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Hemodynamics of the Stomach

I. Resistance-flow Relationship in the Gastric Vascular Bed

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RESISTANCE-FLOW RELATIONSHIPS have been described for various organs including the kidney, forelimb, heart, intestines, and lung.¹⁻⁵ These studies indicate that each vascular bed has a different resistance response to increasing blood flow. For example, renal vascular resistance increases as a function of flow over the intermediate range; conversely, lung resistance declines as flow rises. The relationship of resistance to flow has not yet been defined for the stomach. This report is concerned with the response of perfusion pressure and resistance to change in flow in the vascular bed of the stomach.

METHODS

Acute studies were conducted on 16 mongrel dogs of both sexes whose weights ranged from 12 to 22 kg. The animals were anesthetized with sodium pentobarbital (35 mg./kg.) and administered heparin (5 mg./kg.) as an anticoagulant. A tracheotomy was performed in each animal and artificial respiration utilized in 4.

The blood supply of the stomach is complex. Each of the three divisions of the celiac axis sends branches to the stomach. Since the splenic and hepatic arteries also deliver blood through multiple branches to the spleen, pancreas, omentum, liver, duodenum, and esophagus, it was necessary to ligate these arteries and limit perfusion to the left gastric artery (Fig. 1).

A 10-cm. left subcostal incision was made. The celiac axis was dissected free, and the hepatic and splenic arteries were ligated. The cardiac and

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pyloric ends of the stomach were also ligated. In 6 of the animals the vagi were preserved. Care was observed to minimize trauma to the sympathetic plexus in the operative site.

The right gastric and the right and left gastroepiploic arteries and the

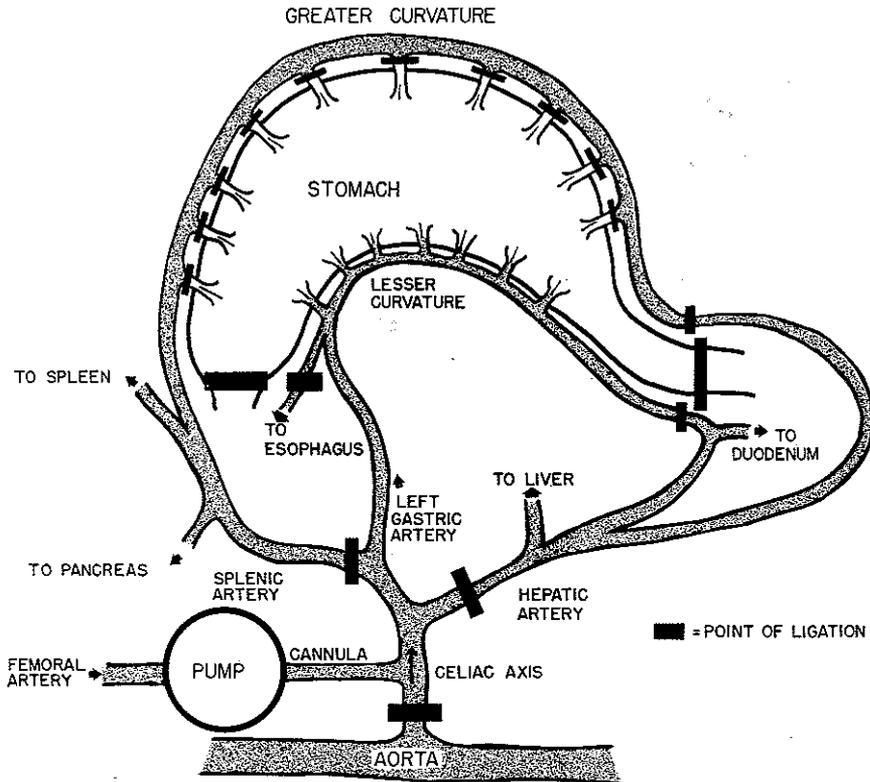


Fig. 1. Diagrammatic representation of the stomach, its arterial supply, and the points of ligation used in the study described.

vessels entering the stomach along the greater curvature were ligated just proximal to the stomach. Splenectomy was also performed.

The celiac axis was clamped at the aorta and a pressure-independent,* variable-speed pump (Sigmamotor pump Model T-6†) interposed between the right femoral artery and the celiac axis. Blood flow to the stomach was interrupted for no longer than 1 min. The extracorporeal circulation was

*The output of this pump did not change despite variation in outflow pressure over the range 0-400 mm. Hg.

†Sigmamotor, Inc., Middleport, N. Y.

passed through a water bath to keep blood temperature at 37°C. Hence, the entire blood supply of the stomach was maintained through the left gastric branch of the celiac axis.

