78.1 mg, and 95.2, respectively. Three weeks after administration of glycine to these animals a mean fasting blood sugar of 101 mg was found. The same dose of insulin according to weight was then given and blood sugar determinations were made hourly. At the end of one hour the blood sugar was 39 mg. At the second, third, fourth, and fifth hours it was 37.4 mg, 48.5 mg, 63.5 mg, and 78.7 mg, respectively. This gives in rough figures a decrease of approximately 10 mg after the first hour and extending to the fifth hour. In the case of animal No. 6 (Fig. 2) the effect was somewhat more striking. Prior to the administration of glycine in this animal the fasting blood sugar was 110 mg. After administration of insulin the lowest blood sugar, 48 mg, as shown in the accompanying graph, occurred at the end of one hour. It rose gradually to 102 mg 5 hours after insulin. When retested 3 weeks after the administration of glycine, a fasting blood sugar of 113 mg was found. Following administration of insulin the lowest blood sugar level was 22 mg occurring 2 hours after insulin and rising to 89 mg 5 hours after insulin.

There was considerable variation in the response of different animals. Rabbit No. 9, for some reason not apparent had a consistently higher blood sugar following the administration of glycine than prior to it. If this animal is excluded, results are considerably more impressive.

Why a normally occurring constituent of the body should have this effect or why it should have any effect when given in these relatively small amounts is not apparent. There are instances in which the body controlling mechanism may be disrupted by the administration of excessive amounts of normally occurring substances.

Summary. 1. The effect of parenteral administration of glycine in rabbits on the effect of insulin is reported. 2. Parenteral glycine under certain conditions renders rabbits more sensitive to insulin.

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Early Histological Changes Following Obstruction of Pancreatic Ducts in Dogs: Correlation with Serum Amylase. (18741)

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Clinical diagnosis of pancreatitis depends largely upon detection of elevated serum amylase levels. The relationship between these and the extent or duration of early pancreatic changes remains relatively obscure. After ligation of the pancreatic ducts in experimental animals, previous investigators have noted a transient rise in serum amylase reaching a maximum during the subsequent few days(1). In cats fasting 48 hours, pancreatic ligation produced no histologic evidence of inflammatory reaction in the pancreas unless stimulants such as secretin or parasympathomimetic drugs were given(2). The early microscopic changes resulting from duct ligation plus stimulation were widening of the septa and presence of inflammatory cells in these spaces. More severe reactions showed inflammatory cells within the lobules and destruction of acinar cells. In rabbits fasted 48 hours before the experiment, vacuolation of some acinar cells and dilatation of a few ducts were present 24 hours after ligation. After 11

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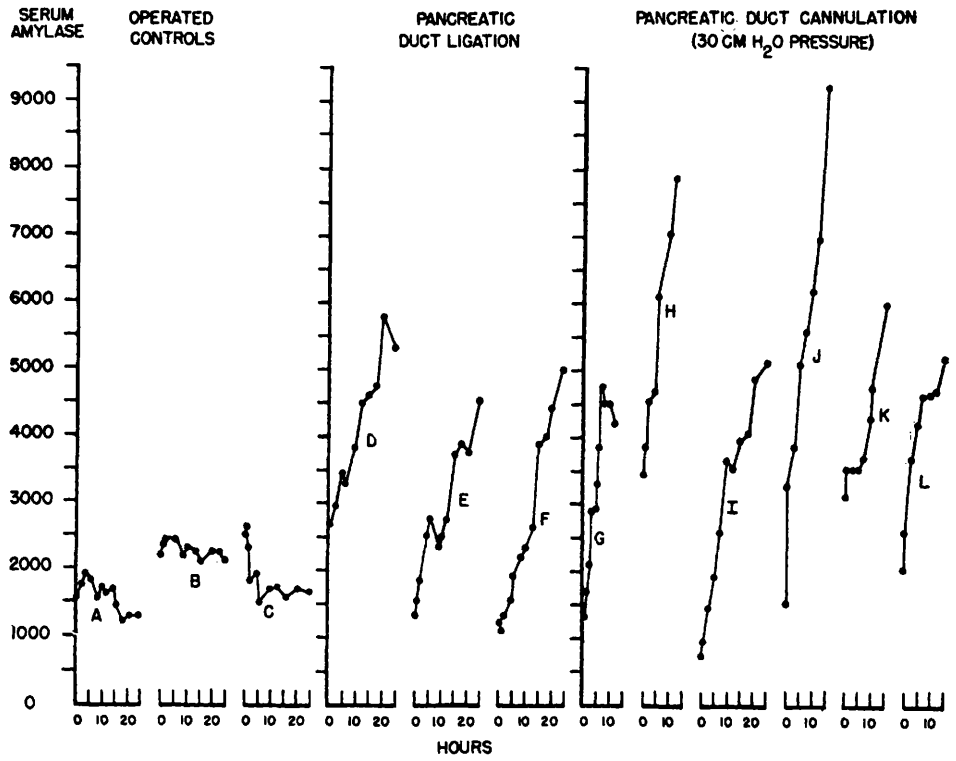

