PT Harjola performed the first median arcuate ligament (MAL) release in 1963. In 1965, Dunbar published a larger series of 15 patients after an MAL release. Over the ensuing 5 decades, numerous articles have appeared that discuss various topics regarding MAL syndrome including diagnostic modalities, outcomes, and surgical techniques. However, the overwhelming majority of these reports entail small case series with limited follow-up. Few series document large cohorts with follow-up beyond a few months.

Radiologically, compression of the celiac artery secondary to the MAL may be found as a normal variant in more than 20% of patients. Based on these findings, MAL syndrome may be a normal variant in a patient with abdominal pain. Therefore, a definitive diagnosis of MAL compression requires evidence of radiologic compression and clinical symptoms. These symptoms may include post-prandial pain, nausea, vomiting, and weight loss. Presently, there are few studies that provide any preoperative factors or tests that accurately predict the impact of surgical intervention with clinical outcomes.

After establishing a diagnosis of MAL syndrome, surgical intervention includes endovascular treatments with angioplasty or stent placement, MAL releases, arterial by-passes, or some type of hybrid mixture of these procedures. Historically, a MAL release has been performed through an open incision, but now laparoscopic approaches are used. To date, 38 patients have undergone a laparoscopic MAL release at our institution. The diagnostic workup and laparoscopic technique for a MAL release at our institution are presented. An overview of the clinical outcomes is provided as well.

METHODS
Preoperative evaluation
Initially, the workup includes consultation with a vascular and general surgeon for a detailed history and physical examination. Typically, patients present with epigastric or diffuse abdominal pain that is worse postprandially. The pain may be accompanied by nausea and vomiting. The patient may report gradual weight loss also. The overwhelming majority of patients have undergone an exhaustive work-up including upper and lower endoscopies, CT scans, MRI, ultrasounds, and psychiatric evaluations. In addition, most patients present with a history of some type of surgical intervention that may include a diagnostic laparoscopy, cholecystectomy, or a lysis of adhesions. On examination, patients are typically thin, with a flat abdomen that is nontender. Despite knowledge of their underlying pathology and their thin body habitus, an abdominal bruit is not detected.

A celiac artery ultrasound is obtained with baseline, peak inspiratory, and peak expiratory velocities. At a minimum, the ultrasound findings require a peak expiratory velocity of greater than 200 cm/second to entertain a preliminary diagnosis of MAL syndrome. Peak expiratory velocities need to be greater than resting velocities and peak expiratory velocities of greater than 500 cm/second have been encountered. Based on the patient’s history, a magnetic resonance arteriogram (MRA), CT angiography (CTA), or an aortogram may be recommended for confirmation. Patients with gastroparesis and gastric electrical stimulators may not undergo an MRA, and these patients require either a lateral aortogram or a CT angiogram. The majority of patients without internal hardware proceed with an MRA to evaluate the visceral organs and the remaining arterial structures. Diagnostically, the MRA may show compression of the celiac artery, a hooked celiac artery, post-stenotic dilatation, or rarely, prominent collaterals to the superior mesenteric artery. If a diagnosis of MAL syndrome is identified, the patient proceeds with a laparoscopic MAL release. Before surgery, a Short Form (SF)-36 questionnaire is completed to assess quality of life parameters.

Laparoscopic median arcuate ligament release
After general anesthesia, a Foley catheter and an orogastric tube are inserted. The patient is positioned in a split-leg fashion in order for the surgeon to stand in the middle of the patient’s legs. A Veress needle is introduced 2 cm below the left costal margin in the mid-clavicular line. The majority of patients have had some type of previous surgery, so the initial port is placed at an alternative site versus the umbilicus. Furthermore, an umbilcal entry does not provide adequate visualization for the celiac artery and MAL. Typically, an angled laparoscope enhances...
visualization over the pancreatic body. Once the abdomen is insufflated to 15 mmHg with carbon dioxide, a Visiport (Covidien) is inserted approximately 16 cm from the xiphoid. Subsequently, four 5-mm ports are placed. A 5-mm port is placed in the subxiphoid region for a liver retractor. A 5-mm port is placed approximately 4 cm in the mid-clavicular line in the right upper quadrant, and another port is placed at the Veress needle site in the left upper quadrant. Finally, a 5-mm port is placed in the left anterior axillary line at the level of the costal margin (Fig. 1). Based on the patient’s previous surgical history, an extensive lysis of adhesions may be required, as many patients have undergone various procedures including Nissen fundoplications, bariatric surgery, ventral hernia repairs, gastric electrical stimulators, small bowel resections, and colon resections.

