Gastric Pain

Gastric pain can be the result of a number of different dysfunctional processes. Thus, it is important to screen out some of the red flags that will require a specialist referral. When investigating gastric pain, we need to consider the nerves first. The nerves can be sensitive in different areas for different reasons. This includes those in the gut. There can be local nerve sensitization and irritation from the gut itself (more information in "Chapter 7: Organs"), central sensitization from the spinal cord and brain (more information in "Chapter 10: Pain"), agitation from stress and/or anxiety influencing both the ANS (more information in "Chapter 16: Dysautonomia") and overall GI function.

Different Issues That May Cause Gastric Pain And Should Be Investigated
• Food sensitivities • Dumping syndrome • Slowed motility • MALS • Abdominal angioedema (or hereditary abdominal angioedema) • Anterior abdominal wall pain • Visceral/organ involvement

Figure 6.6 Multiple issues may be feeding into gastric pain. In those with HSD/EDS, determining what is present will help guide treatment.

Gastric pain can also be orthopedic in nature in what we call "anterior abdominal wall pain." This stems from dysfunction in the thoracic and/or abdominal muscles that leads to trigger points within the abdominal wall, and can cause peripheral nerve entrapment from fascial tension. The nerves can become bound down in an area of dysfunction. This can be felt as burning pain wrapping around the abdomen that may be constant for some. Treatment is sometimes successful, with trigger point injections and/or physical therapy interventions by someone who specializes in this area.

MCAS, HSD/EDS, and the GI System

The GI system hosts a large quantity of mast cells. Unfortunately, mast cell activation in the gut can cause symptoms that affect the whole body, making it difficult to determine the underlying cause and location of the problem. For example, mast cell activation in the gut can cause a headache or aggravate joint pain. Because of this, the physical therapist needs to be aware of MCAS and know when to refer for treatment, to help reduce joint pain that is not mechanical in nature and improve the patient's ability to participate in physical activity.

MCAS reactions that may occur in the gut can appear symptomatic elsewhere as joint pain, nerve pain, etc. This is important for the therapist to be aware of, especially if the patient is not responding to treatment progression as expected.

It can be difficult to determine if MCAS reactions are happening in the gut, as we don't always see GI dysfunction manifest solely in its own terrain. GI dysfunction and inflammation from MCAS can present as brain fog, chronic fatigue, acne breakouts, rashes, eczema, hives, headaches, and many other reactions we don't connect with the gut right away. To determine if these gut reactions are causing some of your symptoms, a thorough investigation with someone who specializes in MCAS is needed.

Aside from the presentations listed above, there are some other, more specific reactions and symptoms we may see in those with MCAS stemming from GI specific involvement. These may include food and drink sensitivities, difficulty swallowing, burning in the mouth and/or stomach, medication sensitivities, abdominal and upper thigh swelling, abdominal bloating, diarrhea and/or constipation, and reports of malabsorption or vitamin deficiencies [16], [21], [40], [41]. More detail on MCAS presentation, signs and symptoms, and treatment strategies for the patient can be found in the specific MCAS chapter of this book ("Chapter 15: Mast Cell Activation Syndrome").

Dysautonomia, HSD/EDS and the GI System

To put it into perspective, the GI system has just as many nerves as the spinal cord. For this reason, the patient and practitioner cannot overlook the ANS when dealing with gut issues. They are one in the same. The GI system's nerve supply primarily comes from the ANS. The vagus nerve part of the ANS innervates the digestive tract, from the esophagus to the splenic flexure of the colon. The sacral parasympathetic nucleus (another part of the ANS) innervates the left colon and rectum. When someone has low vagal tone, or a dysfunctioning ANS, this will impair GI motility. Dysfunction of the vagus nerve can cause poor mobility in the esophagus as well, causing difficulty swallowing (see figure 6.7). On the other hand, a well-functioning vagus nerve may help reduce inflammation in the gut, as low tone in this nerve is associated with higher levels of inflammation within the body. The GI system and the ANS can present a chicken-or-egg scenario when symptoms occur. The good news is that knowing the answer to "which came first?" may not be as important for some patients. Addressing both systems to reduce symptoms. Exploring that complex question can be very helpful in regard to long term care and control of health.