FIG. 1. Changes of serum amylase following pancreatic duct ligation, pancreatic duct cannulation with 30 cm H_2O pressure, and in operated control dogs.

days most of the acinar cells had degenerated and many ducts had dilated(3).

The present study was undertaken to provide a closer correlation between histological findings and serum amylase levels during the first 24 hours following ligation of the pancreatic ducts. Because the pressure within the ligated ducts was not known, the duct systems of other dogs were cannulated and subjected to a definite pressure of 30 cm H_2O by means of a reservoir of sterile saline. This pressure had been found to approximate the maximum which could be produced physiologically by means of repeated injections of secretin. On other dogs, serving as operated controls, the pancreatic ducts were exposed but not ligated or cannulated.

Experimental details. The dogs were last fed approximately 20 hours prior to the experiment. They were kept under anesthesia by occasional intravenous injections of sodium pentobarbital for the entire experimental period of 24 hours (or less). Serum amylase was determined by a modification of the Somogyi method with use of additional NaCl, a phosphate buffer to maintain a pH of 6.8(4), copper sulfate-sodium tungstate deproteinization and the alkaline copper sugar reagent of Somogyi(5). Results were expressed as mg glucose equivalent released per 100 cc serum in 30 minutes.

At the end of the experimental periods, tissue blocks were cut from the pancreas *in situ*, fixed in formalin and stained with hematoxylin and eosin.

Results. The changes in serum amylase activity are shown in Fig. 1. No significant alteration of serum amylase concentration occurred in the operated control dogs A, B, C. No edema of the pancreas was noted grossly. Histologically, there appeared to be no spreading of the interlobular septa. A few inflam-

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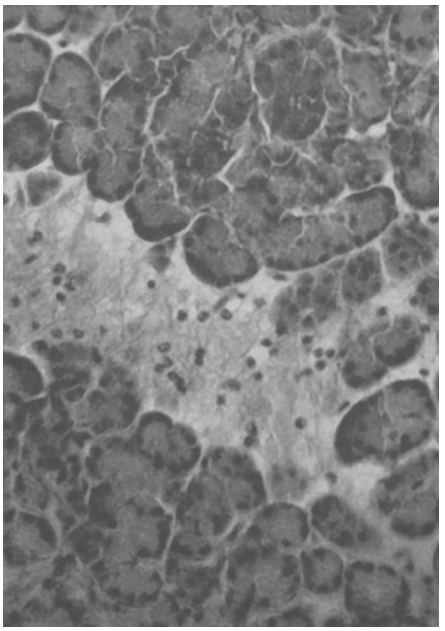

FIG. 2. Inflammatory cells in distended interlobular septum of pancreas, 24 hr following ligation of pancreatic ducts. (Dog D, Fig. 1). ×280.

matory cells were in the capsule.

In the dogs D, E, F with ligation of both pancreatic ducts, a gradual rise of serum amylase occurred throughout the 24-hour period of observation. By the end of that period a value 2.8 times the initial value was reached in the average of the 3 dogs. By 5 to 7 hours the pancreas appeared to be definitely edematous. At 24 hours the histological studies showed spreading of the interlobular septa and inflammatory cells in these spaces as illustrated in Fig. 2. Edema induced spreading also of the capsule. The leukocytic infiltration of the capsule was more marked than in the operated control dogs. No leukocytes appeared among the acini. No noteworthy degeneration of acinar cells, separation of acini or dilatation within acini appeared. The acinar cells were well stocked with zymogen granules. The slightness of the histological alteration suggests that absorption of enzyme-bearing fluid into the blood stream without actual structural damage may account for the elevation of serum amylase.

