the first portion of the duodenum. Two small branch arteries, a continuation of the hepatic, ascend to furnish the arterial supply to the liver. The inferior pancreatico-duodenal artery arises from the superior mesenteric artery, and supplies the head of the pancreas and the second portion of the duodenum. A rich anastomosis can be seen in both the duodenum and pancreas between the superior and inferior pancreatico-duodenal arteries. The pancreas is also visualized by this method. The body and tail of the pancreas, on dissection of the specimens, are seen to be supplied by several small branches arising from the splenic artery. The veins draining the pancreas all enter the portal system. One may notice, then, that the arteries furnishing the most practical approach for direct perfusion of the pancreas are the superior and inferior pancreatico-duodenal arteries.

¹ Gross, L., *The Blood Supply to the Heart*, Paul Hoeber, New York, 1921, pp. 5-8.

² Benzley, R. R., *Am. J. Anat.*, 1911, xii, 297.

³ Page, I. H., *J. Lab. and Clin. Med.*, 1923, ix, 194.

Perfusion experiments of these arteries were then undertaken to determine their effect upon the blood sugar and sugar excretion in the urine. The animals used were dogs. The anesthetic was amytal in doses of 0.6 cc. of a 10 per cent solution per kilo of body weight. The Shaffer-Hartmann method of blood sugar determination was employed, and the Benedict test for the urine. Ten cc. of normal saline at 40° C. were used in all the perfusion experiments.

Epstein,⁴ in an extensive series of experiments, has found that when he perfused the arterial blood supply to the pancreas, hyperglycemia and glycosuria constantly occurred. He interpreted these experimental results as indicating that the process of perfusion in some way caused trypsin formed in the pancreas to inactivate insulin, with consequent production of the diabetic condition. He concluded that this reaction between trypsin and insulin constituted an important etiological factor in the production of diabetes.

Repetition of these experiments of Epstein by the perfusion of saline into the hepatic artery of the dog confirmed his original observations; but when the two terminal hepatic arteries were ligated the pancreas could be perfused without causing either hyperglycemia or glycosuria. On the contrary, when the superior pancreatico-duodenal artery was ligated, with the hepatic branches patent, perfusion resulted in both hyperglycemia and glycosuria. This clearly indicates that the effect is upon the liver and not upon the pancreas.

Furthermore, perfusion of the inferior pancreatico-duodenal artery, which has no relation to the arterial supply of the liver, has no effect whatever upon the blood sugar, and produces no glycosuria.

If saline be introduced directly into the portal vein there appears inconstantly a delayed (one-half to one hour) excretion of traces of sugar in the urine, with no or only a very slight elevation of blood sugar. It appears from this that glycogenolysis is more readily induced through the hepatic artery than through the portal blood stream.

⁴ Epstein, A. A., and Rosenthal, N., E. H. Maechling and V. de Beck, *Am. J. Physiol.*, 1924, lxx, 225; *Ibid.*, 1925, lxxi, 316.