Once the operative field is cleared of adhesions, the lateral segment of the liver is elevated and retracted anteriorly (Fig. 2). The lesser sac is entered with electrocautery or an ultrasonic scalpel. At this point, the angled scope is critical to visualize the celiac artery throughout its origin and distal trifurcation. The left gastric artery and coronary vein are elevated and followed in a retrograde fashion toward the common hepatic and splenic artery (Fig. 3). The peritoneal lining along these 3 vessels is divided with the hook cautery. An L-hook cautery with a diameter of approximately 3 mm provides a fine dissection of not only the peritoneal lining but of the muscular, ligamentous, and neurovascular structures.

The common hepatic and splenic arteries are dissected from the superior border of the pancreas with the cautery. Both arteries are exposed for approximately 4 cm along the superior border of the pancreas. Releasing both of these arteries from the pancreatic border enhances mobilization of the celiac trunk. Once the superior border of

**Figure 1.** Port placement. Standard foregut laparoscopic approach.

**Figure 2.** Retraction of the lateral segment of the liver. The left lateral liver is elevated anteriorly with a retractor, exposing the caudate lobe. The coronary vein and left gastric vessel are visible next to the caudate lobe. This patient had a previous Nissen fundoplication.

**Figure 3.** Dissected trifurcation. The hepatic (HA) and splenic (SA) artery and coronary vein (CV) have been dissected. The coronary vein drains into the portal system. The peritoneal attachments have been divided.
the pancreas is released, the posterior aspect of the celiac artery may be appreciated (Fig. 4). An extensive dissection of the common hepatic and splenic arteries enables division of numerous fibers from the celiac plexus surrounding these vital arteries as well. A window is then made beneath the left gastric artery and the coronary vein to visualize the right crus, MAL, and ligament to the fourth portion of the duodenum (Fig. 5). The peritoneal lining on top of these structures is divided with the hook cautery. Subsequently, these muscular and ligamentous structures are divided with cautery (Fig. 6). Division of these structures proceeds from left to right and starts with the ligament to the fourth portion of the duodenum. The dissection is performed in layers to avoid an underlying injury to the aorta. The dissection should expose at least 4 cm of aorta cephalad to the celiac artery origin. Laterally and medially, the dissection extends posteriorly toward

Figure 4. Dissection of anterior peritoneal attachments to reveal the celiac trunk. Anterior peritoneal attachments are dissected revealing the celiac trunk.

Figure 5. Completed dissection of common hepatic artery and splenic artery. The common hepatic (HA) and splenic (SA) artery have been dissected from the anterior pancreas along with the neurovascular attachments for 4 cm in each direction. The celiac trunk is likewise cleared anteriorly.

Figure 6. Intact median arcuate ligament (MAL). The MAL is exposed from the attachments to the right at the vertebral body to the suspensory ligament of the fourth portion of the duodenum, which is under the tip of the laparoscopic sucker.

Figure 7. Median arcuate ligament (MAL) release with electrocautery. Muscular fibers of the MAL are dissected layer by layer from superficial to deep to avoid injury to the aorta. Nerve bundles of the celiac plexus are intertwined with the MAL and are also divided.
the vertebral bodies to divide the celiac plexus and the associated ganglia. The neurovascular bundles are slightly more tenacious than the overlying fascia and muscular layers (Figs. 7 to 9).

If bleeding is encountered from any of the structures, ultrasonic therapy and/or cautery are used initially. Clips are not used in order to avoid compression of any neural structures. The anterior border of the celiac artery is then denuded of all muscular, ligamentous, and neurovascular structures (Fig. 10). If possible, the entire anterior surface of the celiac artery from its origin to its trifurcation is exposed. Due to the angulation of the celiac artery, it may be possible to dissect only the compressed area of the artery. In many patients, after releasing the overlying structures of the celiac artery, indentations are readily apparent on the celiac artery (Fig. 11). Post-stenotic bulbar dilatation may also be visualized at the region of the trifurcation. Hemostasis is then verified and a final examination is performed of the common hepatic, splenic, left gastric, and celiac arteries (Fig. 12). The aorta and neural structures medially and laterally are examined as well. At this point, the abdomen is desufflated and ports are closed.

