# Pulsatility and Resistivity Indices in Mesenteric Vasculature in Patients Suspected of Chronic Mesenteric Ischemia using 4D Flow MRI

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## Synopsis

**Chronic mesenteric ischemia (CMI) causes blood flow reduction in the intestines, often due to atherosclerosis. This study uses 4D flow MRI to quantify and compare pulsatility (PI) and resistivity indices (RI) in mesenteric vasculature in patients with a suspicion of CMI (N=19) and healthy individuals (N=20). PI and RI were measured in 9 mesenteric vessels before and after meal ingestion. In patients with CMI, aortic PI were significantly decreased both before and after a meal compared to controls, while postprandial SV and SMV PI values were significantly increased. RI values were not significantly different between groups.**

## Introduction

Chronic mesenteric ischemia (CMI) is a rare disease caused by reduced blood flow to the intestines, often the result of atherosclerotic lesions occluding proximal segments of mesenteric vasculature (more than 95% of cases)[1]. CMI is characterized by postprandial abdominal pain, occurring 15-60 minutes after meal ingestion. It is theorized that the relatively early onset of pain (well before food has reached the small bowel) is caused by diversion of blood flow from the superior mesenteric artery (SMA) to the celiac artery (CA) to supplement increased gastric blood flow demand, termed “gastric steal” phenomenon[2]. In individuals with compromised splanchnic circulation, decreased flow in the SMA leads to downstream vasoconstriction, ischemia, and pain. 2D-PC has shown reduced postprandial splanchnic blood flow responses in CMI patients[3–5]. We recently introduced 4D Flow MRI with a meal challenge for a more comprehensive hemodynamic analysis for CMI[6,7]. Here, we expand the analysis of postprandial flow responses to measures of pulsatility and resistivity indices, which have been shown to reflect downstream vascular resistance in other vascular territories.

## Methods

In this patient-compliant and IRB-approved study, 20 healthy volunteers (age range 19-73y, mean=44y, females=8) and 19 patients (age range 21-86y, mean=50y, females=14) with a suspicion of CMI were imaged on clinical 1.5T and 3.0T scanners (GE Healthcare, Waukesha, WI). For all subjects, 4D PC-MR data were acquired before and after ingestion of a meal using radially-undersampled PC-VIPR acquisition[8,9] with full volumetric coverage of the upper abdomen: imaging volume: 32x32x24cm spherical; 1.25mm isotropic resolution; TR/TE=6.6-8.3ms/1.9-2.7ms; tip angle=6-15°; Venc=100-120cm/s; intravascular contrast agent (0.03mmol/kg gadofosveset trisodium (Lantheus, N. Billerica, MA)); with retrospective ECG and respiratory gating. Pre-prandial imaging was performed after 5 hours of fasting. After the first scan, subjects orally ingested 474 mL EnSure Plus® (Abbot Laboratories, Columbus, OH) and scanning was resumed 20 minutes after ingestion. 3D vessel segmentation from the PC data was performed using Mimics (Materialize, Leuven, Belgium), shown in Figure 2. Ensight (ANSYS, Canonsburg, PA) was used for plane placement and flow visualization (Figure 3). Cut planes were placed in 6 arterial vessels: supraceliac aorta (SCAo), infrarenal aorta (IRAo), CA, SMA, right renal (RRA), and left renal artery (LRA). Additionally, planes were placed in 3 venous vessels: splenic (SV), superior mesenteric (SMV), and portal vein (PV). Cut-planes were exported to a customized software package[10] for manual temporal segmentation throughout the cardiac cycle. Pulsatility and resistivity indices were automatically computed from the flow waveforms and were calculated for each subject. After data analysis, diagnoses for the patient group were given. The patient group was then subdivided into a true CMI group (N=6) and negative diagnosis group (N=13) based on these diagnoses. RI and PI indices were compared between controls and patient subgroups using a two-tailed Welch t-test. For all statistical tests, $p<0.05$ was chosen to reflect statistical significance.

