Hemodynamic Basis of the Pain of Chronic Mesenteric Ischemia

John W. Poole, MD, Robert J. Sammartano, BS, and **Scott J. Boley, MD,** Bronx, New York

Abdominal pain is an integral part of the clinical presentation of chronic mesenteric ischemia. Characteristically, the patient does not have pain as long as he does not eat or has small meals. Fifteen to 30 minutes after a large meal, however, severe crampy or colicky abdominal pain begins that typically persists from 1 to 3 hours. This food-pain sequence is the dominant feature of chronic mesenteric ischemia, and ultimately leads to a reluctance to eat and an inevitable loss in weight.

The prevailing concept of the cause for the pain of chronic mesenteric ischemia is that a large postprandial increase in intestinal blood flow is required to supply oxygen for the metabolic processes of secretion and absorption, and for increased peristaltic activity. Then, just as cardiac muscle transmits pain when the supply of oxygen is unequal to the demand, abdominal pain also occurs when the superior mesenteric arterial supply is unequal to the demands of the intestinal smooth muscle activity [*1*]. The similarities of this perceived cause of the abdominal pain to that of angina pectoris have prompted the use of the terms abdominal or intestinal angina to describe the pain of chronic mesenteric ischemia.

The onset of pain usually within 30 minutes of eating, however, conflicts with this theory, as the ingested food has not yet reached the small intestine when the pain begins. The most commonly suggested explanation for this unusual temporal relationship is that a humoral or hormonal substance that affects the small intestine is released when food is introduced into the stomach. We have thought that there may be a better hemodynamic explanation for the time sequence, but, heretofore, we have not found a good experimental model to evaluate this hypothesis.

Recently, Fiddian-Green and co-workers [*2–4*] described the use of tonometry for indirectly measuring intramural intestinal pH as a metabolic marker of the adequacy of oxygenation and hence blood supply in the gut. This technique has been validated by that group [*5,6*] by direct measurement and has been confirmed by us utilizing direct blood flow measurement [*7*]. In both studies, the tonometrically determined intramural pH was confirmed to be a sensitive, reproducible, and linearly related measurement of diminished blood flow to still viable small bowel.

Tonometry is predicated on the fact that the partial pressure of carbon dioxide in the lumen of a hollow organ equilibrates with that in its wall, and the partial pressure of carbon dioxide in fluid placed within a semipermeable balloon in the lumen of the bowel similarly equilibrates with the partial arterial carbon dioxide pressure in the lumen, and in turn with that in the wall. If one combines that knowledge with the assumption that the bicarbonate in the intestinal wall is the same as that being delivered to it in arterial blood, one can then substitute the partial pressure of carbon dioxide in the fluid in the tonometer, and the bicarbonate of the arterial blood, into the Henderson-Hasselbalch equation to determine the intramural pH.

In the present study, we used this new technique for the indirect measurement of intestinal wall perfusion as a means of determining the effect of simulated meals on gastrointestinal blood flow in the presence of a fixed, decreased splanchnic blood flow.

Material and Methods

Fourteen adult greyhounds weighing 30 to 33 kg were anesthetized with pentobarbital sodium (25 mg/kg) and maintained on a respirator and room air during the entire experiment. A peripheral vein was cannulated for administration of normal saline solution at 150 ml/hour. A midline celiotomy was performed and the celiac and superior mesenteric arteries were identified. The celiac artery was dissected from its origin at the aorta to a point 2 cm distal to its trifurcation. The superior mesenteric artery was dissected from its origin at the aorta distally to the pancreaticoduodenal artery, a branch that communicates with the celiac artery. All branches of the superior mesenteric artery proximal to the pancreaticoduodenal artery

From the Department of Surgery, Albert Einstein College of Medicine and Montefiore Medical Center, Bronx, New York.

Requests for reprints should be addressed to Scott J. Boley, MD, Department of Surgery, Montefiore Medical Center, 111 East 210th Street, Bronx, New York 10467.

Presented at the 17th Annual Meeting of the Society for Surgery of the Alimentary Tract, San Francisco, California, May 20–21, 1986.

