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Simultaneous Measurement of Flow in the Superior Mesenteric Vein and Artery with Cine Phase-Contrast MR Imaging: Value in Diagnosis of Chronic Mesenteric Ischemia

Work in Progress¹

PURPOSE: To evaluate the use of measurements of blood flow in the superior mesenteric vein (SMV) and superior mesenteric artery (SMA) simultaneously acquired with phase-contrast cine magnetic resonance (MR) imaging for diagnosing chronic mesenteric ischemia.

MATERIALS AND METHODS: Simultaneous measurements of flow in the SMV and SMA were obtained in six healthy volunteers and eight patients with angiographically proved SMA stenosis (six asymptomatic, two symptomatic). Flow dynamics in both vessels were correlated with the degree of arterial disease seen at angiography and with the presence or absence of ischemic symptoms.

RESULTS: Postprandial SMV and SMA flow increased substantially less in patients with atherosclerosis than in volunteers. Comparison of simultaneous SMV and SMA flow measurements provided more information about collateral flow to and from the mesenteric circulation than did either the SMV or SMA flow measurement alone.

CONCLUSION: Simultaneous SMV and SMA flow measurement with cine phase-contrast MR imaging may be useful in diagnosing and understanding chronic mesenteric ischemia.

Index terms: Arteries, mesenteric, 792.266, 955.12944 • Blood, flow dynamics, 955.72 • Magnetic resonance (MR), phase imaging • Magnetic resonance (MR), vascular studies

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CHRONIC mesenteric ischemia, almost always due to atherosclerosis, results in a classic clinical triad of postprandial abdominal pain, food avoidance, and weight loss (1). Stenotic or obstructive lesions of the celiac axis, superior mesenteric artery (SMA), or inferior mesenteric artery (IMA) are typically demonstrated at angiography (1,2). Pain, which has been likened to that of angina, is thought to occur when the blood supply through the stenotic arteries fails to meet the oxygen requirements of the small intestine. Yet pain does not occur when food reaches the small intestine, at which time oxygen requirements are maximal; rather, it begins 15-20 minutes after food ingestion, when the bulk of the food remains in the stomach. For this reason, some investigators have proposed a "gastric steal" phenomenon, hypothesizing postprandial diversion of blood flow away from the small intestine to the full stomach, with resultant small bowel acidosis and pain (1-3).

To better understand the pathophysiologic mechanism of chronic mesenteric ischemia, researchers have attempted to quantitate changes in mesenteric blood flow in relation to meals and symptoms. We demonstrated previously that measurements of blood flow in the SMA obtained with cine phase-contrast magnetic resonance (MR) imaging enable differentiation of symptomatic patients with chronic mesenteric ischemia, asymptomatic patients with mesenteric atherosclerosis, and healthy volunteers (4). Burkart et al (5) suggested that measurements of SMV flow obtained with cine phase-contrast MR imaging might be more accurate in assessment of global mesenteric blood supply, particularly in the setting of SMA stenosis with collateral arterial supply from the celiac and IMA circulations. Major collateral venous pathways generally do not develop in the absence of portal hypertension or

other outflow obstruction. Thus, venous drainage of the small intestine occurs predominantly through a single vessel—the superior mesenteric vein (SMV)(6). While SMA flow measurements alone might overestimate the degree of mesenteric ischemia by failing to take collateral flow into account, SMV flow measurements should reflect supply both from the stenotic SMA and from anastomotic pathways.

This study compares measurements of postprandial SMV and SMA blood flow in healthy subjects with those in patients known to have various degrees of atherosclerotic disease involving the SMA, IMA, or celiac axis. The purpose is to determine whether SMV flow measurements obtained simultaneously with SMA flow measurements provide additional information in the diagnosis of chronic mesenteric ischemia.

