Abstract—To evaluate the influence of duodenal feeding on splanchnic blood flow, 14 patients with normal coeliac and superior mesenteric arteries underwent intra-abdominal duplex scanning of the systemic and splanchnic circulation under standardised basal and meal-stimulated conditions. Doppler samples and diameter measurements were taken of the aorta, coeliac artery, common hepatic artery, splenic artery, superior mesenteric artery, and inferior mesenteric artery. Duodenal meal stimulation has no systemic effects ($p > 0.4$). However, duodenal meal stimulation results in coeliac artery vasoconstriction ($p < 0.06$) and superior mesenteric artery vasodilatation ($p < 0.05$). This study supports other reported results that gastrointestinal blood flow is dependent on the site of food stimulation. © 1998 World Federation for Ultrasound in Medicine & Biology.

Key Words: Splanchnic circulation, Ultrasound, Meal stimulation.

INTRODUCTION

Chronic splanchnic ischaemia is caused by occlusive disease in the coeliac and/or superior mesenteric artery. It is a rare disease, and its diagnosis is difficult because the symptoms are often aspecific. The correct diagnosis can be made retrospectively only when the symptoms have disappeared after successful vascular reconstruction. Several tests have been proposed to determine the haemodynamic significance of stenosis in the splanchnic arteries. Theoretically, stimulation with food should increase the splanchnic blood flow and produce symptoms if the flow fails to meet expectations. Duplex sonography currently is used to evaluate splanchnic flow noninvasively. However, the effects of meal stimulation on the duplex flow parameters of the splanchnic circulation are not completely understood. The various test meals used provide different results that are not obviously related to increasing volumes or caloric intake (Geelkerken and van Bockel 1995). This may be due to physiological differences between patients, technical difficulties inherent in transabdominal duplex (Burns et al. 1988; Perko and Just 1993; Zierler et al. 1991), or the abundant collateral circulation, which may compensate for significant occlusive disease in the coeliac and superior mesenteric arteries. Intra-abdominal, unlike transabdominal, duplex provides easy access to the abdominal vasculature and is not hampered by technical problems. A higher frequency probe can be used with resultant improved image quality. The improved resolution and greater magnification obtained intra-abdominally lead to a more accurate assessment of the beam-to-flow angle, which increases the probability of accurate velocity calculations (Delahunt et al. 1996; Geelkerken et al. 1996).

In this study, we used intra-abdominal duplex sonography for precise evaluation of the effects of duodenal feeding on systemic and splanchnic blood flows by assessing duplex flow parameters under completely standardised conditions in humans with normal splanchnic arteries.

PATIENTS AND METHODS

In a series of 14 patients, intra-abdominal basal and postprandial duplex of the aorta and the arterial splanchnic circulation was performed. All patients underwent elective abdominal vascular reconstructive surgery. The indications for operation were infrarenal aortic aneurysm-
mal disease (nine patients), distal aortic-iliac obliteratorive disease (three patients), and renovascular disease (two patients). The mean age of the 12 men and two women was 65 y (range 39–81 y). No patients had upper abdominal symptoms, related or unrelated to food. Previous abdominal surgery was not performed. None of the patients had diabetes mellitus. The procedures were explained in detail to the patients and consent was obtained from all in accordance with the Hospital’s Ethical Committee.

Abdominal angiography

Preoperatively, all patients underwent rotational intra-arterial digital subtraction angiography of the abdominal aorta and its branches (Philips 3000 Integris system, Philips). Multiple projections of the abdominal aorta and of the origin and outflow of the splanchnic arteries were obtained. The lymphatic fillings of the abdominal aorta, coeliac artery, and superior mesenteric artery were measured. Stenosis were graded from 0–100% by consensus of two radiologists. Patients with stenosis (≥ 50% luminal reduction) or occlusions of the coeliac or the superior mesenteric arteries were excluded from the study. The 14 patients selected had a normal proximal abdominal aorta, coeliac artery, and superior mesenteric artery inflow. These 14 patients were included in the present study.

