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Chronic Mesenteric Ischemia: Evaluation with Phase-Contrast Cine MR Imaging[1]

PURPOSE: **To compare superior mesenteric artery (SMA) blood flow in healthy volunteers and patients with stenoses in the fasting state and after food intake by using phase-contrast (PC) cine magnetic resonance (MR) imaging.**

MATERIALS AND METHODS: **Ten healthy subjects, four asymptomatic patients (three with 50% stenosis, one with 70% stenosis), and one symptomatic patient (with 80% stenosis) were studied. All subjects were studied after fasting at least 8 hours and 15, 30, and 45 minutes after ingesting a standard meal.**

RESULTS: **In healthy volunteers, SMA blood flow at all postprandial intervals increased significantly compared with that obtained after fasting ($P \leq .0005$). The percentage change in SMA blood flow 30 minutes after food intake provided the best distinction between the healthy subjects, the asymptomatic patients, and the symptomatic patient.**

CONCLUSION: **Cine PC MR imaging is an effective, noninvasive technique for measuring SMA blood flow.**

Index terms: Arteries, mesenteric, 792.266, 955.12944 • Blood, flow dynamics, 955.72 • Intestines, ischemia, 955.266, 955.761 • Magnetic resonance (MR), phase imaging • Magnetic resonance (MR), vascular studies

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ATHEROSCLEROSIS, which causes stenosis or occlusion of the proximal portions of the mesenteric vessels, is responsible for more than 95% of cases of chronic mesenteric ischemia. Chronic mesenteric ischemia produces a well-defined clinical syndrome consisting of postprandial abdominal pain, food aversion, and weight loss (1). The pathophysiology of this syndrome, however, is not well understood. Because pain occurs only with food ingestion, it seems likely it is caused by a discrepancy between the increased oxygen demand of the small intestine and the ability of the blood supply to keep up with the metabolic requirement. However, pain usually occurs 15–20 minutes after food intake, well before the food has reached the small intestine. Therefore, some researchers postulate that food ingestion increases demand for gastric blood flow, thereby diverting the blood from the remaining gastrointestinal tract and creating a "gastric steal" phenomenon (1,2). Subsequent small intestine acidosis then causes the typical abdominal pain.

Demonstration of stenosis or occlusion of at least two of the major vessels at angiography is the most common confirmatory test used in chronic mesenteric ischemia. However, many investigators (1,2) believe angiography alone is insufficient to establish the diagnosis of arterial insufficiency or chronic mesenteric ischemia. 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 [IMA]). Because of this rich arterial supply, chronic stenosis or even occlusion of all three major mesenteric arteries frequently occurs without abdominal symptoms (1,2).

Duplex sonography has been used to study the blood flow in mesenteric vessels, and the physiologic increase in superior mesenteric artery (SMA) blood flow after food intake has been demonstrated in healthy volunteers (3–7). However, to our knowledge, there are no clinical data suggesting that abnormal mesenteric blood flow alone, with or without bolus food challenge, is a good predictor of chronic mesenteric ischemia. In addition, duplex sonography is operator dependent, and overlying bowel gas or excess adipose tissue may preclude an optimal examination. Therefore, there is a need for a better means to accurately diagnose chronic mesenteric ischemia.

Phase-contrast (PC) cine magnetic resonance (MR) imaging can depict motion and flow throughout the cardiac cycle and can be used to quantify flow velocities and volumes. A version of this technique has been implemented with our MR imagers, and its accuracy was validated in vivo in a canine model (8). The purpose of this study was to evaluate the blood flow in the SMA in healthy volunteers and patients with angiographically proved SMA stenosis, in the fasting state and after bolus food challenge, by using cine PC MR imaging. This is an initial study evaluating the feasibility of using MR imaging to measure blood flow in the mesenteric circulation in vivo in humans.

MATERIALS AND METHODS

Cine PC MR Imaging

The cine PC technique employed has been previously described (8–10). Briefly, cine PC MR imaging combines the flow-dependent contrast of PC MR imaging with the ability to produce images throughout the cardiac cycle. Cine PC MR imaging produces images in which con-

Abbreviations: ANOVA = analysis of variance, IMA = inferior mesenteric artery, PC = phase-contrast, ROI = region of interest, SMA = superior mesenteric artery.

trast is related to flow velocity, as well as magnitude images such as those of conventional cine MR imaging. The images can be interpreted qualitatively to demonstrate the presence, magnitude, and direction of flow. In addition, the data obtained can be used to provide estimates of flow velocity, volume flow rate, and displaced volumes.