Postoperatively, all patients are admitted to the hospital and advanced to a clear liquid diet. Patients are advanced to solid food as tolerated and discharged when performing activities of daily living with pain well controlled on oral medications. Postoperatively, a repeat celiac artery ultrasound is obtained at 6-month intervals and SF-36 questionnaires are performed at 12-month intervals.

DISCUSSION

Median arcuate ligament syndrome remains an elusive diagnosis, with only a few large series available in the literature. Several of these series use a variety of surgical treatments without a standard protocol. Presently, a systematic algorithm does not exist due to the rarity of this disease. Our institution has chosen to incorporate a laparoscopic release of the MAL and the surrounding structures as

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**Figure 8.** Exposed aorta under released median arcuate ligament (MAL). The MAL has been released overlying the aorta (A). This demonstrates the wide swath of tissue that is released in order to prevent recurrence and complete the neurovascular dissection.
the initial treatment in our algorithm. At this time, a laparoscopic MAL release has been performed on 38 individuals. The laparoscopic release was successfully completed in 37 individuals. One patient required a conversion due to adhesions from 4 previous procedures, which included a gastric bypass, a lysis of adhesions, and 2 ventral hernias with insertion of mesh.

Based on our experience with this rare entity, we believe that this disease does not have a vascular etiology. We consider this disease to be a neurogenic entity secondary to compression of the celiac plexus. The celiac plexus is composed of pain fibers and inhibitory motor fibers to the stomach. By decompressing or dividing the neural fibers, patients theoretically would benefit from a decrease in pain and normal gastric motility. This has been documented in a previous case report that was published in 1998. The case report details a patient with gastroparesis who underwent a gastric electrical stimulator and then subsequently was diagnosed with MAL syndrome. After a MAL release, the patient’s pain resolved and gastric cycling normalized, with resolution of nausea and vomiting.

From a technical perspective, a wide dissection on the aorta is performed intraoperatively with the L-hook cautery. The dissection exposes at least 4 cm of aorta to avoid compression of the celiac artery postoperatively. Obviously, the procedure is performed with the patient supine with paralytic medications. When the patient stands, the
diaphragm and its dynamic nature might compress the celiac artery and celiac plexus again unless a wide swath of muscular and ligamentous structures are divided. All of the intraoperative efforts are intended to avoid recurrent compression of the celiac plexus. Of note, we performed intraoperative ultrasonography of the celiac artery in our first 15 patients but have abandoned this in our last 33 patients. The ultrasound findings were difficult to interpret. More importantly, the intraoperative ultrasound did not alter our technique in any way.

Two patients require intermittent narcotics postoperatively, and both of these patients have undergone further aortograms. One aortogram showed normal vascular anatomy, and 1 patient was treated with a celiac artery stent for refractory compression of the celiac artery. Five patients were readmitted to the hospital. One patient was diagnosed with a segmental pulmonary embolus that was treated with 3 months of Coumadin (Bristol-Myers Squibb) and complete resolution. The other 4 patients were admitted for tachycardia and pain and were treated with intravenous fluids, with resolution of tachycardia and pain. No abnormalities were documented on their admission CT scans. Overall, complete resolution or partial resolution of preoperative pain has been accomplished in 33 of 38 patients. Four patients have undergone gastric electrical stimulators. The four patients noted a decrease of their visceral pain; however, their gastroparetic symptoms continued, necessitating a gastric electrical stimulator.

Postoperatively, 1 patient required a near total gastrectomy for persistent nausea and vomiting. This patient’s postoperative SF-36 noted a significant improvement in bodily pain, but the patient still suffered from refractory nausea and vomiting. After her near total gastrectomy, her nausea and vomiting resolved and she is tolerating a regular diet. Four patients required nutritional supplementation preoperatively in terms of either enteric tubes or total parenteral nutrition. Both enteric tubes have been removed and the 2 patients who required total parenteral nutrition receive only intermittent intravenous fluids on a weekly basis.

CONCLUSIONS
In conclusion, the technique described here has been used in a relatively large group of patients diagnosed with MAL syndrome. To date, our outcomes data support performing only a release of the MAL and do not incorporate any endovascular or vascular procedures initially. Clearly, this cohort needs to be followed with subsequent ultrasound studies and SF-36 surveys to accrue data with at least 12-month follow-up to document durability. At this time, we are developing a predictive formula for operative success using the SF-36 values and the celiac ultrasound values. As we continue to accrue patients, we plan to verify our preoperative predictive formula to help guide future therapy for MAL syndrome and patient expectations.

REFERENCES