Anaesthesia

All 14 patients received standardized anaesthesia (balanced or total intravenous anaesthesia (Shafer 1991; White 1990). Premedication was achieved with temazepam 10 or 20 mg. After induction with etomidate and sufentanil, a balanced anaesthesia was maintained with nitrous oxide and isoflurane (eight patients). After induction with propofol and sufentanil, total intravenous anaesthesia was maintained with continuous infusion of propofol and ventilation with oxygen in air (six patients). Adjustment of sufentanil, propofol, and isoflurane was according to haemodynamic responses. A pulmonary artery catheter (Viggo Spectramed, Ohmeda, Hatfield, UK) was placed immediately after the administration of anaesthesia. The patients were intubated and artificially ventilated with a volume-steered mechanical ventilator. Intermittent positive pressure ventilation was applied with 10 mL/kg tidal volume and a frequency of 12–14 beats/min, adjusted to keep the end-tidal CO2 between 3.5 and 4.0 kPa. Basic infusion was 10 mL/kg crystalloids, with extra gelofusine in case of hypovolaemia. All patients were completely stable with respect to pulse rate, blood pressure, urinary production, central venous pressure, pulmonary artery wedge pressure, cardiac output, systemic vascular resistance index, and frequency of respiration during the basal and postprandial duplex sonography.

Meal stimulation

A gastric suction tube was placed in the horizontal part of the duodenum. A soft bowel clamp was placed just distal to the pylorus to prevent reflux of the meal. The basal duplex measurements were performed. Subsequently, the test meal (100 mL of FortimelR, Nutricia, Zoetermeer, The Netherlands) containing 420 kJ, 2.1 g fat, 9.7 g protein, 4.4 g lactose, 4.2 g saccharose, and 1.8 g dextrose/maltose was instilled by the gastric suction tube to the duodenum within 1 min. The 30-min postprandial duplex measurements were reported.

Duplex sonography

All the duplex measurements were performed with the same colour duplex machine (Aloka 2000, Biomedic, Almere, The Netherlands). The intra-abdominal duplex measurements were performed by one vascular surgeon (RHG) after a laparotomy was performed. The test was completed before the vascular reconstructive procedure was performed. The intra-abdominal measurements were performed with a 7.5-MHz steerable linear array probe. A sterile plastic sleeve filled with acoustic gel was used to cover the transducer. With the aid of the colour flow mapping, the splanchnic arteries were identified easily. The flow-to-Doppler-beam angle was minimized. An angle of insonation above the 60° was never accepted; these measurements were excluded from the analysis. All Doppler measurements were performed during the expiration phase of normal artificial respiration. The aorta immediately proximal to the origin of the coeliac artery and the origin of the coeliac artery, common hepatic artery, splenic artery, superior mesenteric artery, and inferior mesenteric artery were evaluated with duplex sonography and flow spectra were obtained. All the duplex images and flow spectra were recorded on video tape. Postoperatively, the duplex flow parameters were analysed.

Duplex flow parameters

The following parameters of the Doppler signal were evaluated: the peak systolic velocity (PSV), peak diastolic reversed velocity (PDRV), peak diastolic forward velocity (PDFV), and end-diastolic velocity (EDV), all in cm/s. In addition, the pulsatility index (PI) and the resistive index (RI) were calculated, and the diameter of the artery (DA), in mm, and the scanning angle of vessel insonation (SA) were noted. The PI was defined as peak-to-peak velocity/mean velocity (Johnston 1993). The RI was defined as (PSV – EDV)/PSV.

Data management and statistics

All data were entered in a computer database. To test for statistically significant differences between
paired data, the paired t-test of the equality of the means was used. To study unpaired data, the Student’s t-test for independent samples was used. Results were checked with Wilcoxon’s signed-rank test and Mann–Whitney test, in view of the non-normality of some parameters. Both tests gave similar results (not reported). A \( p \) value < 0.05 was considered significant. A \( p \) value < 0.10 was considered to indicate a trend.