MATERIALS AND METHODS

Subjects

The subjects included six healthy volunteers (five men, one woman; mean age, 35.3 years \pm 6.6; mean weight, 75.3 kg \pm 18.7) and eight patients (seven men, one woman; mean age, 63.9 years \pm 10.4) in whom arteriosclerosis had been documented angiographically. Six asymptomatic patients were divided by their angiographic findings into four groups. Group 1 consisted of two patients (mean weight, 78.6 kg \pm 34.1) with an SMA stenosis of less than 50% but with a normal celiac axis and IMA. Group 2 consisted of two patients (mean weight, 79.8 kg \pm 15.7) with an SMA stenosis of less than 50%, a normal celiac axis, and an IMA occlusion. Group 3 consisted of one patient (weight, 76.8 kg) with an SMA stenosis of greater than 70%

Abbreviations: ANOVA = analysis of variance, IMA = inferior mesenteric artery, SE = standard error, SMA = superior mesenteric artery, SMV = superior mesenteric vein, TE = echo time, TR = repetition time.

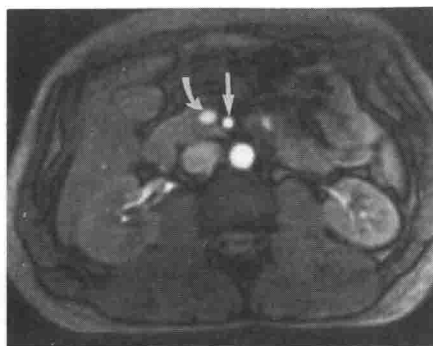


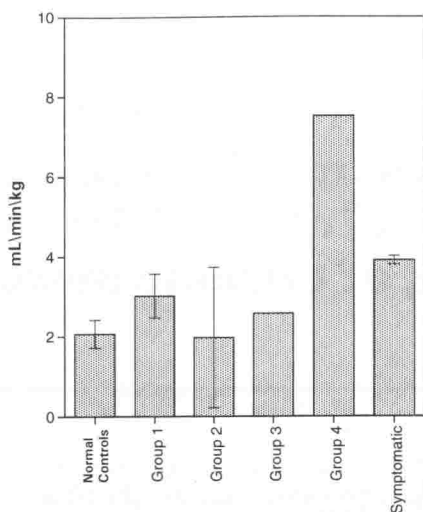
Figure 1. Axial localized image (gradient-recalled acquisition in the steady state, 34/13, 30° flip angle [TR msec/TE msec]) illustrates the typical level for cine phase-contrast MR acquisitions. Notice that the plane of section is at the level of the pancreatic head proximal to the first branch of the SMA (straight arrow) and distal to the last branch of the SMV (curved arrow). Also note the rounded configurations of the SMA and SMV, indicating that these vessels are close to perpendicular to the axial plane at this level.

but with a normal celiac axis and IMA. Finally, group 4 consisted of one patient (weight, 66.8 kg) with an SMA stenosis of greater than 70%, a celiac stenosis of greater than 50%, and an IMA occlusion. Two patients (mean weight, 57.5 kg \pm 11.2) demonstrating classic symptoms of chronic mesenteric ischemia were also evaluated, one with an SMA stenosis of greater than 90%, a normal celiac artery, and an occluded IMA and one with a 99% SMA stenosis, an occluded celiac artery, and a normal IMA.

