**Introduction:**

Chronic mesenteric ischemia (CMI) is a disease caused by inadequate blood flow to the small intestine due to underlying stenotic and occlusive diseases affecting mesenteric vasculature. In normal individuals, an increase in mesenteric blood flow is necessary for satisfying increased metabolic demands of the gastrointestinal tract after a meal1–3. In patients with CMI, this blood flow response is stunted by underlying pathophysiology, thus, causing dull postprandial abdominal pain 15-30 minutes after ingestion of a meal with pain continuing up to 2 hours4–6. This subsequently leads to fear of food, severe weight loss, and malnutrition4. Since the onset of pain occurs relatively rapidly, well before food has entered the small intestine, it is hypothesized that blood is diverted from the distal intestines to the gastric region where metabolic demand exists. This creates a “gastric steal” phenomenon, leading to hypoperfusion of the small intestine, subsequently causing acidosis and pain.

The primary etiology of CMI is atherosclerotic lesions (approximately 90% of cases) affecting proximal segments of the celiac, superior mesenteric, and inferior mesenteric arteries4. However, CMI may also arise from other, less common etiologies such as median arcuate ligament syndrome (MALS), various forms of vasculitis, fibromuscular dysplasia, or arterial dissection. It is commonly recognized that at least 2 of the main mesenteric arteries must be compromised to result in true CMI, due to the pre-existing collateral network that exists between the main mesenteric vessels as well as other compensatory collaterals that may form in response to chronic stenosis or occlusion4–8. Due to the limited prevalence of the disease and the symptoms that CMI shares with other, more common abdominal pathologies, diagnosis of ischemia is difficult, often requiring a high index of clinical suspicion.

Diagnostic imaging, specifically contrast-enhanced CT, has been successful in both identifying and locating regions of stenosis and occlusion, as well as simultaneously excluding other abdominal pathologies. Several studies have previously shown that CT angiography (CTA) has sensitivities and specificities greater than 90% for diagnosing atherosclerotic CMI. However, CTA does not provide functional information regarding blood velocity and blood flow, a crucial physiological component of the disease. Other imaging modalities have attempted to provide functional hemodynamic information to assist in the diagnosis in CMI, specifically duplex ultrasonography. While duplex sonography , it is prone to error due to the curvature of the mesenteric vessels, possible bowel gas overlying the vessels of interest, as well as systematic errors when measuring blood flow, such as angle of insonation used and manual measurement of cross-sectional area of blood vessels9,10. MR flow techniques have recently been proposed as a way to functionally analyze patients with suspected ischemia by measuring blood flow before and after a meal challenge. MR flow has the added benefit of simultaneous functional and anatomical assessment (MR Angiogram). Previous studies have looked at cine phase-contrast (3D MR flow) as a method to functionally and anatomically evaluate mesenteric vasculature in patients with suspected CMI with promising results. This study further investigates the use of 4D flow MRI to non-invasively assess the hemodynamics of mesenteric circulation in patients with suspected CMI.

**Materials and Methods:**

The data had already been collected. Any patient with suspected ischemia (referred from vascular surgery) was imaged with the ischemia protocol, including a pcvipr scan.

Flow was measured in 9 vessels before and after meal challenges for 19 subjects suspected of CMI and 6 controls. Post-prandial flow increased significantly in the supraceliac aorta, superior mesenteric artery, superior mesenteric vein, and portal vein. The flow increase was drastically less in patients with CMI. This demonstrates the potential for 4D flow MRI in assisting the challenging diagnosis of CMI.

When computing flow, all velocity images were viewed to ensure no aliasing occurred.

**Results:**

### SCAo

40 healthy control (of 40 total datasets), 26 (of 26) negative diagnosis, and 10 (of 12) CMI measurements were made in the SCAo. In the healthy control subjects, the SCAo blood flow increased significantly 20 minutes after a meal (p = 5.62e-05, Cohen’s d = 0.381) as did the negative diagnosis group (p = 0.006, d = 0.641). In the CMI group, the average SCAo blood flow remained relatively constant. The postprandial SCAo flow for the CMI group was significantly less than controls (p = 0.013, d = 0.875) and significantly less than the negative diagnosis group (p = 0.018, d = 0.825). Average percent changes in flow ± 1 standard deviation for the controls, negative diagnosis, and CMI groups were 15.7% ± 14.8, 41.5% ± 90.6, -2.57% ± 12.1 respectively. The average percent changes in flow for the negative diagnosis group were not significantly different from the controls. However, the average percent changes for the CMI group were significantly less than both the negative diagnosis group (p = 0.008, d = 1.15) and the control group (p = 0.022, d = 0.956).

### IRAo

39 healthy control, 24 negative diagnosis, and 10 CMI measurements were made in the IRAo. In all groups, the IRAo blood flow remained relatively constant after a meal. The postprandial IRAo flow for the negative diagnosis group was significantly higher than controls (p = 0.043, d = 0.543). Average percent changes in flow for the controls, negative diagnosis, and CMI groups were -9.99 ± 27.0, 36.2 ± 81.0, -3.16 ± 27.1 respectively. The only significant difference between groups was a significantly higher percent change in flow in the negative diagnosis group compared to controls (p = 0.047, d = 0.559).