**RESULTS**

The flow characteristics in the abdominal aorta were not affected by meal stimulation (Table 1).

In the superior mesenteric artery, the PSV under basal conditions was 119 cm/s; the postprandial PSV increased to 142 cm/s (increase of 19%; \( p = 0.09 \)). The PDFV under basal conditions was 23 cm/s; the postprandial PDFV increased to 37 cm/s (increase of 62%; \( p = 0.03 \)). The EDV under basal conditions was 9 cm/s; the postprandial EDV increased to 21 cm/s (increase of 121%; \( p = 0.02 \)). The PDRV decreased after meal stimulation (decrease of 220%; \( p = 0.02 \)). Consequently, the PI and the RI decreased significantly after meal stimulation (decrease of 23% \([ p = 0.01]\) and 5% \([ p = 0.05]\), respectively) (Table 1).

Although Doppler velocities of the inferior mesenteric artery demonstrated, to some extent, the same changes after meal stimulation as the superior mesenteric artery velocities, the differences between basal-to-postprandial Doppler velocities never reached statistical significance (Table 1).

In the coeliac artery, the PSV under basal conditions was 161 cm/s; the postprandial PSV decreased to 140 cm/s (decrease of 13%; \( p = 0.06 \)). The PDFV under basal conditions was 76 cm/s; the postprandial PDFV decreased to 61 cm/s (decrease of 20%; \( p = 0.03 \)). The EDV under basal conditions was 47 cm/s; the postprandial EDV decreased to 37 cm/s (decrease of 23%; \( p = 0.01 \)). Consequently, the PI and the RI increased after meal stimulation (increase of 9% \([ p = 0.06]\) and 4% \([ p = 0.06]\), respectively) (Table 2).

The splenic and, particularly, the hepatic artery velocities and indices demonstrated the same changes after meal stimulation as the coeliac artery velocities. However, the differences between basal-to-postprandial splenic and hepatic Doppler velocities never reached statistical significance (Table 2).

For all the arteries studied, the basal-to-postprandial diameter and the flow-to-Doppler-beam angle were similar (Tables 1 and 2).

The mean increase or decrease of the Doppler velocities in the balanced anaesthesia group was compared to the mean increase or decrease of the Doppler velocities in the total intravenous anaesthesia group. A relevant

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Aorta</th>
<th>Superior mesenteric artery</th>
<th>Inferior mesenteric artery</th>
</tr>
</thead>
<tbody>
<tr>
<td>( n )</td>
<td>( \text{Mean SD} )</td>
<td>( \text{Mean SD} )</td>
<td>( \text{Mean SD} )</td>
</tr>
<tr>
<td>PSV</td>
<td>13</td>
<td>21.7 (4.5)</td>
<td>15.5 (4.7)</td>
</tr>
<tr>
<td>PDRV</td>
<td>9</td>
<td>9.3 (0.8)</td>
<td>3.4 (0.3)</td>
</tr>
<tr>
<td>PDFV</td>
<td>13</td>
<td>11.3 (4.8)</td>
<td>7.4 (3.2)</td>
</tr>
<tr>
<td>EDV</td>
<td>13</td>
<td>6.1 (1.2)</td>
<td>5.2 (1.2)</td>
</tr>
<tr>
<td>PI</td>
<td>13</td>
<td>0.6 (0.4)</td>
<td>2.7 (0.4)</td>
</tr>
<tr>
<td>RI</td>
<td>13</td>
<td>1.8 (0.4)</td>
<td>1.2 (0.4)</td>
</tr>
<tr>
<td>SA</td>
<td>9</td>
<td>1.8 (0.3)</td>
<td>3.0 (0.3)</td>
</tr>
<tr>
<td>SEM</td>
<td>13</td>
<td>1.7 (0.3)</td>
<td>1.7 (0.3)</td>
</tr>
</tbody>
</table>

\( p \) values determined by two-tailed paired t-test.

