cocci and agar is similar to the hypersensitiveness in patients with acute rheumatic fever, and the fact that hypersensitive animals may be made nonsensitive and the further progress of the lesions retarded by giving intravenous injections of streptococci led to the inquiry whether patients with acute rheumatic fever, who are known to be hypersensitive to streptococci, might be made nonsensitive, the further progress of the lesions retarded, and the development of new lesions prevented by giving intravenous injections of streptococci.

Eight patients having acute rheumatic fever were injected intravenously from 4 to 9 times with killed streptococci. The initial doses contained from 25 to 100 million organisms and the final doses one billion each. The agglutinating titers of the sera before any injections were made were from 1:0 to 1:400. The final titers following the injections ranged from 1:6400 to 1:100,000.

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Effect of Gastrostomy Feedings on Occurrence of Experimental Acute Pancreatic Necrosis After Ampullary Obstruction.

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Gall bladder disease is a factor common to the majority of cases of acute pancreatic necrosis. The mechanism by which gall bladder disease predisposes to pancreatic necrosis has not been established. In this study we have tried to evaluate the factor of reflux through the agency of mechanical obstruction at the ampulla in the cat. The terminal portion of the common bile duct embedded in the wall of the duodenum was exposed and a ligature placed creating a common channel of the bile and pancreatic ducts.

The criterion of pancreatic necrosis used in this work was the actual histological demonstration of necrosis in the pancreas. Postmortem autolysis and fat necrosis were differentiated from true pancreatic necrosis by the absence of microscopic cellular reaction in these areas.

In a group of 7 cats, 1 developed pancreatic necrosis following simple establishment of ampullary obstruction. In another group air was injected into the gall bladder at the time the obstruction was established until the gall bladder became so distended that its contents were spontaneously evacuated. Through the agency of the

block at the ampulla regurgitation occurred into the pancreatic duct. In a group of 13 cats, 8 developed pancreatic necrosis. The pressure in the common bile duct occasioned by the emptying of the gall bladder was measured in 5 cats and was found to vary between 190 mm. and 240 mm. water.

In another group ampullary obstruction was established and gastrostomy was also done, after which the cats were given frequent feedings of olive oil, cream, and bile salts through the gastrostomy tube. Of 31 cats pancreatic necrosis was observed in 15.

To another group alcohol was fed through the gastrostomy tube. Of 9 cats, 2 developed pancreatic necrosis. In another group frequent feedings of glucose were similarly given. Pancreatic necrosis was observed 2 times in 9 cats. The incidence of pancreatic necrosis in the latter 2 groups is definitely less than in the group given a fat diet.

Of 6 cats treated by gastrostomy feedings of cream, olive oil, and bile salts, but given subcutaneous injections of pilocarpine hydrochloride, 3 developed pancreatic necrosis.

In another group active pancreatic extract was injected into the gall bladder at operation. Pancreatic necrosis was observed once in 3 cats.

In 19 cats in which infections had been established in the gall bladder, pancreatic necrosis occurred in 5. It is to be noted that the incidence of pancreatic is but little greater in this group than in simple ampullary ligation.

In an effort to demonstrate reflux in the pancreatic duct by x-ray, lipoidol was injected into the gall bladder in 6 cats, 1 of these developed pancreatic necrosis. In none of these was x-ray evidence of reflux into the pancreatic duct present.

The more frequent occurrence of pancreatic necrosis in the group in which fatty meals were administered by which emptying of the gall bladder was stimulated, indicates the significance of the emptying of the gall bladder for the development of pancreatic necrosis through the reflux mechanism. It is well known that pancreatic necrosis not infrequently follows large meals in the human.

Mann and Giordano[1] have shown that emptying of the gall bladder causes but little rise in intraductal pressure. However, in the presence of mechanical or functional ampullary obstruction this seems to be a significant factor.

[1] Mann, F. C., and Giordano, A. S., *Arch. Surg.*, 1923, vi, 1.