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THE COMMONLY EMPLOYED stimulants and inhibitors of gastric secretion are vasoactive substances. In glands of embryologic derivation similar to that of the stomach, the relation between glandular secretion and blood supply has been established.[1, 2] It seems reasonable, therefore, that changes in gastric blood flow should also be associated with changes in secretion.

Thompson and Vane investigated the relation between gastric blood flow and secretion in the cat.[3] They found that increases in blood flow to the histamine-stimulated stomach were correlated with increases in gastric secretory rate. Since histamine may act directly on secretory cells independently from its action on blood vessels, the inferences of Thompson and Vane are limited.

The investigation described below was primarily concerned with the relation of gastric secretion to its blood supply under conditions in which no extrinsic stimuli were delivered to the stomach and in which the arterial inflow could be held constant. Since vagal stimuli influence the gastric secretory rate, the vagi were ligated in half the animals investigated.

METHODS

Fourteen mongrel dogs of both sexes weighing 12–25 kg. were subjects of these studies. They were anesthetized with pentobarbital sodium (35 mg./kg.) and administered heparin sodium (5 mg./kg.) as an anticoagulant. A tracheotomy was performed and artificial respiration was employed if the thorax was violated during ligation of the cardia.

The method used in this study was described in detail previously.[4] Essentially it consisted of the interposition of a pump between the femoral and left gastric arteries and the ligation of all other arterial branches to the

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stomach. Both ends of the stomach were also ligated so as to include the vagi; however, in half of the animals the dorsal vagus nerve was excluded from the ligation. Care was employed to minimize trauma to the celiac plexus. A glass cannula, 1 in. in diameter, was sutured into the lumen of the stomach to allow collection of gastric juice. Left gastric arterial blood flow was set at 35, 60, and 85 ml./min. for 20-min. periods in a random sequence from experiment to experiment. During each period gastric juice was collected and the pressure gradient across the gastric vascular bed was determined. The volume and *p*H of the gastric secretion were measured.

At the completion of eight of the preceding experiments, histamine phosphate* (13 μg. base per minute) was infused into the left gastric artery for 20 min., while blood flow was fixed at 60 ml./min.

*Histamine phosphate (Eli Lilly and Co., Indianapolis, Ind.) contains 0.10 mg. base per milliliter of solution.

TABLE 1. CHANGES IN GASTRIC SECRETION IN THE DOG IN RESPONSE TO ALTERATIONS IN BLOOD FLOW

	*Vagi ligated**					*Dorsal vagus preserved**			
Dog	*Q (ml./min.)*	*GSR (ml./20 min.)*	*GSpH*	*R (mm. Hg/ml./min.)*	*Dog*	*Q (ml./min.)*	*GSR (ml./20 min.)*	*GSpH*	*R (mm. Hg/ml./min.)*
1	35	2	5.6	4.83	8	35	6		3.63
	60	3	2.1	3.20		60	10		3.03
	85	0		3.15		85	15		2.28
2	35	1	4.2	8.00	9	35	10		2.26
	60	7	1.2	3.61		60	12		2.27
	85	3	2.1	3.59		85	18		2.18
3	35	0		4.71	10	35	2	4.5	4.86
	60	3	1.7	3.55		60	9	6.0	3.80
	85	1	2.8	3.31		85	12	7.0	3.82
4	35	0		3.91	11	35	5	4.0	3.26
	60	1	6.8	3.35		60	10	6.9	3.03
	85	0		2.56		85	18	7.5	3.41
5	35	0		4.00	12	35	3	3.4	3.66
	60	0		2.90		60	7	6.8	3.67
	85	0		2.44		85	12	7.1	2.66
6	35	1	4.0	3.20	13	35	4	1.7	4.86
	60	0		2.60		60	4	1.9	3.61
	85	0		2.38		85	11	7.0	3.41
7	35	0		3.69	14	35	3	3.8	4.57
	60	0		3.80		60	15	6.7	4.00
	85	0		2.96		85	30	7.0	3.70
MEAN	35	1		4.62	MEAN	35	5		3.87
	60	2		3.28		60	10		3.34
	85	1		2.91		85	17		3.07

*Key: *Q* indicates gastric artery blood flow; *GSR*, gastric secretory rate; *GSpH*, *p*H of gastric secretions; and *R*, vascular resistance across stomach.

RESULTS

The secretory responses of the two groups of stomachs to change in blood flow were different. In the group with the dorsal vagus intact, gastric secretion increased with increments in blood flow. In the group with the vagus ligated there was little or no gastric secretion and changes in blood flow had no influence on the secretory rate. These results are shown in Table 1 and Fig. 1.

It can be seen from Fig. 2 that when blood flow remained at 60 ml./min., gastric secretion was significantly increased in response to local histamine infusion in either group of animals. Whether gastric secretion was increased by changing blood flow or by infusing histamine, the *p*H of gastric secretion rose (Table 1).

DISCUSSION

In this study gastric secretory rate was a function of changes in blood flow only in those stomachs in which the dorsal vagus nerve was preserved.