\( rA \) = diameter of the artery; \( EDV \) = end-diastolic velocity; \( PDFV \) = peak diastolic forward velocity; \( PDRV \) = peak diastolic reversed velocity; \( PI \) = pulsatility index; \( PSV \) = peak systolic velocity; \( RI \) = resistive index; \( SA \) = scanning angle of vessel insonation; \( SD \) = standard deviation; \( SEM \) = standard error of the mean.
difference was not measurable for any of the Doppler parameters.

**DISCUSSION**

In a well-defined study in humans under stable anaesthesia, meal stimulation of the duodenum had no effect on systemic flow parameters. However, meal stimulation of the duodenum results in coeliac artery outflow vasoconstriction and superior mesenteric artery outflow vasodilatation (Table 3).

The mechanism whereby intraduodenal food instillation affects splanchnic artery blood flow has not been fully elucidated (Dauzat et al. 1994). The increase in coeliac artery outflow resistance is in accordance with result of others (Joynt et al 1995; LaFortune et al. 1993; Lautt et al. 1990), who reported an increase in postprandial hepatic artery resistance. The increase in superior mesenteric artery blood flow in response to intestinal feeding is in line with previous reports indicating that products of food digestion play an important part in this increased superior mesenteric blood flow (Moneta et al. 1988; Sidery et al. 1994; Sieber et al. 1992). Neurohumoral mechanisms, both systemically and locally, may be involved in this phenomenon (Fara et al. 1972; Parker et al. 1995; Premen et al. 1985). It has been demonstrated in dogs that administration of nutrients into a segment of the small intestine induces a local increase in blood flow to the segment, pointing to a predominant role of local regulatory mechanisms (Galavan et al. 1980). Among these local regulatory mechanisms, local reflexes and various bioactive substances, such as histamine, prostaglandins, serotonin, and purine nucleotides, have been suggested to play a part (Parks et al. 1987). Because the stomach and proximal duodenal were devoid of food, coeliac artery blood flow after duodenal feeding was expected not to be different from the basal condition. However, we found a decrease of coeliac blood flow. The mechanism whereby distal duodenal feeding inhibits coeliac blood flow is more puzzling. The inhibition of coeliac blood flow most likely is mediated by neurohumoral mechanisms originating in the small intestine. In this respect, it is interesting to note that food in the small intestine inhibits gastric acid secretion and gastric motility (Debas 1987). Several “enterogastrones” have been suggested as possible hormonal candidates, such as GIP, GLP-1, CCK, neurotensin, and PYY (Debas 1987; Walsh 1987). It is possible that a similar inhibitory mechanism is involved in the decrease of coeliac blood flow upon intestinal meal stimulation. In this study, we applied a liquid mixed hyperosmotic meal to be certain that intestinal neurohumoral mechanisms were activated adequately. Activation of bile secretion and digestive enzymes has been reported to be important, because
stimulation of splanchnic blood flow predominantly is mediated by digested food products (Chou et al. 1978). This is in analogy with the previously reported entero-gastrone effect, in which digested food also is required for stimulation (Debas 1987). Thus, the gastrone effect by food in the small intestine not only comprises inhibition of gastric acid and gastric motility but also inhibition of gastric blood flow. It is tempting to speculate that the inhibition of gastric acid is secondary to the reduced gastric blood flow.

Other explanations for the different effects of intraduodenal food on coeliac and superior mesenteric blood flows are not obvious. The design and the conduct of our study could not been the explanation. All our patients had normal upper abdominal anatomy and no pathology of the proximal abdominal aorta, coeliac artery, and superior mesenteric artery. Diabetic gastropathy (Best et al. 1991), which could influence gastric blood flow, could not explain our results because none of our patients was diabetic. Differences in arterial origin diameter and the flow-to-Doppler-beam angle could not have been the explanation, because basal and stimulated artery diameter and Doppler beam angles were similar.