Subject Preparation and Imaging Protocols

Ten healthy subjects (eight men, two women; mean age, 34.1 years ± 5.2) were imaged with a 1.5-T whole-body imager (Signa; GE Medical Systems, Milwaukee, Wis) after fasting for at least 8 hours. Velocity-encoded cine PC MR images of the SMAs were obtained in 5-mm-thick axial sections by using cardiac or peripheral gating, respiratory compensation, flow-encoding strength of 100 cm/sec through plane, a 24-cm field of view, 16 phases per cardiac cycle, a 256 × 128 matrix, a repetition time of 25 msec, an echo time of 10 msec, a 30° flip angle, and two signals averaged.

After the fasting image was obtained, one can (240 mL) of Ensure (Ross Laboratories, Columbus, Ohio) was given to each volunteer. Cine PC MR images of the SMAs were then obtained at 15, 30, and 45 minutes after food intake by means of the same parameters described above with the exception of a flow-encoding strength of 150 cm/sec. Five men (mean age, 63.6 years ± 6.7) with angiographically proved SMA stenosis, four without symptoms and one with classic symptoms of chronic mesenteric ischemia, underwent imaging with the same protocol.

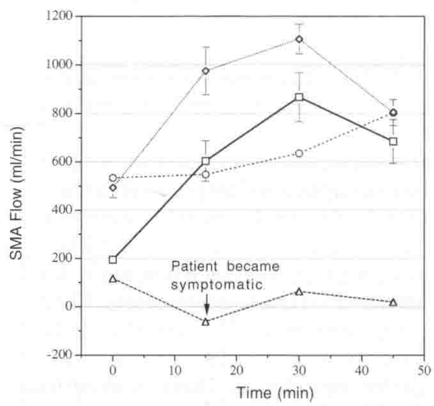
After data acquisition, SMA blood flow was determined by means of analysis of regions of interest (ROIs) over the SMAs with the method described by Pelc et al (8). In brief, before ROI selection, display window level and window width were both set to half the maximum intraluminal signal intensity in the magnitude image. ROIs were then drawn on the magnitude images of the cine set and applied to the velocity data. When substantial vessel motion during the cardiac cycle was seen, separate ROIs were selected for each frame. The rate of blood flow during each of the 16 frames of the cardiac cycle and average rate of blood flow were calculated.

Statistical Analysis

The percentage change in SMA blood flow after food intake, defined as (postprandial SMA blood flow − fasting SMA blood flow) × 100%/fasting SMA blood flow, was calculated for all time intervals for all subjects. Analysis of variance (ANOVA) (Statview II; Abacus Concepts, Berkeley, Calif) was used to identify statistically significant differences in the postprandial SMA blood flow and the percentage change in postprandial SMA blood flow between the healthy volunteers and the asymptomatic and symptomatic patients. A two-tailed, paired Student *t* test was used to compare the pre- and postprandial SMA blood flow at different time intervals.

RESULTS

The results of the SMA blood flow measurements and the percentage change in postprandial SMA blood flow are summarized in Figures 1 and 2, respectively. In the healthy subjects, the mean fasting SMA blood flow ± standard error was 217 mL/min ± 25. Fifteen minutes after food intake, the mean SMA blood flow increased significantly, to 604 mL/min ± 83. SMA blood flow reached a peak at 30 minutes (868 mL/min ± 101), an increase of 361% ± 45 from the fasting state. Forty-five minutes after food intake, SMA blood flow dropped to 685 mL/min ± 91. The SMA blood flow measurements at 15, 30, and 45 minutes were all significantly higher than the corresponding measurements during the fasting state (two-tailed, paired Student *t* test, P = .0005, .0001, and .0001, respectively).