## Results

4D flow data were successfully obtained for all 39 subjects (Table 1-2). SCAo preprandial PI for the negative diagnosis group was significantly lower compared to healthy subjects (p=0.018), but this relationship was not seen in the CMI-group (p=0.115). Preprandial IRAo PI for the CMI group was significantly lower than the healthy control group (p=9.64e-04) and the negative diagnosis group (p=0.039). Likewise, preprandial IRAo PI for the negative diagnosis group was lower than healthy controls (p=0.048). Similarly, postprandial IRAo PI for the CMI group was significantly lower than for the healthy controls (p=0.002) but was not lower for the negative diagnosis group (p=0.131). However, postprandial IRAo PI for the negative diagnosis group was significantly lower than for the healthy control group (p=0.013). Compared to the control group, postprandial SMA PI for the CMI group was significantly higher (p=0.033). This was not the case for the negative diagnosis group (p=0.105). Postprandial SV pulsatility indices for the CMI group (p=0.024) and negative diagnosis group (p=0.021) were significantly higher than healthy controls. Resistivity indices (for any vessel) were not significantly different between groups.

## Discussion

In this work, we investigated PI and RI as measures of vascular resistance. Significantly increased postprandial SMA PI for the ischemia group suggests higher downstream vascular resistance after a meal, supporting the “gastric steal” theory[2]. Average PI in the aortic segments (SCAo and IRAo) were highest among healthy control subjects, were decreased in the negative diagnosis group, and were lowest in the ischemia group, possibly reflecting progressive worsening of the atherosclerotic disease between subgroups.

## Conclusion

Along with flow measurements, pulsatility may provide additional hemodynamic information into downstream vascular resistance, which could aid in the challenging diagnosis of CMI.

## Acknowledgements

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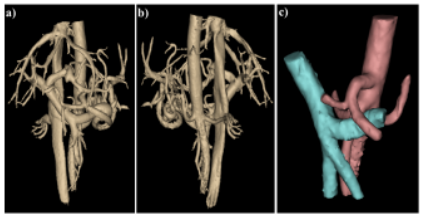
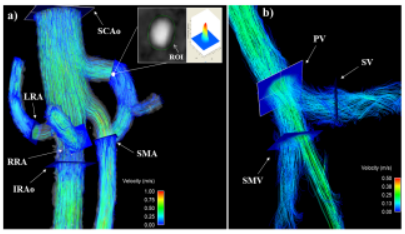
## Figures




PC angiograms of the mesenteric vasculature created from 4D flow MRI complex difference data. Figure 1a) and 1b) respectively show anterior and posterior views of the thresholded angiogram for a healthy (61-year-old, male) control subject. Note the fine vascular detail that can be visualized. Figure 1c) shows a “trimmed” angiogram from the same patient, including only relevant venous (blue) and arterial (red) vasculature needed for the quantitative analysis used here.




Velocity color-coded pathlines (a) in the arterial vasculature of a healthy (61-year-old, male) control subject and streamline images (b) in the venous system. 2D cut-planes (a,b) are shown for all mesenteric vessels being quantitatively analyzed. Inset image shows time-resolved manual segmentation of the exported velocity values from the 2D cut-plane in the customized software package.

**Table 1: Resistivity Indices**

| | SCAo | IRAo | LRA | RRA | SMA | CA | SMV | SV | PV |
|---|---|---|---|---|---|---|---|---|---|
| Control (Pre) | 0.86 ± 0.10 | 0.84 ± 0.13 | 0.82 ± 0.16 | 0.80 ± 0.11 | 0.72 ± 0.18 | 0.58 ± 0.15 | 0.55 ± 0.20 | 0.27 ± 0.08 | 0.21 ± 0.09 |
| (Postprandial) | 0.79 ± 0.18 | 0.81 ± 0.18 | 0.81 ± 0.21 | 0.79 ± 0.18 | 0.54 ± 0.20 | 0.81 ± 0.18 | 0.28 ± 0.16 | 0.28 ± 0.17 | 0.24 ± 0.14 |
| Neg. Diag. (Pre) | 0.77 ± 0.21 | 0.89 ± 0.13 | 0.68 ± 0.18 | 0.81 ± 0.19 | 0.79 ± 0.31 | 0.58 ± 0.28 | 0.34 ± 0.22 | 0.34 ± 0.24 | 0.27 ± 0.21 |
| (Postprandial) | 0.72 ± 0.17 | 0.81 ± 0.22 | 0.53 ± 0.24 | 0.65 ± 0.18 | 0.51 ± 0.18 | 0.84 ± 0.20 | 0.30 ± 0.15 | 0.30 ± 0.15 | 0.23 ± 0.16 |
| CMI (Pre) | 0.65 ± 0.21 | 0.68 ± 0.16 | 0.82 ± 0.24 | 0.61 ± 0.23 | 0.80 ± 0.18 | 0.55 ± 0.07 | 0.42 ± 0.21 | 0.34 ± 0.14 | 0.32 ± 0.21 |
| (Postprandial) | 0.76 ± 0.10 | 0.72 ± 0.21 | 0.88 ± 0.11 | 0.77 ± 0.16 | 0.80 ± 0.21 | 0.88 ± 0.18 | 0.38 ± 0.11 | 0.39 ± 0.18 | 0.34 ± 0.20 |