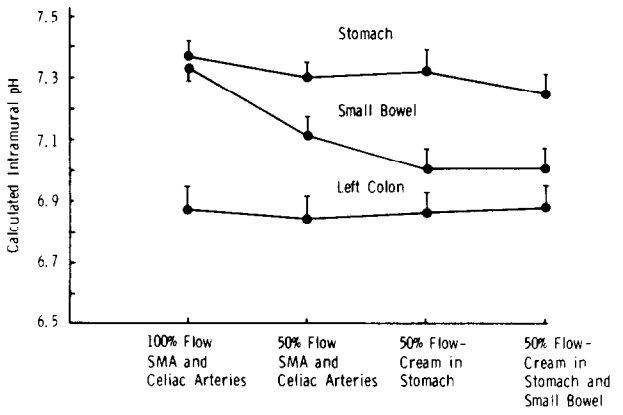

Figure 1. Changes in intramural pH in the stomach, small bowel, and colon with diminution in splanchnic blood flow and with simulated meals in the stomach and small bowel. SMA = superior mesenteric artery.

were ligated and divided. Electromagnetic flow probes and hydraulic occluders were placed on the celiac artery and superior mesenteric artery. The flow probes were connected to a pulsed logic flow meter (Biotronics Laboratories, Silver Springs, MD) and continuously recorded on a four channel recorder. A femoral artery was cannulated for arterial blood sampling and to monitor systemic blood pressure.

Through enterotomies, tonometers constructed from Silastic® silicone elastomer (ASTM designation, type VMQ, Dow Corning, Midland, MI) were placed in the stomach, jejunum, and left side of the colon and fixed in place with pursestring sutures. A catheter was inserted through the antral wall of the stomach and sutured in place for the administration of cream. Another catheter was inserted in similar fashion through the jejunal wall approximately 50 cm distal to the tonometer already in place.

Intramural pH was determined in the small bowel, stomach, and colon at normal superior mesenteric artery and celiac artery blood flow, at 50 percent of superior mesenteric artery and celiac artery blood flow, and at 50 percent superior mesenteric artery and celiac artery flow after the instillation of cream into the stomach and again after the instillation of cream into the small bowel. In the two control dogs, intramural pH was determined after 1 hour of 100 percent flow, and hourly during 3 hours of 50 percent flow. The partial pressure of carbon dioxide of the appropriate tonometer aspirates and the bicarbonate in the sample of arterial blood that had been simultaneously collected, were placed in the Henderson-Hasselbalch equation to determine the intramural pH.

In all animals, the tonometers were filled with 3 ml of normal saline solution and allowed to equilibrate for 1 hour. The saline solution was then aspirated and the partial pressure of carbon dioxide of the samples obtained during normal splanchnic blood flow was determined by a blood gas analyzer (model ABL-1, Radiometer America, Westlake, OH). Celiac artery and superior mesenteric artery blood flows were then decreased to, and maintained at, 50 percent of normal values, and the tonometers were refilled and allowed to equilibrate for 1 hour at the 50 percent blood flow. The saline solution in the tonometers and an arterial blood sample were again analyzed, and the occlusions released on both arteries. After 20 minutes at 100 percent blood flow, both arterial blood flows were again decreased to 50 percent of normal flow and the tonometers refilled.

In 12 dogs, the pylorus was ligated with umbilical tape. Then, with the splanchnic blood flow at 50 percent, 60 ml of cream was introduced into the stomach through the catheter already in place. After 1 hour of equilibration, the tonometric aspirates and an arterial blood sample were analyzed and the arterial occlusions released. Full blood flow was again maintained for 20 minutes, after which the blood flows were decreased to 50 percent of normal flow and the tonometers were refilled. Sixty milliliters of cream were then instilled into the jejunum through the catheter already in place; after 1 hour, the final tonometer and arterial blood samples were obtained and analyzed.

In the two remaining dogs, all experimental procedures were identical, but there was no cream instilled into the stomach or small bowel. All animals were sacrificed at the end of the experiments.

The significance of the changes in intramural pH were analyzed by the Wilcoxon signed-ranks test.