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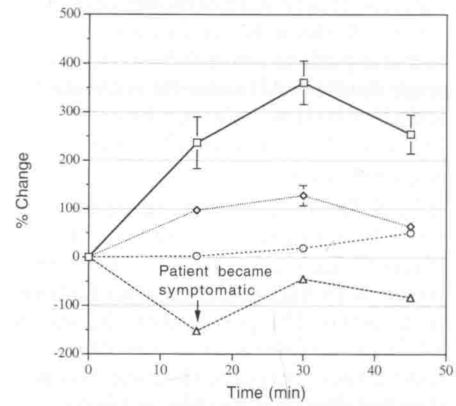
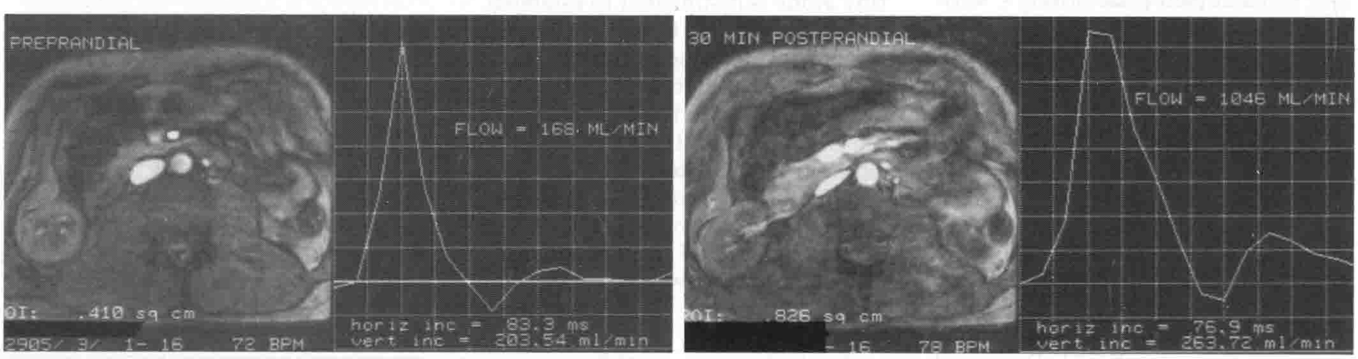


2.

Figures 1, 2. **(1)** SMA blood flow versus time after food intake in healthy subjects and patients with SMA stenosis. Note the higher baseline blood flow in the patients with 50% and 70% stenosis and the large increase in SMA blood flow in the healthy subjects and patients with 50% stenosis. **(2)** Percentage change in SMA blood flow versus time after food intake in healthy subjects and patients with SMA stenosis. Note the percentage change in postprandial SMA blood flow in the healthy subjects is greater than that in the patients. The difference in postprandial blood flow between the four groups is best seen at 30 minutes. □ = healthy subjects, ◇ = asymptomatic patients with 50% stenosis, ○ = asymptomatic patient with 70% stenosis, △ = symptomatic patient with 80% stenosis.



a. b.

Figure 3. Cine PC MR images obtained with cardiac gating and plots of SMA blood flow in a healthy subject before **(a)** and 30 minutes after **(b)** food intake. Note the low SMA blood flow and the reverse early diastolic flow in the fasting state. There is a marked increase in SMA blood flow 30 minutes after food intake, and the reverse early diastolic flow has disappeared.

Three of the four asymptomatic patients had 50% stenosis of the SMA, and one had 70% stenosis. The symptomatic patient had 80% SMA stenosis. The mean fasting SMA blood flow of the asymptomatic patients was significantly higher than that of the healthy subjects ($P < .05$). The IMAs were completely occluded in these patients. The symptomatic patient had an occlusion of the distal abdominal aorta, but the fasting SMA blood flow was not significantly different ($P > .05$) from that of the healthy subjects.

As seen in Figures 1 and 2, the percentage change in SMA blood flow 30 minutes after food intake provides the best separation between the groups. Postprandial blood flow in each of the three patient groups is significantly different from that of the healthy subjects with this parameter ($P < .05$ by using ANOVA). However, the differences between the patient groups are not statistically significant. The absolute change in SMA blood flow in the patients with 50% stenosis after food intake is very similar to that of the healthy subjects (Fig 1). However, due to the greater fasting SMA blood flow in these patients, the percentage changes in SMA blood flow after food intake differ from those of the healthy subjects, reaching statistical significance at 30 and 45 minutes (Fig 2). In the patient with 70% stenosis, the percentage increase in postprandial SMA blood flow was much less than that in healthy subjects, reaching a peak of only 51% at 45 minutes.