In dogs G-L, in which the pancreatic duct system was placed under pressure of 30 cm water, the rise in serum amylase was more rapid. Dogs J, K, L died at approximately 15 hours. In Dog I the rise of serum amylase was slower and the dog survived for the full 24 hours. Dogs G and H were killed at 12 hours. Shock was marked in all for many hours. The average rise of serum amylase in 12 hours in the 5 dogs was to a value 2.7 times the initial value. Thus the rise in serum amylase was approximately twice as rapid in the dogs with artificial pressure as it was in those where an indefinite amount of pressure developed following simple ligation of the ducts. In the dogs with artificial pressure, the pancreatic edema appeared earlier and more markedly than in the ligated dogs. Fat necrosis was extremely slight, and that mainly in areas traumatized by dissection or rubbing with respiratory movements. Some fluid exuded from the surface of the pancreas in most instances and accumulated in the peritoneal cavity. No definite point of leakage could be detected. Microscopically, there was more intense infiltration of inflammatory cells into the interlobular spaces. The most consistent and distinctive feature of the histological change in this group of dogs was the presence of inflammatory cells among the acini (Fig. 3). In some instances there were foci of infiltration with total disappearance of acinar cells. Some of these foci were in the vicinity of small ducts and in the 2 instances, L and K, there were leukocytes in the ducts. Vacuo-

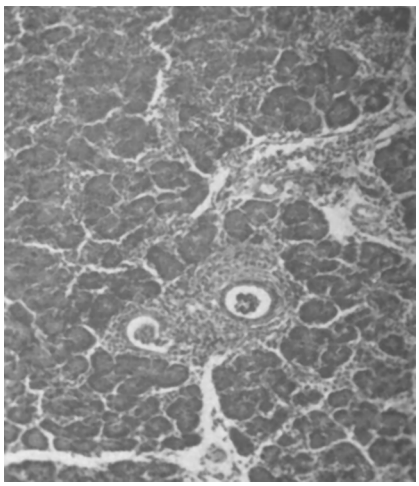

FIG. 3. Inflammatory cells in septa, among acini, and in pancreatic ducts after 15 hr of intraductal pressure of 30 cm H_2O. (Dog K, Fig. 1). ×140.

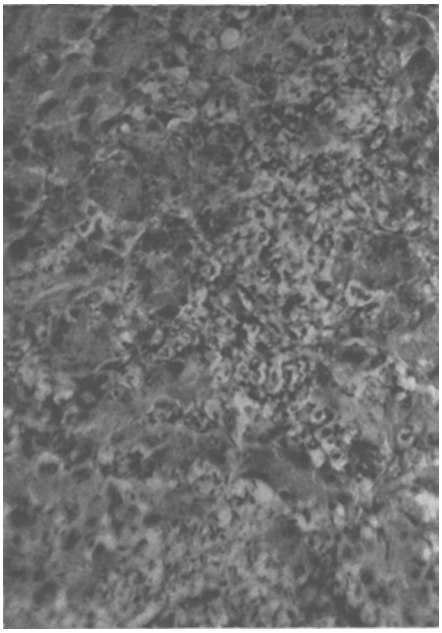

FIG. 4. Degeneration of acinar cells after 15 hr of intraductal pressure of 30 cm H_2O. (Dog J, Fig. 1). ×280.

lation of acinar cytoplasm was limited largely to the group with artificial pressure. In dog J, this progressed to an extreme degree. In small areas the staining properties of the acinar cells were completely abnormal but little or no inflammatory reaction was observed, in association with this change (Fig. 4). In most other areas, however, the cells appeared normal and well stocked with zymogen granules. In most of the dogs subjected to definite pressure, erythrocytes appeared in the interlobular spaces and in one instance infiltrated an acinar area. There was thus a pancreatitis which might be described as hemorrhagic as well as purulent or necrotic.

Summary. Both pancreatic ducts were ligated in 3 dogs. Over a period of 24 hours a gradual rise of serum amylase and a moderate edema with a slight leukocytic infiltration of the interlobular connective tissue of the pancreas occurred. The pancreatic duct systems of five dogs were subjected to a pressure of 30 cm water. Serum amylase rose twice as fast as in the dogs with simple ligation. Histologically, the principal changes in the pancreas were a more intense leukocytic infiltration, including acinar areas, and degeneration of some acinar cells.

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Electron Microscope Study of Anaplasmosis in Bovine Red Blood Cells. (18742)

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In a recent work on the virus host relationships of the foot and mouth disease virus with erythrocytes a technic was developed by which regular blood smears of the infected red cell membranes can be observed under the electron microscope (Epstein, Fonseca, and De Robertis(1). This technic was found to be useful for the study of parasites of erythrocytes. Some preliminary observations on piroplasmosis and especially on anaplasmosis will be reported here.

The *Anaplasma marginalis* Theiler, 1909 is well suited for electron microscope research since it is only about 0.4 to 1 μ in diameter. It is generally described as a round marginal body simply constituted of a single mass of chromatin (Fig. 1a), with no cytoplasm and multiplying by binary division(2). Recently there has been a tendency to consider the

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