### LRA

38 healthy control, 26 negative diagnosis, and 12 CMI measurements were made in the LRA. In all groups, the LRA blood flow remained relatively constant after a meal. There was no significant difference between groups in the average pre- and postprandial LRA flow values. Average percent changes in flow for the controls, negative diagnosis, and CMI groups were 3.58% ± 15.4, 2.03% ± 21.0, -19.9% ± 26.1 respectively. The average percent changes in flow for the negative diagnosis group were not significantly different from the controls. For the CMI group, the average percent change in LRA flow was not significantly less than controls (p = 0.080, d = 0.774) or the negative diagnosis group (p = 0.108, d = 0.654).

### RRA

40 healthy control, 22 negative diagnosis, and 10 CMI measurements were made in the RRA. In all groups, the RRA blood flow remained relatively constant after a meal. There was no significant difference between groups in the average pre- and postprandial RRA flow values. Average percent changes in flow for the controls, negative diagnosis, and CMI groups were 6.97% ± 17.5, -0.95% ± 19.0, -35.7% ± 37.1 respectively. The average percent changes in flow for the negative diagnosis group were not significantly different from the controls. For the CMI group, the average percent change in RRA flow was not significantly less than controls (p = 0.060, d = 1.04) or the negative diagnosis group (p = 0.146, d = 0.466).

### SMA

40 healthy control, 26 negative diagnosis, and 12 CMI measurements were made in the SMA. In the healthy control subjects, the SMA blood flow increased significantly after a meal (p = 5.20e-06, d = 1.26) as did the negative diagnosis group (p = 0.016, d = 0.491). The CMI group did not see a significant increase in SMA flow (p = 0.193, d = 0.465). There was not significant difference in pre- and postprandial SMA flow values between groups. Average percent changes in flow for the controls, negative diagnosis, and CMI groups were 98.8% ± 80.7, 59.6% ± 69.3, 23.5% ± 32.7 respectively. The average percent changes in flow for the negative diagnosis group were not significantly different from the controls. Likewise, the average percent change in flow for the CMI group was not significantly different from that of the negative diagnosis group. However, the average percent change in flow for the CMI group was significantly less than controls (p = 0.003, d = 0.865).

### CA

38 healthy control, 24 negative diagnosis, and 12 CMI measurements were made in the CA. The CA blood flow did not increase significantly after a meal for any group. The preprandial CA flow in the negative diagnosis was significantly higher than controls (p = 0.023, d = 0.581) but postprandial CA flow in the negative diagnosis group was not significantly different from controls (p = 0.070, d = 0.468). There was no significant differences in pre- and postprandial values between the other groups. Average percent changes in flow for the controls, negative diagnosis, and CMI groups were -3.73% ± 18.9, 0.93% ± 35.6, 4.52% ± 8.53 respectively. None of the groups showed any significant difference in average percent change in the CA.

### SMV

38 healthy control, 26 negative diagnosis, and 12 CMI measurements were made in the SMV. In the healthy control subjects, the SMV blood flow increased significantly after a meal (p = 2.51e-08, d = 2.10) as did the negative diagnosis group (p = 3.05e-06, d = 1.67). The CMI group did not see a significant increase in SMV flow (p = 0.120, d = 0.777). For the CMI group, the average preprandial flow in the SMV was significantly higher than that of controls (p = 0.040, d = 0.905) but not significantly higher than the negative diagnosis group (p = 0.074, d = 0.665). There was no significant differences in preprandial flow between the controls and the negative diagnosis group. Also, there was no significant differences in average postprandial SMV flow between any of the groups. Average percent changes in flow for the controls, negative diagnosis, and CMI groups were 133% ± 80.2, 178% ± 147, 40.3% ± 55.6 respectively. The average percent changes in flow for the negative diagnosis group were not significantly different from the controls. However, the average percent change in flow for the CMI group was significantly less than controls (p = 0.008, d = 0.944) and significantly less than the control group (p = 0.009, d = 0.875).

### SV

35 healthy control, 26 negative diagnosis, and 12 CMI measurements were made in the SV. The SV blood flow did not increase significantly after a meal for any group. There was no statistically significant differences in the pre- and postprandial SV flow values between groups. Average percent changes in flow for the controls, negative diagnosis, and CMI groups were -4.76% ± 32.3, -3.77 ± 36.0, -11.7% ± 19.4 respectively. There was no statistically significant differences in the average percent change between groups.

### PV

38 healthy control, 26 negative diagnosis, and 12 CMI measurements were made in the PV. In the healthy control subjects, the PV blood flow increased significantly after a meal (p = 1.17e-05, d = 1.137) as did the negative diagnosis group (p = 1.60e-05, d = 1.690). There was no statistically significant differences in the pre- and postprandial PV flow values between groups. Average percent changes in flow for the controls, negative diagnosis, and CMI groups were 56.7% ± 47.9, 72.1% ± 50.4, 11.8% ± 30.9 respectively. The average percent changes in flow for the negative diagnosis group were not significantly different from the controls. However, the average percent change in flow for the CMI group was significantly less than controls (p = 0.018, d = 0.788) and significantly less than the control group (p = 0.006, d = 1.02).

**Conclusion:**

Methods to automatically segment need to be explored. This will reduce the time of obtaining flow values as well as help maintain consistency of segmentation between users.

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