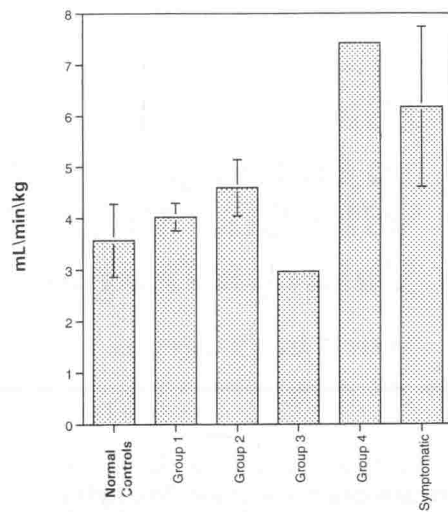
Data Acquisition and Flow Calculation

Subjects were imaged in a 1.5-T whole-body imager (Signa; GE Medical Systems, Milwaukee, Wis) after fasting for at least 8 hours and again 15, 30, and 45 minutes after ingestion of 240 mL of a liquid nutritional supplement (Ensure; Ross Laboratories, Columbus, Ohio). Previously described methods of performing velocity-encoded cine phase-contrast MR imaging were used to image the SMV and SMA vessels simultaneously throughout the cardiac cycle (4,7,8). For each subject, one 5-mm-thick axial section was obtained at the level of the proximal vessels (Fig 1) with the following parameters: cardiac or peripheral gating, respiratory compensation, 24-cm field of view, 16 phases per cardiac cycle, 256 \times 128 matrix, 25-msec repetition time (TR), 10-msec echo time (TE), 30° flip angle, and two signal averages. Peripheral gating was used when cardiac gating was technically difficult. Through-plane flow-encoding strength was 100 cm/sec after fasting and 150 cm/sec after meals. The flow-encoding strengths were chosen to be just high enough to avoid aliasing in the SMA.

Methods yielded both magnitude images like those of conventional cine MR imaging and phase-contrast images with

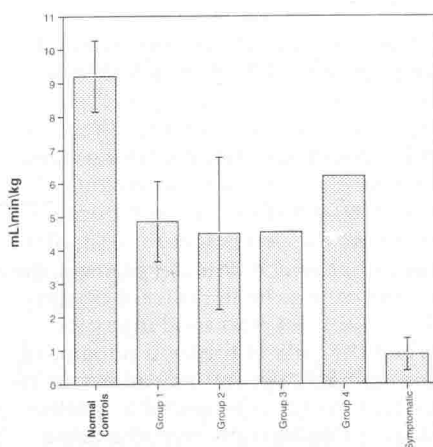


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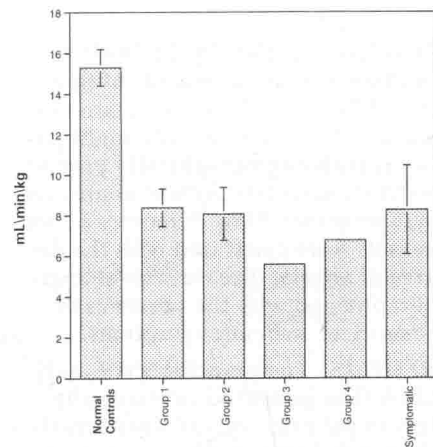


3.

Figures 2, 3. Flow rate per kilogram body weight (mean \pm standard error [SE]), after fasting, in the SMA (2) and SMV (3) for control subjects and patients in different groups.



4.



5.

Figures 4, 5. Flow rate per kilogram body weight (mean \pm SE), 30 minutes after a meal, in the SMA (4) and SMV (5) for control subjects and patients in different groups.

flow-dependent contrast in vessels. Flow velocity in the SMV and SMA was determined with techniques described by Pelc et al (7,8). Regions of interest drawn around the SMV or SMA on magnitude images were applied to velocity data from corresponding phase images with use of a computer program developed at the Department of Radiology, Stanford University School of Medicine. The rate of blood flow was calculated for each frame of the cardiac cycle. The average blood flow velocity for each vessel was then determined and normalized to body weight. For each postprandial time point, we also calculated the increases in blood flow in the SMA and SMV per kilogram of body weight and ratios comparing the increase in SMV flow to the increase in SMA flow.