Results

In all 14 dogs, a significant decrease in intramural pH occurred in the small bowel when the superior mesenteric artery and celiac artery blood flows were decreased from 100 percent to 50 percent of normal flow ($p < 0.05$). In the 12 dogs in which a meal was simulated, the intramural pH of the small bowel decreased again when cream was instilled into the stomach while splanchnic blood flow was maintained at 50 percent of normal ($p < 0.05$). Thus, the mean intestinal intramural pH decreased from 7.33 to 7.11 when flow was decreased from 100 percent to 50 percent and decreased further from 7.11 to 7.02 when cream was placed into the stomach (Figure 1). When cream was also instilled into the small bowel with the superior mesenteric artery and celiac artery blood flows still at 50 percent, the mean intestinal intramural pH decreased insignificantly from 7.02 to 7.01.

Intramural pH changes in the stomach were statistically significant when the splanchnic blood flow was decreased from 100 percent to 50 percent ($p < 0.05$) and also when cream was placed into the small bowel with 50 percent blood flow ($p < 0.05$). The mean gastric intramural pH decreased from 7.37 at 100 percent flow to 7.30 at 50 percent flow, but there was no further significant change when cream was instilled into the stomach (Figure 1). However, the mean gastric intramural pH did decrease significantly from 7.30 to 7.25 when cream

was placed into the small bowel while flow was maintained at 50 percent.

In the left side of the colon, where the inferior mesenteric artery flow was unimpeded, the colonic intramural pH showed no significant change (Figure 1).

In the two remaining control animals in which cream was not instilled, the mean small bowel intramural pH decreased from 7.26 to 7.06 when splanchnic flow was decreased from 100 percent to 50 percent, but showed no further decrease for the remainder of the experiment at 50 percent flow. The mean gastric intramural pH in these animals did not change significantly throughout the experiment.

Comments

A unique aspect of the experimental model in this study is the use of intramural pH, determined indirectly by tonometry, to assess perfusion and oxygenation of the gastrointestinal tract. This metabolic marker may even prove to be superior to direct measurements of partial arterial pressure of oxygen in the intestinal wall because it measures the adequacy of oxygen delivered related to tissue needs. The pH in the wall of the gut is maintained at normal levels in the face of decreasing oxygen delivery until a critical level is reached. Any reduction in oxygen delivery below this critical level is accompanied by a precipitous decrease in tissue pH [2]. This decrease is a consequence of the disruption of cellular metabolism, which leads to anaerobiosis and cellular acidosis. The ability to measure intramural pH by tonometry in effect allows indirect monitoring of cellular metabolism and is a useful tool to assess tissue oxygenation.

Intramural pH is affected by both blood supply and partial arterial pressure of oxygen, but when the partial pressure of oxygen of the inspired air, and therefore partial arterial pressure of oxygen, are kept constant (as in this experiment), the intramural pH is an indirect measure of blood flow. Thus, using intramural pH, together with regulating and continuously measuring arterial inflow into the splanchnic vascular bed with the electromagnetic flow probes, we were able to study the redistribution of a fixed, decreased splanchnic arterial inflow during conditions simulating ingestion of a meal. By ligating the communication between the superior (cranial) mesenteric artery and the inferior (caudal) mesenteric artery, the main source of collateral blood flow to the splanchnic circulation was interrupted. Hence, except for minor collateral pathways to the celiac artery and superior mesenteric artery, splanchnic arterial inflow could be accurately controlled.

The data from these experiments show that decreasing and maintaining splanchnic arterial inflow at 50 percent of normal flow produced mild small bowel intramural acidosis, and that the addition of a simulated meal in the stomach resulted in further, more severe acidosis. The latter observation indicates increased small bowel ischemia when the meal entered the stomach, which in the presence of a fixed splanchnic arterial inflow suggests that the blood has been redistributed from the small bowel to the stomach, the hemodynamic phenomenon termed a steal syndrome.

The lack of change in the colonic intramural pH is evidence that the alterations in the gastric and intestinal intramural pH are the result of changes in blood flow, and are not the result of trauma from the creation of the experimental model. The lower intramural pH of the colon, compared to that in the stomach and small bowel, has been noted by Fiddian-Green et al [8] both in animals and human subjects.

