is stated that dogs with totally ligated hepatic arteries usually survived when given massive doses of penicillin, whereas the control animals usually died. These findings also emphasize the importance of bacteria in causing death due to liver autolysis in dogs.

To our knowledge the relatively benign effect of placing segments of human liver in the peritoneal cavity of dogs has not been previously described. The fact that devitalized human liver is less toxic than devitalized dog liver may be due to the absence in the human liver of the anaerobic, spore-forming, gas-producing organism found normally in dogs' liver. No such organism was found in the human liver specimens used in this experiment, though *Aerobacter aerogenes* and *staphylococci* were found. Shann and Fradkin(13) describe a patient who recovered following sequestration of a large fragment of liver and review the literature describing several other cases of recovery following liver sequestration secondary to biliary tract surgery or abdominal trauma. These clinical findings seem to be in agreement with our observation as to the relative non-toxicity of the devitalized human liver as compared with dog liver.

Conclusions. 1. Treatment with penicillin in oil reduces the fatality of peritonitis due to canine liver autolysis in dogs. 2. Autolysis of devitalized human liver in dogs is less fatal than autolysis of canine liver in dogs.

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Hormonal Mechanisms in Nervous Mechanism of Gastric Acid Secretion in Humans.* (17735)

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It has been shown that the so-called chemically induced secretion of gastric acid occurring in response to the ingestion of food depends upon the elaboration of an histamine-like hormone, which has been named gastrin(1-8). Moreover, the pyloric end of the stomach has been identified as the source of this hormone(1-8), although hydrochloric acid secreting parietal cells are known to be generally distributed throughout the whole stomach(9). Recently several workers have adduced evidence that the secretion of gastric acid in response to vagal impulses also requires the mediation of histamine (9-12) or the histamine-like hormone gastrin(13-15). As a by-

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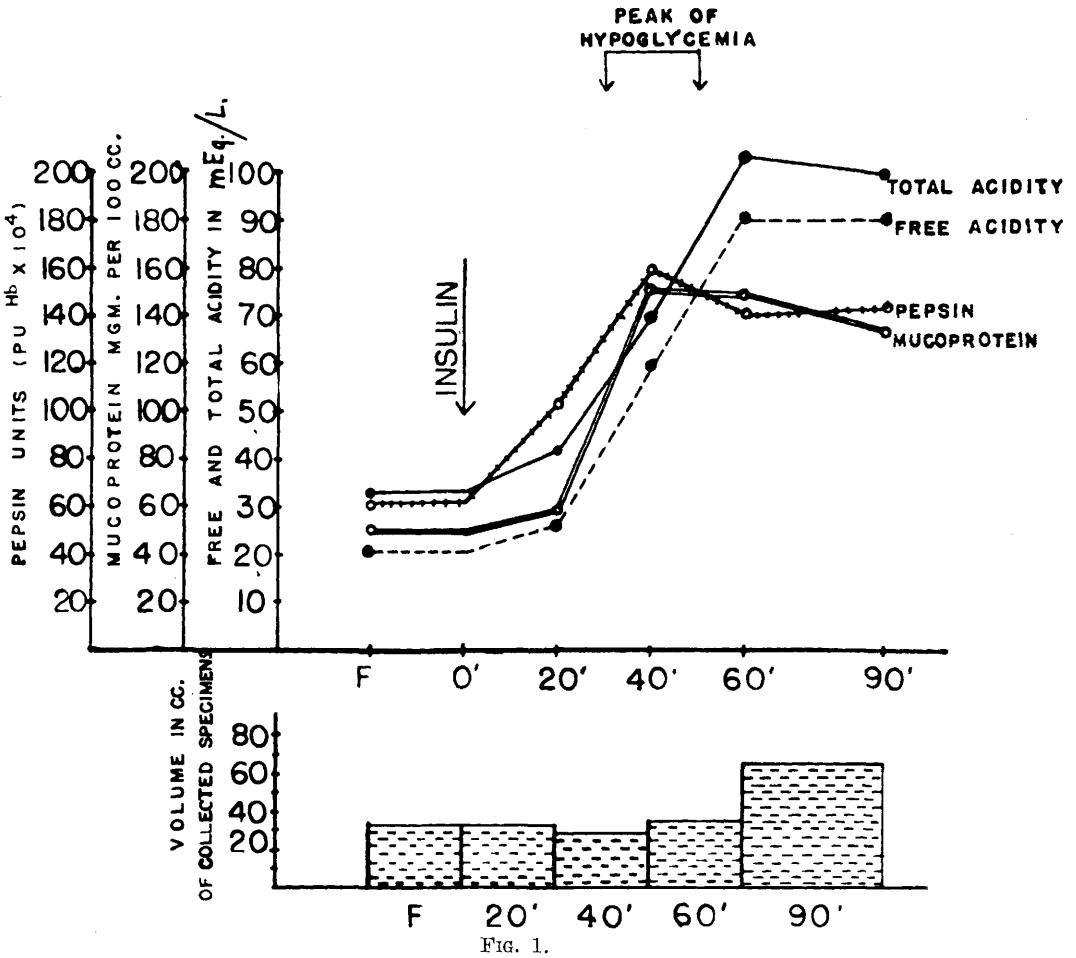

FIG. 1.