Conditions to Investigate with Difficulty Swallowing
• Dysautonomia • Esophageal dysfunction (structural or functional) • Local nerve irritation to the throat musculature • Upper cervical instability • Eagle syndrome • Upper thoracic or cervical spine involvement • Oral myofunctional issues (mouth, jaw, tongue dysfunction and/or impaired neuromuscular control)

Figure 6.7 Difficulty swallowing can be due to a number of conditions. These should be explored with the patient to avoid missing a condition that will require a referral to a specialist.

Dysautonomia and gut issues can also work in reverse, as events that happen in the gut can trigger episodes of dysautonomia. For example, we can see this with gastric dumping [19], [20]. For someone with impaired motility/movement of the food through the GI system, their gut will lose the rhythmical small contractions, and the stomach may dump its contents rapidly into the small intestine. With this, the food is not fully broken down, and one is left with larger particles than the small intestine is used to breaking down and absorbing, along with a larger portion of food at one time. That can cause some stretching of the small intestine. When the small intestine registers this stretch, the nerves try to increase its response to help move food through the area and this can cause local pain in the small intestine. Along with a physical pain response, the body releases additional hormones and shifts the blood flow back to the GI system to assist with this "emergency" of a dumping response. When the blood flow is adjusted to the system, this begins to trigger a shift in the sympathetic nervous system and may cause an overall systemic autonomic response as the body struggles to find balance again (see Figure 6.8).

Early or late dumping responses may occur within the same patient. Early dumping is an ANS response from the reaction that occurs within 30 minutes of eating because the small intestine is being stretched, there is an increase in hormone release, and the ANS overcompensates to assist in the event. This can be seen in the GI system as malnutrition in protein energy and manifest in the rest of the body as the following:

- Heart palpitations
- Tachycardia (increased heart rate)
- Feeling the need to lie down after meals
- Flushing or paleness
- Sweating
- Lightheadedness
- Drop in blood pressure
- Headaches
- Possible fainting
- Feeling full soon into eating
- Stomach pain
- Nausea
- Abdominal area cramping
- Bloating
- Rumbling/gurgling in the GI system after eating