Our meal stimulation was adequate, which clearly is demonstrated by the increase of superior mesenteric artery blood flow. The timing of the flow measurements could not have been the explanation either, because, after stimulation of the splanchnic flow, the greatest effect on coeliac and superior mesenteric artery flow is reached within 45 min (Geelkerken and van Bockel 1995). We measured splanchnic flow 30 min after duodenal meal stimulation, when the FortimelR meal was not completely resorbed or displaced. It is very unlikely that anaesthesia itself produced the contrary effects in the coeliac and superior mesenteric artery outflow. During the measurements, all our patients were under stable, standardised, anaesthesia, and both mutual anaesthesia techniques produced comparable effects on the coeliac and superior mesenteric artery outflow. Although we could not exclude that laparotomy itself may have some effect on coeliac and superior mesenteric flow, it is not comprehensible that laparotomy produced contrary effects on the coeliac and the superior mesenteric artery blood flows. Also, the effects of placing a postpylorus soft bowel clamp on splanchnic blood flow is unknown; however, we feel that this manoeuvre could not explain the findings.

The present report and the literature (Galavan et al. 1980; Goo et al. 1987; Qamar and Read 1987; Sieber et al. 1992; Takagi et al. 1988) demonstrate that normal coeliac and superior mesenteric artery blood flows are highly variable, due to the site and the type of physiological stimuli. Thus, when studying splanchnic blood flow in normal volunteers, the test should be performed under standardised conditions. Due to the unknown influence of collateral circulation, use of the meal stimulation test for splanchnic flow in patients suspected of having stenosis of the splanchnic arteries still seems unclear.

Acknowledgement—We greatly appreciate the support given to this study by Leo J. Schultze Kool, M.D., Ph.D., interventional radiologist.

REFERENCES
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Table 3. Summary of statistical analysis of differences between the basal and postprandial flow characteristics.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Abdominal aorta</th>
<th>Coeliac</th>
<th>Common hepatic</th>
<th>Splenic</th>
<th>Superior mesenteric</th>
<th>Inferior mesenteric</th>
</tr>
</thead>
<tbody>
<tr>
<td>PSV</td>
<td>0.47</td>
<td>0.06</td>
<td>0.07</td>
<td>0.21</td>
<td>0.09</td>
<td>0.21</td>
</tr>
<tr>
<td>PDRV</td>
<td>0.48</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>0.55</td>
</tr>
<tr>
<td>PDFV</td>
<td>0.32</td>
<td>0.03</td>
<td>0.26</td>
<td>0.60</td>
<td>0.03</td>
<td>0.20</td>
</tr>
<tr>
<td>EDV</td>
<td>0.70</td>
<td>0.01</td>
<td>0.34</td>
<td>0.50</td>
<td>0.02</td>
<td>0.81</td>
</tr>
<tr>
<td>PI</td>
<td>0.83</td>
<td>0.06</td>
<td>0.38</td>
<td>0.90</td>
<td>0.001</td>
<td>0.35</td>
</tr>
<tr>
<td>RI</td>
<td>0.43</td>
<td>0.06</td>
<td>0.38</td>
<td>0.63</td>
<td>0.05</td>
<td>0.84</td>
</tr>
<tr>
<td>DA</td>
<td>0.73</td>
<td>0.27</td>
<td>0.76</td>
<td>0.44</td>
<td>0.86</td>
<td>0.20</td>
</tr>
<tr>
<td>SA</td>
<td>0.84</td>
<td>0.13</td>
<td>0.48</td>
<td>0.38</td>
<td>0.33</td>
<td>0.57</td>
</tr>
</tbody>
</table>

Abbreviations as in Table 1.