Support for the occurrence of a hemodynamic steal under the conditions created in our experiments is provided by previous investigations reported from our laboratory on the effects of altering the resistance of segments of the splanchnic circulation, and by reports from a number of other investigators on the segmental nature of postprandial hyperemia. The borrowing and lending or steal concepts of distribution of blood flow are based on the premise that increased blood flow to one segment of the vascular bed is achieved at the expense of flow to another area [9]. In the subclavian steal syndrome, for example, cerebral ischemia occurs when cerebral blood flow is detoured to supply the undervascularized arm.

The splanchnic circulation is really one vascular bed with two major sources of arterial inflow (the celiac and superior mesenteric arteries) and a minor collateral source (communications with the inferior mesenteric artery). Because of this rich arterial supply, it has often been observed that two, or all three, of the arteries supplying the splanchnic organs must be narrowed or occluded for chronic mesenteric ischemia to occur. Despite this experience, some investigators have attributed various intestinal ischemic syndromes to steal phenomona.

In previous experiments, we used an experimental model simulating the clinical situation of superior mesenteric artery stenosis and showed that decreasing superior mesenteric artery flow by partial occlusion with a balloon occluder resulted in increased blood flow from the celiac artery through the pancreaticoduodenal artery into the superior mesenteric artery circulation. This increased collateral circulation was accompanied, however, by increased, not decreased, flow through the left gastric and splenic arteries [10,11]. When in addition to the simulated superior mesenteric artery stenosis, vascular resistance in the superior mesenteric artery bed was also lowered with locally infused papaverine to increase flow to this segment of the splanchnic circulation, celiac artery flow further increased, as

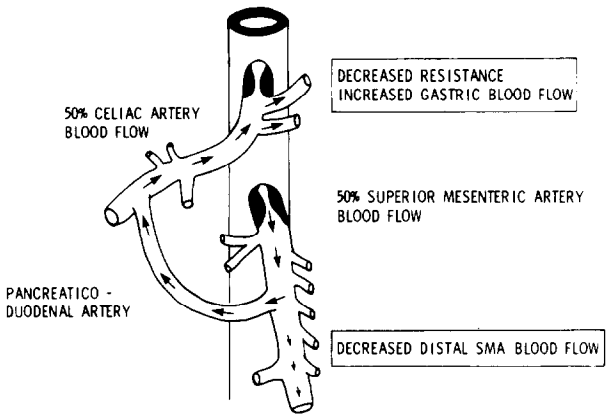

Figure 2. Hemodynamic changes that occur with fixed, decreased splanchnic blood flow and decreased vascular resistance in the gastric circulation. SMA = superior mesenteric artery.

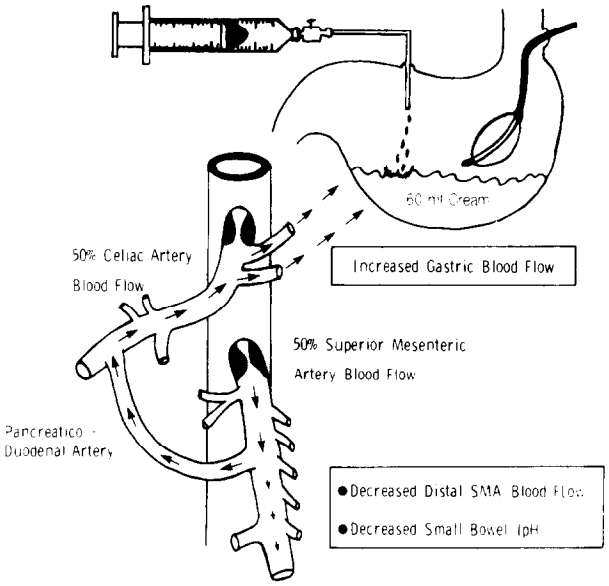

Figure 3. A postulated hemodynamic explanation for small intestinal ischemia (and thus pain) soon after eating demonstrating that local gastric hyperemia is accomplished by a steal of blood from the small bowel. IpH = intramural pH; SMA = superior mesenteric artery.