In the symptomatic patient, there was net retrograde SMA flow of 61 mL/min 15 minutes after food intake. The SMA flow peaked at 64 mL/min 30 minutes after food intake; this was still lower than the fasting flow rate of 117 mL/min in this patient. The patient complained of mild abdominal discomfort starting at about 10 minutes after food intake; this discomfort persisted for at least 1 hour. The percentage change in postprandial SMA blood flow at all three time intervals was significantly different ($P < .05$) from the corresponding value in the healthy subjects (Fig 2). Note the decrease in postprandial SMA blood flow at all three time intervals compared with that of the fasting state (Fig 1).

Qualitatively, the triphasic waveforms of the SMA blood flow in the healthy subjects and the patients with 50% stenosis were well demonstrated with cine PC MR imaging (Fig 3). Both systolic and diastolic SMA blood flows increased after food intake. In the asymptomatic patient with 70% stenosis (Fig 4), the rise in the SMA blood flow in systole was slow and dampened in the fasting state. After meal ingestion, the systolic rise in the SMA blood flow in the patients with 50% and 70% stenosis is earlier and higher. In the symptomatic patient, the systolic rise in postprandial SMA blood flow was lower than that in the fasting state (Fig 5), and loss of the normal distinct triphasic flow pattern also occurred after meal ingestion.

DISCUSSION

Our results show that the physiologic increase in SMA blood flow in healthy subjects is well demonstrated with cine PC MR imaging. In all three groups of patients with SMA stenosis, the percentage change in postprandial SMA blood flow at 30 minutes was significantly less than that seen in the healthy subjects. The percentage change in SMA flow in response to a standard meal in the four subgroups studied (healthy subjects, asymptomatic patients with 50% stenosis, asymptomatic patient with 70% stenosis, and symptomatic patient with 80% stenosis) demonstrates a gradation consistent with the gradation of severity of the abnormalities demonstrated at angiography and the patient's symptoms. Obviously, a larger study with more patients is required to determine whether this gradation of changes of SMA flow response is consistently demonstrated.

The symptomatic patient demonstrated net retrograde flow in the SMA 15 minutes after food intake, the time of maximum symptoms in most patients. Postprandial SMA blood flow in this patient was decreased

a.

Figure 4. Asymptomatic patient with 70% SMA stenosis. **(a)** Lateral aortogram shows stenosis of the SMA and lack of opacification of the IMA. **(b, c)** Cine PC MR images obtained with cardiac gating before **(b)** and after **(c)** food intake. Note the slow and dampened rise in SMA flow in early systole in the fasting state. The systolic rise is earlier and higher at 30 minutes after food intake.

b.

c.

compared with that in the fasting state even at 45 minutes. The decrease in SMA blood flow coincided with the onset and duration of symptoms in this patient.

Proponents of the gastric steal theory hypothesize that ingestion of food initially creates gastric hyperemia and a decrease in vascular resistance in the gastric circulation (11). This leads to a difference in vascular resistance between the gastric and SMA circulation and results in diversion of blood flow from the distal SMA through the collateral circulation to the gastric circulation. The observation that blood flow in the proximal SMA increases in healthy subjects and asymptomatic patients with SMA stenosis soon after food ingestion, before the food reaches the small bowel, supports this theory.

This observation is consistent with the hypothesis that in healthy subjects and patients with sufficient reserve in the splanchnic circulation, the increase in proximal SMA blood flow is largely diverted to the stomach, and the proportion of blood reaching the distal SMA from the proximal SMA is less than that in a fasting state. It can further be hypothesized that in patients with insufficient reserve in the splanchnic circulation, the diversion of blood from the distal SMA causes mesenteric ischemia, which results in pain and vasoconstriction in the distal SMA and the collateral vessels. The vascular resistance transmitted to the proximal SMA is, therefore, higher, and decreased blood flow from the aorta to the proximal SMA and increased reverse diastolic flow may occur. Our observation of reduced postprandial blood flow in the symptomatic patient is consistent with this hypothesis.

The observation that the patients with 50% and 70% SMA stenosis had greater fasting SMA blood flow than the healthy subjects is also consistent with known physiologic characteristics of the mesenteric circulation. This probably resulted from occlusion of the IMAs in these patients, which increased blood flow in the SMAs to supply, via collateral vessels, the tissues normally supplied by the IMAs. In the symptomatic patient, the fasting SMA blood flow was not significantly different from that of the healthy subjects, even with occlusion of the inferior abdominal aorta. This likely indicates a diminished reserve in the splanchnic circulation of this patient, so SMA blood flow cannot increase properly to compensate for the occluded IMA. When the splanchnic circulation was stressed further after ingestion of food, the gastric steal phenomenon occurred with a resulting decrease in SMA blood flow in this patient.