The vascular pressures in the left gastric artery and coronary vein were monitored continuously by means of a pressure transducer connected to a Sanborn Twin Viso recorder.* Average arterial and venous blood pressures were used for the calculation of the pressure gradient across the stomach.

In each experiment, pump flow was set at the lowest level (20 ml./min.) and raised by irregular increments to the highest flow (120 ml./min.). After each alteration in flow, pressures were allowed to stabilize for 30 sec. before flow was changed again. After the maximal perfusion rate was reached, flow was reduced to the lowest rate.

India ink injected into the left gastric artery at the end of the experiment demonstrated that the perfusion system supplied the entire stomach without leakage.

Resistance was calculated as pressure gradient divided by flow.

RESULTS

Gastric vascular pressure rose as a function of increasing flow. In 16 dogs the rise in pressure was less than directly proportional to increments in flow. As flow rose, therefore, resistance fell. The rate of decline of resistance was rapid up to a flow of 60 ml./min., after which it became less rapid. The changes in pressure gradient and resistance as functions of increasing flow are shown in Fig. 2.

The relationship of pressure gradient to resistance in these animals is presented in Fig. 3. Resistance declined sharply over the pressure range 60–100 mm. Hg and then fell only slightly over the range 100–160 mm. Hg.

When flow was subsequently reduced from a high to a low level, pressure fell and resistance rose, so that, for the same rate of flow, resistance was usually higher on the decrement than the increment (Fig. 4).

DISCUSSION

The intrinsic ability of an organ to maintain its flow in the face of changing pressures has been termed autoregulation. When flow is the independent variable, autoregulation may be identified with certainty by a rise in resistance as a function of flow. The results of this investigation suggest that autoregulation, if it occurs at all, is very imperfect in the stomach.

*Sanborn Co., Cambridge, Mass.

The relation of resistance to flow in the gastric vascular bed was biphasic. As flow was raised from 20 to 60 ml./min. resistance fell sharply. Subsequent increases in flow resulted in a far less marked decline in resistance. The sharp decrease in resistance seen in the first phase could have been

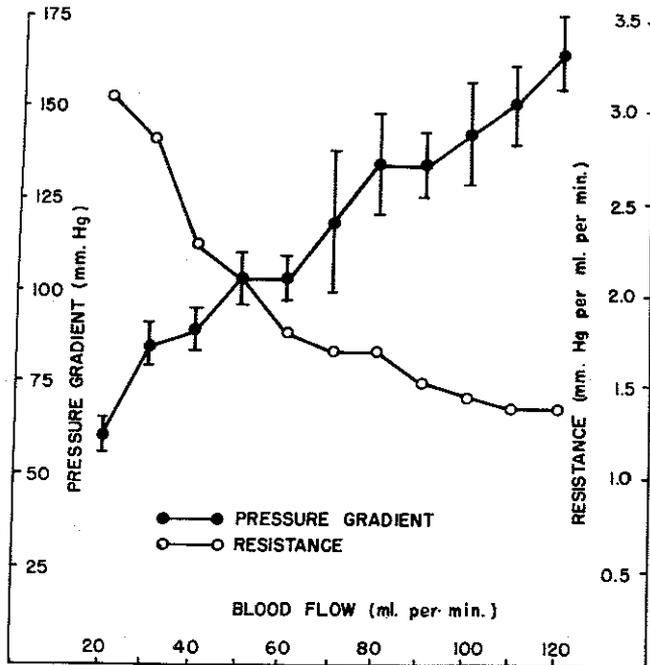


Fig. 2. Pressure gradient and vascular resistance as functions of blood flow in the gastric vascular bed of the dog. One standard error of the mean is plotted above and below each point on the pressure curve.⁸ The values shown represent the average from 16 dogs.

due to opening of anastomotic channels within the stomach. In the experiment described, ligation of three of the four arteries supplying the stomach was performed, but the gastric branches of the ligated vessels were left intact and in communication with the perfusing left gastric artery. In addition, increasing flow may distend open vessels and thereby cause a decline in resistance. Raising velocity also diminishes viscosity of blood, but the viscosity change is probably greatest at very low velocities.⁷ The extent to which these factors affected the rapid early fall in the resistance-flow curve is not clear from this investigation.