did flow through the pancreaticoduodenal artery to the superior mesenteric artery. Again, this increased collateral circulation was accompanied by an increase in gastric and splenic arterial flow. These studies showed that as long as inflow through one of the main arteries supplying the splanchnic bed was not restricted, increased flow in one segment was not detrimental to flow to other areas. In subsequent experiments, however, both the superior mesenteric artery and celiac artery flows were reduced by partial occlusions before injecting papaverine into the superior mesenteric artery. During those studies splanchnic arterial inflow was fixed and limited, and under those conditions, increasing flow to the mesenteric bed did produce a decrease in flow to the gastric and splenic beds. Hence, when both the superior mesenteric artery and celiac artery are involved, as in most patients with chronic mesenteric ischemia, and inflow through the arteries is restricted, an increased demand in blood flow to one area of the splanchnic circulation is satisfied only by diminished flow in other areas. Such a situation could explain the increased intramural acidosis after the simulated meal in our study if the introduction of the cream into the stomach lowered gastric resistance (Figure 2) and produced local gastric hyperemia.

Several investigators have shown that postprandial hyperemia is localized to the regions in which the food is present or in which digestive processes are occurring [*12–14*]. Most pertinent to our present report is the observation by Chou and associates [*13*] that introduction of food into the stomach of the conscious dog increased blood flow only in the celiac artery and did not affect superior mesenteric artery flow. Although the mechanism or mechanisms by which this local hyperemia occurs remains controversial, it appears that the initial hemodynamic response to an ingested meal is local vasodilatation.

Although the observations we have made in this and previous studies have all been from long-term experimental models, these hemodynamic changes do provide a possible explanation for the temporal relationship of the postprandial pain of chronic mesenteric ischemia. Based on these findings, we postulate that patients with chronic mesenteric ischemia have a fixed decreased splanchnic arterial blood flow, and ingestion of food creates initially localized gastric hyperemia.

Increased blood flow to the stomach is achieved at the expense of superior mesenteric artery blood flow, producing small bowel ischemia severe enough to cause pain. The hemodynamic explanation for this ischemia is a true gastrointestinal steal phenomenon. The increased intestinal ischemia when food is introduced into the stomach would then explain the early onset of postprandial pain in patients with chronic mesenteric ischemia (Figure 3).

An intriguing development evolving from the observations made in this study is the possibility of a new method for clinically diagnosing chronic mesenteric ischemia. By passing a tonometer into the proximal small bowel in the same manner as a long intestinal tube, intestinal intramural pH can be measured. An abnormally low intestinal intramural pH might be enough to identify ischemia, but if not, a decrease in the intestinal intramural pH after a test meal is introduced into the stomach could es-

tablish the diagnosis. This diagnostic approach is presently being investigated.

Summary

Tonometry, a new technique to indirectly assess intestinal blood flow, was used to determine the hemodynamic changes produced by a simulated meal in animals with a fixed, decreased splanchnic blood flow. In experiments on 14 dogs, celiac artery and superior mesenteric artery blood flow was maintained at 50 percent of normal flow by occluders and flow probes, and tonometers were placed in the stomach and small bowel to measure intramural pH, a metabolic marker of intestinal perfusion. Intramural pH was determined at 100 percent and 50 percent splanchnic blood flow, at 50 percent flow after instillation of cream into the stomach, and again when cream was placed into the small bowel. Intestinal intramural pH decreased significantly when blood flow was decreased to 50 percent, as expected, but decreased significantly again when cream was placed in the stomach. The hemodynamic explanation of the decrease when cream was placed in the stomach is a steal from the intestinal to the gastric circulation stimulated by food in the stomach. Such a steal could explain the temporal nature of the pain experienced by patients with chronic mesenteric ischemia.