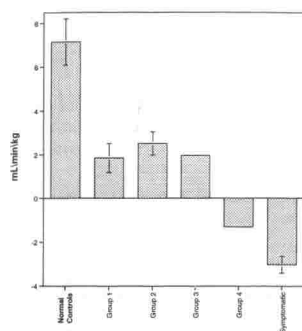
Statistical Analysis

Analysis of variance (ANOVA) (Statview II; Abacus Concepts, Berkeley, Calif) was used to compare weight-corrected SMV

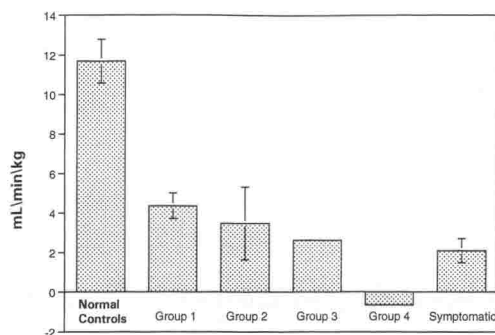
and SMA flow rates, as well as weight-corrected increases in SMV and SMA flow rates for different patient subgroups. Ratios of SMV flow increase to SMA flow increase 30 minutes after meals were also compared for the different subgroups with use of ANOVA.

RESULTS

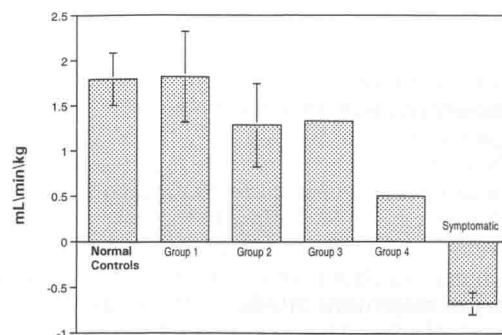
Figures 2 and 3 show the mean weight-corrected flow rates in the SMA and SMV, respectively, after fasting. The mean fasting SMA flow in symptomatic patients was 3.90 mL/min/kg \pm 0.11 (mean \pm SE), which is significantly ($P < .05$) different from that of the asymptomatic patient in group 4 (7.52 mL/min/kg) but not significantly different from that of asymptomatic patients in group 1 (3.01 mL/min/kg \pm 0.55), group 2 (1.97 mL/min/kg \pm 1.76), and group 3 (2.57 mL/min/kg) or healthy volun-



6.



7.



8.

Figures 6–8. (6, 7) Increase in flow rate per kilogram body weight (mean \pm SE), at 30 minutes after a meal, in the SMA (6) and SMV (7) for the control subjects and patients in different groups. (8) Ratio of the increase in SMV flow to the increase in SMA flow (mean \pm SE) at 30 minutes after a meal.

teers ($2.06 \text{ mL/min/kg} \pm 0.35$). Similarly, there was no significant difference among fasting SMV flow values in healthy subjects ($3.57 \text{ mL/min/kg} \pm 0.71$), asymptomatic patients (group 1, $4.02 \text{ mL/min/kg} \pm 0.27$; group 2, $4.59 \text{ mL/min/kg} \pm 0.55$; group 3, 2.96 mL/min/kg ; group 4, 7.42 mL/min/kg), and symptomatic patients ($6.17 \text{ mL/min/kg} \pm 1.57$). Figures 4 and 5 summarize the mean weight-corrected flow rates in the SMA and SMV, respectively, at 30 minutes after a meal. These rates in symptomatic patients were significantly different from those in healthy subjects but not significantly different from those in patients in groups 1–4.