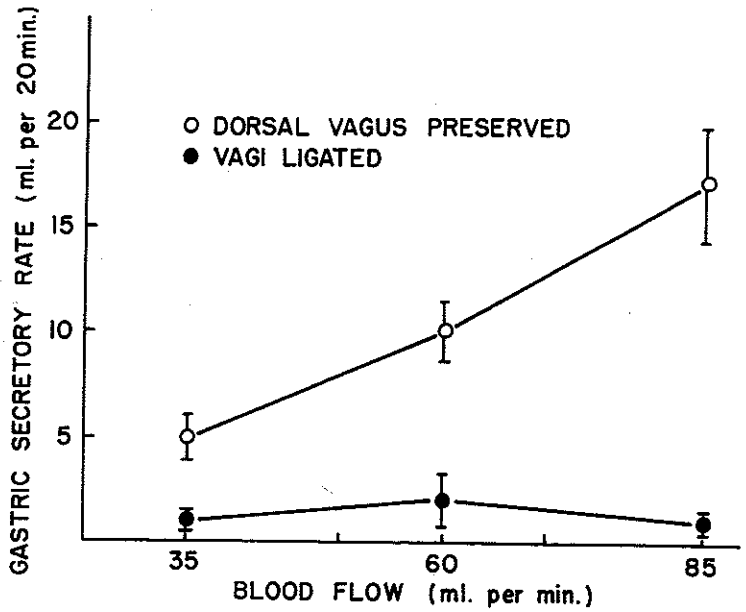

Fig. 1. Effect of change in blood flow to stomach on gastric secretory rate in stomachs with dorsal vagus preserved and in those with it ligated. Seven animals are included in each group. One standard error of the mean is plotted above and below each average value.[5]

Histamine increased gastric secretion independent of changes in gastric blood flow in both the vagus-ligated and preserved stomachs.

The stomach responds, therefore, in a manner analogous to the pancreas and the salivary glands.[1, 2] In all three organs blood flow can be correlated with secretory activity. However, the glandular secretory rate may be altered without changes in blood flow.[2, 6]

The mechanism whereby histamine increased gastric secretion in this preparation was not defined; however, it seems likely that it acted primarily on secretory cells, independent of its vascular effects. Peters and Womack suggested another possibility whereby histamine closed submucosal shunts and redistributed blood flow primarily to the secretory mucosa.[7] It is possible that both mechanisms are operative.

The reason for the failure of parietal-cell secretion to increase with in-

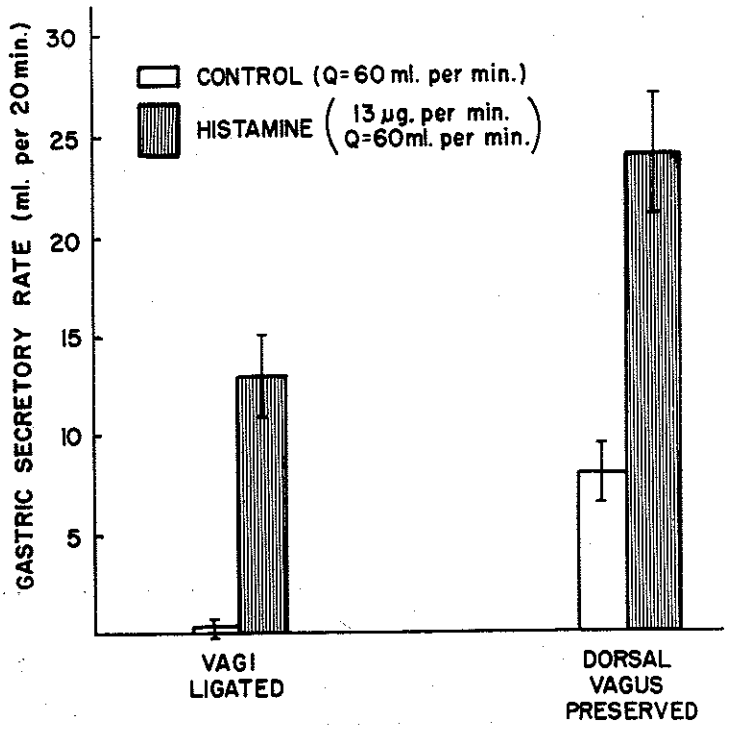

Fig. 2. Effect of histamine infusion on the gastric secretory rate of stomachs with dorsal vagus ligated and in those with it preserved. Four animals were in each group. Gastric blood flow (Q) was maintained at 60 ml./min. for 20-min. control and histamine periods in each group. One standard error of the mean[5] is plotted above and below the average values.

crements in gastric secretion, observed in the present study, is not apparent. This was also noted in a similar investigation by Thompson and Vane,[3] who suggested that the wide pulse pressure characteristic of their pump had induced a primarily nonparietal-cell gastric juice, possibly in part a transudate. In our study pulse pressure narrowed with increasing flow while nonparietal secretion increased. It would also be difficult to explain, on the basis of a pump artifact, the appearance of nonparietal cell secretions in response to flow increments only in stomachs with an intact dorsal vagus. Moreover, histamine stimulated secretion without any change in pulse pressure.

The results of this study suggest that, in the normally secreting stomach, blood flow changes may result in corresponding changes in gastric secretion. Recently, Womack and his associates reported correction of gastric hypersecretion in 2 patients by surgical reduction of the blood supply to the stomach.[8] Our experiments suggest further that alteration in blood flow to the stomach is not the sole determinant of gastric secretory rate.

SUMMARY

In 14 dogs the response of the gastric secretory rate to alterations in blood flow was measured. In 7 animals whose dorsal vagus was intact, increasing blood flow of the stomach was followed by increases in the secretory rate. In the remaining dogs whose vagi were ligated, changes in blood flow had no effect upon gastric secretion. In either vagus-intact or vagus-ligated stomachs histamine infusion increased or initiated gastric secretion without any change in blood flow.

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