Resistivity indices are expressed as mean ± 1 standard deviation. **Bold** indicates statistical significance ($p < 0.05$) compared to controls. Underline indicates statistical significance ($p < 0.05$) between CMI and Neg. Diag. group. SCAo = supraceliac aorta, IRAo = infrarenal aorta, LRA = left renal artery, RRA = right renal artery, SMA = superior mesenteric artery, CA = celiac artery, SMV = superior mesenteric vein, SV = splenic vein, and PV = portal vein.

**Table 2: Pulsatility Indices**

| | SCAo | IRAo | LRA | RRA | SMA | CA | SMV | SV | PV |
|---|---|---|---|---|---|---|---|---|---|
| **Control (Pre)** | 3.00 ± 0.78 | 3.88 ± 2.4 | 1.26 ± 0.41 | 1.37 ± 0.38 | 2.22 ± 0.74 | 1.25 ± 0.43 | 1.01 ± 0.45 | 0.671 ± 0.38 | 0.335 ± 0.29 |
| (Postprandial) | 2.52 ± 0.84 | 6.34 ± 3.3 | 1.42 ± 0.51 | 1.47 ± 0.88 | 1.41 ± 0.55 | 1.43 ± 0.48 | 0.65 ± 0.27 | 0.637 ± 0.28 | 0.305 ± 0.25 |
| **Neg. Diag. (Pre)** | **2.32 ± 0.71** | **4.21 ± 2.9** | 1.39 ± 0.38 | 1.37 ± 0.81 | 1.84 ± 0.95 | 2.46 ± 2.7 | 1.09 ± 0.58 | 0.955 ± 0.33 | 0.723 ± 0.38 |
| (Postprandial) | 2.38 ± 0.64 | **3.88 ± 1.7** | 1.34 ± 0.38 | 1.37 ± 0.31 | 1.71 ± 0.34 | 2.18 ± 1.3 | 0.789 ± 0.23 | **1.11 ± 0.56** | 0.941 ± 0.33 |
| **CMI (Pre)** | 1.76 ± 1.4 | **2.26 ± 1.1** | 1.41 ± 0.83 | 1.41 ± 0.23 | 2.02 ± 0.48 | 1.28 ± 0.46 | 1.29 ± 0.35 | 1.23 ± 0.17 | 0.821 ± 0.03 |
| (Postprandial) | 2.17 ± 1.4 | **2.48 ± 1.5** | 2.39 ± 1.5 | 1.82 ± 0.75 | **1.86 ± 0.55** | 1.75 ± 0.41 | 0.869 ± 0.28 | **1.19 ± 0.55** | 1.14 ± 0.82 |

Pulsatility indices are expressed as mean ± 1 standard deviation. **Bold** indicates statistical significance ($p < 0.05$) compared to controls. Underline indicates statistical significance ($p < 0.05$) between CMI and Neg. Diag. group. SCAo = supraceliac aorta, IRAo = infrarenal aorta, LRA = left renal artery, RRA = right renal artery, SMA = superior mesenteric artery, CA = celiac artery, SMV = superior mesenteric vein, SV = splenic vein, and PV = portal vein.