Differences in flow dynamics among the control group and the different groups of patients were most pronounced 30 minutes after meals, at which time increases in weight-corrected flow in the SMA (Fig 6) and SMV (Fig 7) were significantly less ($P < .05$) for all patient groups than for healthy volunteers. The weight-corrected increase in SMA flow at 30 minutes after a meal in symptomatic patients was significantly different from that in volunteers and patients in groups 1 and 2 but not significantly different from that in patients in groups 3 and 4. The weight-corrected increase in SMV flow at 30 minutes after a meal in symptomatic patients was significantly different from that of healthy volunteers but not significantly different from that of any of the other patient groups. Figure 8 shows the ratios of the increase in SMV flow to the increase in SMA flow 30 minutes after meals for healthy subjects and patients in the different groups. Notice that with an increase in severity of disease in the mesenteric circulation, there was a trend toward a decrease in the ratio. The ratio of the increase in SMA flow to the increase in SMV flow at 30 min-

utes after a meal in the symptomatic patients was significantly different from that of all the other subject groups, except for the patient in group 4.

DISCUSSION

Like SMA flow, SMV flow showed a physiologic increase after meals in healthy subjects (9–13). In patients with atherosclerosis, postprandial increases in SMV flow diminished as the degree of SMA stenosis increased (patients in groups 3 and 4 and symptomatic patients) and as the number of stenotic mesenteric arteries rose (patients in groups 2 and 4 and symptomatic patients).

Increases in both SMA and SMV flow after meals were dampened by progressively severe atherosclerotic disease. However, in isolation, these measurements reveal relatively little about the complex pathophysiologic mechanism of mesenteric ischemia. When SMV and SMA flow changes are compared, additional information can be gained about the direction and significance of collateral flow pathways. Flow dynamics in our patients were best appreciated by using the ratio of the increase in SMV flow to the increase in SMA flow 30 minutes after meals. In healthy subjects and in patients with significant stenoses in either none (group 1) or only one (groups 2 and 3) of the mesenteric vessels, the ratio was greater than 1. This indicates that, in these groups, increased blood flow through the small bowel after meals resulted from more than just an increase in SMA flow; collateral flow from the celiac or IMA circulation must have occurred to explain the greater rise in SMV as compared to SMA flow.

With increasing severity of mesenteric disease, the ratio of the increase in SMV flow to the increase in SMA

flow decreased. In fact, the ratio in symptomatic patients was significantly lower than that in any other subject group, except for the patient with disease in three vessels (group 4). In the two symptomatic patients, both with significant stenoses of two mesenteric arteries, a negative ratio was seen because proximal SMA flow in these patients decreased after meals, coinciding with onset of abdominal pain. SMV flow in these patients increased slightly at 30 minutes after meals. One explanation for these findings would be that blood flow from the SMA to the gastric circulation was diverted after the meal, eventually leading to vasoconstriction in the small bowel vessels, resulting in decreased flow measurements in the proximal SMA (gastric steal). Concomitantly, some degree of collateral supply from distal IMA or celiac branches to the superior mesenteric circulation may have resulted in the increase in SMV flow. The presence of symptoms in these patients suggests that oxygen uptake by the small bowel remained inadequate despite some working collateral pathways.

The single asymptomatic patient with disease in three vessels (group 4) had a decrease in both SMA and SMV flow at 30 minutes after the meal, with a ratio that was positive but less than 1. These results indicate that, while a decrease in SMA flow occurred proximally after meals in this patient owing to gastric steal, there was insufficient collateral flow from the branches of the stenotic celiac artery or IMA to the distribution of the SMA to compensate for this steal phenomenon, leading to a decrease in SMV flow also at 30 minutes after eating. The absence of symptoms suggests that oxygen uptake by the small bowel was adequate in this patient despite a net decrease in blood flow to the small bowel after the meal.

Our results indicate that cine phase-contrast MR imaging is a promising technique for evaluating chronic mesenteric ischemia. Information gained from comparing SMV to SMA flow dynamics is likely to be of value. In this preliminary study, the ratio of the increase in SMV flow to the increase in SMA flow at 30 minutes after a meal correlated well with the number of mesenteric arteries with significant stenoses. However, flow data alone may not be adequate for distinguishing symptomatic patients from asymptomatic patients with significant stenoses of more than one mesenteric artery. Study of a larger number of patients will be necessary to confirm trends seen in this preliminary study. ■

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