References

1. Spiro HM. Clinical gastroenterology. London: Macmillan, 1970: 993.
2. Grum CM, Fiddian-Green RG, Pittenger GL, Grant BJB, Rothman ED, Dantzker DR. Adequacy of tissue oxygenation in the intact dog intestine. J Appl Physiol 1984; 5: 1065–9.
3. Fiddian-Green RG. A sensitive and specific diagnostic test for intestinal ischemia using Silastic tonometers. Eur Surg Res 1984; 16: 32.
4. Wakefield TW, Whitehouse WM Jr, Pittenger GL. Early diagnosis of acute colon ischemia by hollow viscus tonometry in a canine model. Gastroenterology 1983; 84: 1344.
5. Fiddian-Green RG, McGough E, Pittenger GL, Rothman E. Predictive value of intramural pH and other risk factors for massive bleeding from stress ulceration. Gastroenterology 1983; 85: 613–20.
6. Fiddian-Green RG, Pittenger GL, Whitehouse WM. Back diffusion of CO_2 and its influence on the intramural pH in gastric mucosa. J Surg Res 1982; 33: 39–48.
7. Poole JW, Sammartano RJ, Boley SJ. The use of tonometry in the early diagnosis of acute mesenteric ischemia. Curr Surg (in press).
8. Fiddian-Green RG, Amelin PM, Herrmann JB, et al. Prediction of the development of sigmoid ischemia on the day of aortic surgery. Arch Surg 1986; 121: 654–60.
9. DeBakey ME, Burch G, Ray T, Ochsner A. "Borrowing-lending" hemodynamic phenomenon (hemometakinesia) and its therapeutic application in peripheral vascular disturbances. Ann Surg 1947; 126: 850.
10. Regan JA, Tunick PA, Kalter J, Winslow PR, Veith FJ, Boley SJ. Effects of alterations in segmental resistance on blood flow to other segments of the splanchnic circulation. Fed Proc 1970; 29: 260.
11. Boley SJ, Gliedman ML. Circulatory responses to mesenteric ischemia. In: Boley SJ, Schwartz SS, Williams LF Jr, eds. Vascular disorders of the intestines. New York: Appleton-Century-Crofts, 1971: 323–33.
12. Bond JH, Prentiss RA, Levitt MD. The effects of feeding on blood flow to the stomach, small bowel and colon of the conscious dog. J Lab Clin Med 1979; 93: 594–9.
13. Chou CC, Hsieh CP, Yu YM, et al. Localization of mesenteric hyperemia during digestion in dogs. Am J Physiol 1976; 230: 583–9.
14. Fara JW, Rubinstein EM, Sonnenschein RR. Intestinal hormones in mesenteric vasodilation after intraduodenal agents. Am J Physiol 1972; 223: 1058–67.

Discussion

Gregory B. Bulkley (Baltimore, MD): Dr. Boley, as I understand it, you are proposing that there is selective vasodilatation in the gastric vascular bed that sets up a differential change in gastric versus mesenteric vascular resistance, which would cause a steal to take place.

The hemodynamic requirements for a steal are three: First, there has to be a differential change in vascular resistances; second, there has to be a decrease in blood pressure downstream from the occlusion; and third, either a reduction or a reversal of collateral blood flow. You have not measured any of these parameters directly. In your experiments, you measured only intramural pH to infer a reduction in nutrient blood flow. Moreover, you did not measure perfusion pressures distal to the occlusion, although it would have been easy to do so. Since you had isolated the pancreaticoduodenal arcade, as you described in your study, you could also have measured the direction and volume of flow through the primary collateral circuit in question. You could then have calculated the resistances. Had you obtained the very simple measurements of these most basic of hemodynamic parameters, you could have established or refuted your hypothesis in a most straightforward manner.

This issue is not trivial, because the intramural pH (or the state of intestinal wall ischemia) is due to the balance between blood flow and metabolic demand. The underlying assumption of your study is that a change in pH is a reflection only of changes of flow.

When you put cream into the stomach, can we assume that there are absolutely no changes in metabolic demand in the small bowel? We might expect that a whole host of neural and humoral factors could be released in response to feeding, any of which could well change the metabolic activity and therefore the metabolic demand in the bowel. Chu's data from the nonischemic bowel do not really eliminate that possibility. If this were the case, it would be possible to explain your results on the basis of changes in metabolic demand. Indeed, perhaps your own data suggest this as a possible explanation. When you added cream to the small bowel as the last step in your experiment, there was no alleviation of the decrease in pH; it stayed the same. One possibility is the metabolic explanation I have just outlined. That is, perhaps intestinal metabolic demand had already increased. The other possible explanation is that the small intestinal resistance vessels were so dilated that they could not dilate any further in response to stimulation. If that is your explanation, then

you have to propose that the stomach is not maximally dilated, and can therefore dilate in response to cream instillation, whereas the small bowel cannot. This could also explain your results, in a manner more consistent with your hypothesis.