Average response of gastric secretion to I.V. injection of 16 U. insulin in eight cases of gastro-duodenal ulcer.

product of recent studies of the secretory activities of the gastric glands(16-18) new light has been cast upon this question.

Material and methods. Studies were carried out on 4 subjects with normal stomachs, 8 with duodenal ulcer and 7 subjects in whom subtotal gastric resection had been performed for peptic ulcer. After 2 fasting specimens of gastric juice had been withdrawn through a Levine tube at intervals of 20 minutes, 16 units of regular insulin was administered to each subject intravenously. Subsequently gastric specimens were aspirated at 20, 40, 60 and 90 minutes after injection. In each specimen acidity was determined by the usual titration technic with Toepfer's reagent and phenolphthalein. Pepsin concentration was measured by the photometric modification of the hemoglobin method(19) and gastric mucoprotein by the colorimetric tyrosine methods of Glass and Boyd(20).

Observations. A. Normal subjects and those with duodenal ulcer. In gastric juice from the intact stomach, whether or not duodenal ulcer was present, there was observed uniformly a rise in the concentration of all three secretory products of gastric glands, namely acid, pep-

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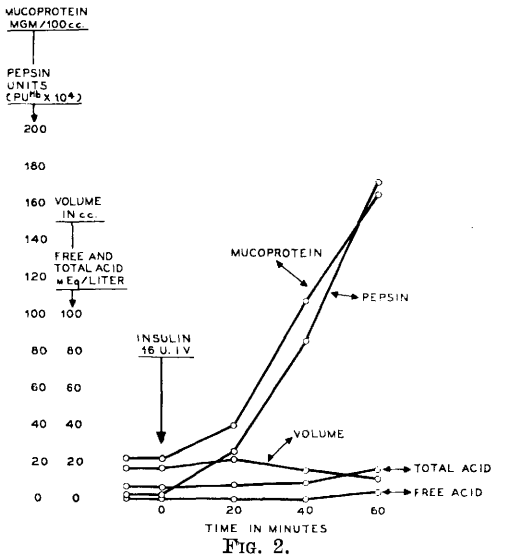

FIG. 2.

Average response of gastric secretion to I.V. injection of 16 U. of insulin in seven subjects, following removal of the distal ⅔ to ¾ of the stomach for peptic ulcer.

sin and mucoprotein following intravenous injection of insulin. The full data are reported elsewhere(16,17). Fig. 1 illustrates graphically the average changes. It will be noted that the peak of elevation of pepsin and mucoprotein coincided with the symptoms of hypoglycemia (pallor, sweating, weakness and hunger) and that the peak of acid response was delayed 20-40 minutes. A moderate elevation of acid, however, was present coincidental with the peak of pepsin and mucoprotein.

Comment. Numerous workers have shown that the lowest levels of blood sugar occur 20-30 minutes after intravenous administration of insulin (21-23). The above data indicate that the elevation of pepsin and mucoprotein correspond much more closely in point of time to the hypoglycemic reaction than does the rise in acid concentration. This suggests the possibility of an intermediary mechanism being involved in the latter reaction although some direct effect is suggested by the fact that moderate elevation of acid often coincided with the peak of pepsin and mucoprotein concentration.

B. Observations following partial gastric resection. Surgical removal of the distal 2/3-3/4 of the stomach for peptic ulcer altered the response of these 3 constituents to insulin administration. While elevation of pepsin and mucoprotein still occurred following insulin injection, a significant increase in acid and volume of secretion occurred in only 1 out of the 7 subjects. Fig. 2 illustrates graphically the average changes observed. Full data on this dissociated response by the gastric glands are presented elsewhere.

Comment. It has been shown that parietal cells are generally distributed throughout the body of the stomach (9,24,25). Thus the failure in most cases of insulin to induce an increased concentration of acid when only the antral end of the stomach has been removed, while elevation of pepsin and mucoprotein continue to occur, requires special explanation. The data suggest that the gastrin containing pyloric end of the stomach is involved in the acid secreting mechanism which is set in motion by vagus stimuli.

Conclusion. 1. The peak of elevation of gastric acidity in the intact stomach following insulin injection occurs significantly later than that of pepsin and mucoprotein, and 30-45 minutes after the maximal fall in blood sugar.

2. The acid stimulating effect of insulin is diminished and often abolished following resection of the distal end of the stomach while the pepsin and mucoprotein response are undiminished.

3. These observations add further strength to already existing evidence that while vagal stimuli appear to affect directly the glandular cells which secrete pepsin and mucoprotein and may act in part directly on the parietal cells, they also effect an increased elaboration of HCl in the human stomach by the intermediation of another mechanism that may involve the hormone "gastrin".

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