the control group and the Clamping-Cortisone group. From these findings, one of the reasons of prolongation of the ulcer induced by the Clamping-Cortisone method seemed to be attributed to decreases of mucous secretion from the mucosalcell and of polysaccarides in the connective tissue.

116. STUDIES ON EXPERIMENTAL PEPTIC ULCER (VIII) INFLUENCE OF PANCERATIC LIPIDE ON PATHOGENESIS OF EXPERIMENTAL PEPTIC ULER

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In the previous report, as the pathogenesis of Mann-Williamson ulcer produced by Keefer's method, it was assumed that important factors associated with the development of peptic ulcer (ulcer inhibiting substance) might be contained in inactivated fraction (enzymes) of extract of the pancreas. Furthermore, it was proved that ulcer inhibiting substance was not contained in trypsin or amylase of the pancreas, but contained in the mixture of pancreatic lipase and inactivated extract of the pancreas. This time, in order to examine whether ulcer inhibiting substance might be contained in pancreatic lipide, the following substances were administered orally to the ulcer provoking dogs divided into six groups. Namely, mixture of lipase and inactivated extract of the pancreas in Group I. Mixture of lipase and filtrate of inactivated extract of the pancreas in Group II. Mixture of lipase and dregs from inactivated extract of the pancreas in Group III. Mixture of lipase and pancreatic lipide extracted from the pancreas in Group IV. Mixture of lipase and dregs from extract of pancreatic lipide in Group V. Mixture of lipase, pancreatic lipide and dregs from extract of pancreatic lipide in Group VI. Four or five weeks after the operation, concerning the development of ulcer, no ulcer was found in all cases of Group I. However, ulcer was found in 50% of Group II. On the other hand, ulcer was observed in all cases of Group III which was administered dregs from inactivated extract of the pancreas. Namely, it is assumed that ulcer inhibiting effect does not exist in protein of the pancreas alone. No ulcer was observed in one out of three of Group IV which was administered lipide extracted from the pancreas. On the other hand, ulcer was observed in all cases of Group V which was given dregs from extracted pancreatic lipide. While, no ulcer was found in two out of three cases of Group VI which was administered all component of the pancreas. From the above mentioned results, it is assumed that there is close relation between ulcer inhibiting substance and pancreatic lipase plus pancreatic lipide, and this action is increased by the addition of all component of the pancreas. Therefore, pancreatic lipide is necessary factor for ulcer inhibiting action and other component of the pancreas is sufficient factor. Through this experiment, there was no definite relation between the development of ulcer and the change of gastric acidity or the histologic findings of the liver and the pancreas.

117. RESPONSES OF THE HEPATIC VAGAL AND SYMPATHETIC RECEPTORS TO MECHANICAL AND CHEMICAL STIMULI

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Mode of excitation of the hepatic receptors were examined in dogs anesthetized with pentobarbital. The afferent impulses were led off from the hepatic branches of the vagus and lower thoracic sympathetic trunks, and were amplified for display on an oscilloscope. At the same time, the integrator unit was derived from the electroneurogram, and the effects on the impulses of mechanical and pharmacological stimuli were studied.

Vagal afferent impulses were evoked by compressing slightly the surface of the liver, elevating the portal, hepatic venous, and hepatic arterial pressure. Also, they increased concomitantly