Dr. Boley, this is a very original study, and it asks a serious question. However, I am not certain that the absolute hemodynamic criteria for a steal have been established.

Edward T. Peter (Oakland, CA): I spent several years looking at splanchnic blood flow using a number of different methods, one of which was using flowmeters of various types. I have looked at the effect of perhaps a dozen different vasoactive substances on the splanchnic circulation, those that increased resistance, decreased flow, and so forth. Only one substance, histamine, had a paradoxic effect on the celiac and superior mesenteric circulation. If an infusion of histamine was administered, an increase in gastric circulation or celiac flow and a decrease in superior mesentery artery flow took place. Dr. Boley, have you looked at whether there was an increase in circulating histamine present, or have you infused histamine to see if this paradoxic effect occurred?

Richard G. Fiddian-Green (Worcester, MA): Dr. Boley, your data showed that in the face of stenosis of the celiac axis and of the superior mesenteric artery, vasodilatation of the gastric bed may induce a steal of blood from the bowel and hence acidosis in the small bowel. You propose that the acidosis in the small bowel might be the cause for pain in patients with these lesions. However, there are alternative explanations for the pain. In the patients we have studied, the cause of the postprandial pain may not have been due to acidosis in the small bowel induced by a steal away from it, as you have proposed, but might have been due in part to an impairment of the ability to dispose of gastric acid diffusing back from the lumen in the gastric wall. The cause of the postprandial acidosis in the wall of the stomach in our patients must have been related to underlying vascular disease, and might alternatively have been precipitated by a steal of blood from the stomach induced by the vasodilatation in the small bowel that occurs with eating. Dr. Boley, have you considered these explanations as possible causes for the pain in patients with mesenteric vascular disease?

Scott J. Boley (closing): Tonometry is a new technique, and it would be inappropriate for me to discuss it with Dr. Fiddian-Green in the audience, since he deserves both the credit for the technique and the responsibility for convincing you that it is a reliable test. I am already a believer.

Dr. Bulkley, in previous investigations of potential steal syndromes within the splanchnic circulation, we did use blood flow and pressure studies and showed the precise changes in pancreaticoduodenal, splenic, and gastric blood flows that you indicated. We used tonometry in this experiment to add another dimension to the previous blood flow studies.

You are absolutely correct that the intramural pH does not measure only blood flow. In fact, one of the advantages of the technique is that it is a measure not only of the oxygen that is provided to the bowel, but is also an indirect measure of the metabolic needs of the bowel. We obviously must begin to consider both of these factors.

In our experiments, the partial pressure of oxygen of the inspired air was kept constant, so we believed that we were measuring changes in blood flow. The relatively steady arterial partial pressure of oxygen supports this belief.

Dr. Peter, we did not investigate any aspect of the role of histamine in this study.

Dr. Fiddian-Green, you and I have discussed the question of the intramural pH in isolated celiac artery stenosis, and you have described patients in whom you used the test to indicate the presence of celiac artery obstruction and ischemia. The only problem we have with that is that neither your group nor ours has been able to demonstrate that the gastric intramural pH is a sensitive indicator of gastric ischemia. In our studies, we had to approach a 100 percent diminution of celiac artery flow before we were able to demonstrate a consistent change in the gastric intramural pH. Therefore, I find it very difficult to accept that the gastric intramural pH changes you have seen clinically are indicative of gastric ischemia. I accept your statement that perhaps the changes in the intramural pH are indicative of something other than altered blood flow. As you know, and as Dr. Bulkley knows, I do not believe you can have a steal syndrome in the splanchnic circulation unless you have fixed inflow through both the superior mesenteric artery and the celiac artery. If either one of them is patent, I do not believe a steal syndrome can be created.