In conclusion, our preliminary data indicate that the percentage change in SMA blood flow 30 minutes after meal intake appears to be a promising criterion for differentiating healthy subjects from patients with ischemia. Our preliminary results are consistent

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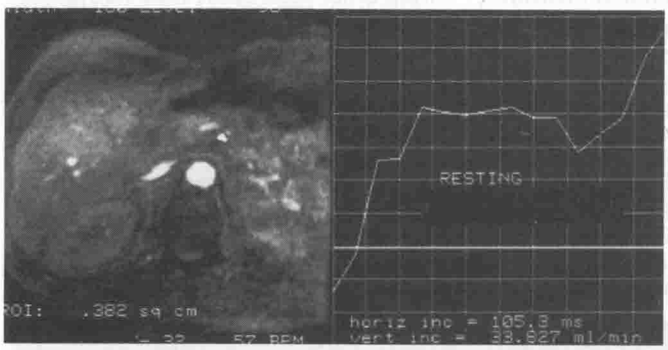

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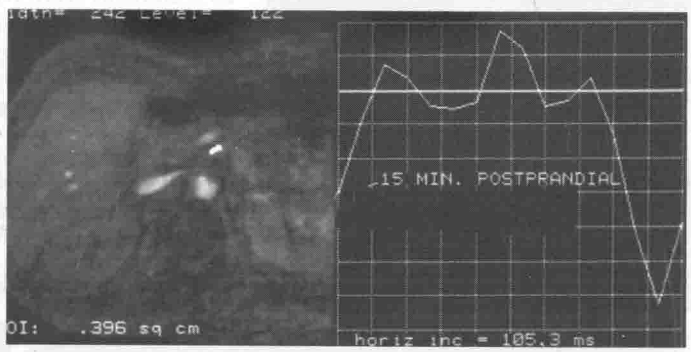

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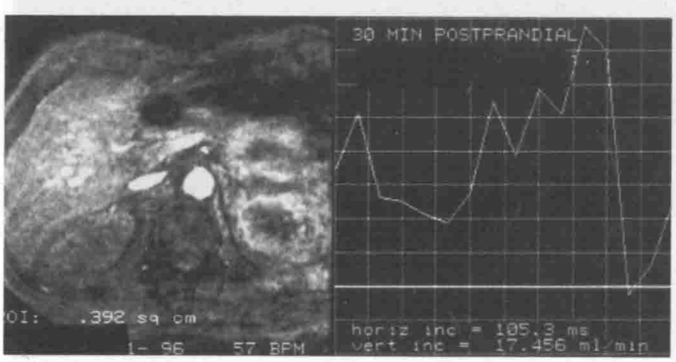

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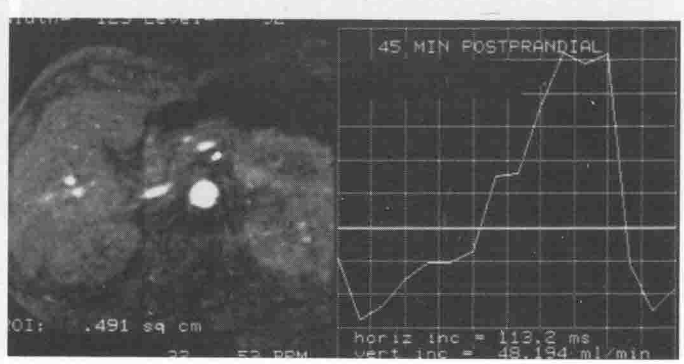

e.

Figure 5. Symptomatic patient with 80% SMA stenosis. **(a)** Lateral aortogram shows stenosis of the SMA and occlusion of the distal abdominal aorta. **(b–e)** Cine PC MR images obtained with peripheral gating before **(b)** and 15 **(c)**, 30 **(d)**, and 45 **(e)** minutes after food intake. Note the loss of the normal triphasic flow pattern after meal ingestion and the increased reverse diastolic flow.

with the hypothesis that abdominal pain in chronic mesenteric ischemia is caused by a gastric steal phenomenon. One important question to be answered in the future is whether SMA blood flow measurement alone is adequate as a prognostic indicator for selecting patients for treatments such as revascularization surgery. Further study with a larger number of patients is warranted. ■

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