Beyond flows of 60 ml./min. the resistance slope became very nearly horizontal with respect to further increases in flow. Several potential influ-

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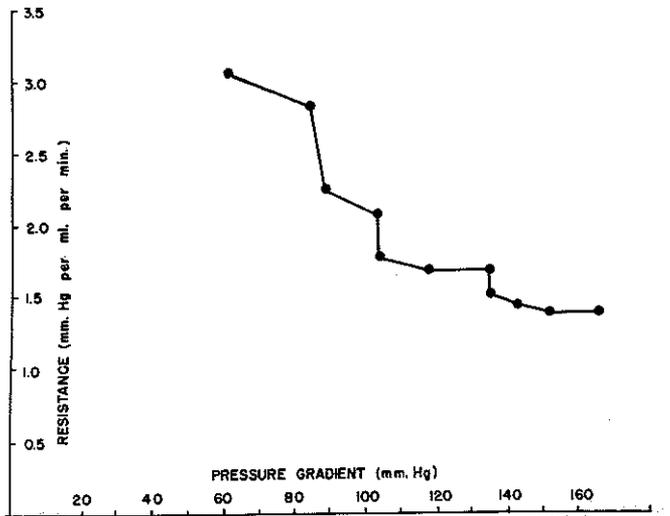


Fig. 3. Resistance to blood flow through the vascular bed of the stomach as a function of pressure gradient. Values are an average of 16 experiments.

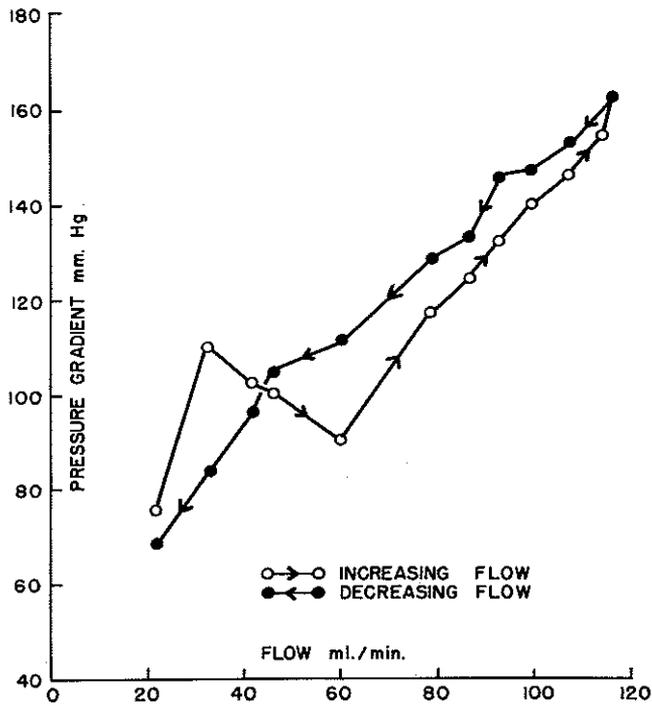


Fig. 4. Comparison of the relationship of pressure gradient to flow in a single stomach when flow was raised and when flow was lowered. Arrows indicate direction of flow.

ences might have been responsible for this change in the resistance response to acceleration of flow in the higher range. Greater intravascular pressures could have induced increasing ultrafiltration rates of plasma fluid into the extravascular tissue. This would have served to slow the decline in resistance to blood flow by limiting further vascular distention. In addition, each vascular bed has a limit of distention. If this limit had been approached a less marked decline in resistance would have resulted. Both of these factors are passive in nature. On the other hand, the slower rate of fall of resistance might have resulted from enhanced contractility of the smooth muscle in the vessel wall in response to stretch, metabolic factors, or autonomic nervous stimuli. The degree to which passive and active factors contributed to this phase of the response is not apparent.

The resistance pattern observed in the stomach is similar to that described for the coronary circulation.³ The change in slope seen in both beds develops at a flow of approximately 60 ml./min. In the stomach the pressure at this flow was about 100 mm. Hg. Flow and pressure values in excess of these levels are most consistent with a stomach in its normally functioning state, where resistance was found to decline gently with further increments in flow. Whether this reluctance of resistance to fall as flow rose represented some degree of active contraction cannot be stated firmly. Only when resistance clearly rises with increases in flow is active contraction established with some certainty under drug-free conditions.

Gastric resistance was decidedly higher than resistance values described for the heart, whether resistance was calculated from flow to the organ or flow per gram of tissue.³ These resistance values, however, agree with resistances calculated from data reported for the cat stomach.⁸

The generally higher resistances found when flow was lowered over the same scale of values used in raising flow suggests a hysteresis pattern due to inertia in residually filled vessels.

SUMMARY

A method is described for measuring the change in blood pressure gradient across the stomach of the dog in response to changes in blood flow.

In 16 dogs pressure-gradient responses were measured and vascular resistance calculated. The rise in pressure gradient was not proportionate to the increase in blood flow. Resistance, therefore, decreased as blood flow rose. The decline in resistance to increments in flow was especially marked till flow reached a rate of 60 ml./min. Beyond this flow the fall in resistance became slight. It appears that autoregulation does not occur to any appreciable degree in the gastric vascular bed.

When flow was lowered over the same range used in raising flow, pressure values somewhat different were obtained, suggesting a hysteresis effect.

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