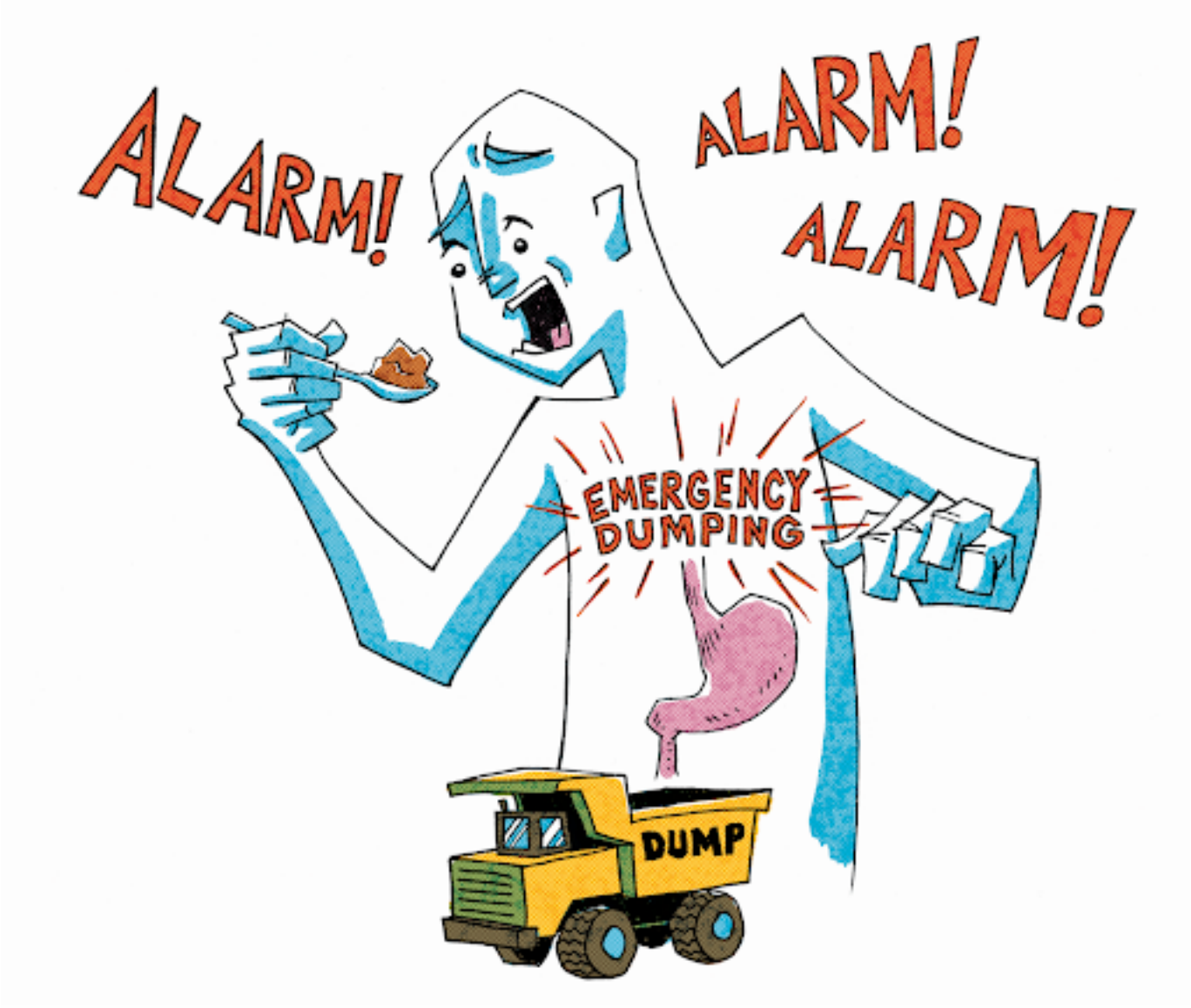

Figure 6.8 Dumping syndrome is a reaction of the ANS seen in those with dysautonomia and/or motility issues in the gut.

Late dumping can be seen one to three hours after eating a meal. This can occur through an increase in the hormones secreted into the GI system, chronic GI inflammation, dysautonomia, or a history of diabetes. Symptoms include:

- Sweating
- Faintness
- Decreased concentration, brain fog
- Varying levels of consciousness

Late dumping is less common than early dumping. That said, the same individual can have both early and late dumping occur with meals. Specific meal strategies can be found later in this GI management section. Treatments for the ANS can be found later in this book ("Chapter 16: Dysautonomia"). Both are recommended in combination as treatment strategies when dumping is suspected.

It is imperative to investigate the state of the ANS with GI functional issues. As mentioned previously, POTS (a form of dysautonomia) is the independent predictor of gut dysmotility [35]. Having HSD and POTS increases the odds of having GI dysmotility eight-fold [22]. Gut symptoms appear to be mediated by the gut-brain axis. This is most likely due to excess sympathetic tone, impaired peripheral autonomic function, hypovolemia, small fiber neuropathy, volume

dysregulation, and autonomic dysfunction. When HSD/EDS and POTS are present together there is a greater prevalence of chronic fatigue, increased fibromyalgia symptoms, and elevated symptoms of depression with increased reports of functional GI disorders, having both increased frequency and more afflicted gastrointestinal regions [35]. A thorough ANS investigation needs to be performed when functional gastrointestinal disorders and or motility issues are present.

Functional gastrointestinal motility disorders are more closely linked with autonomic nervous system dysfunction. Those that present with functional GI disorders must be screened for ANS involvement, and treatment should be integrated into the overall plan accordingly.

Hypermobility, the GI System, and Psychology

Studies have found a correlation between hypermobile individuals with reports of GI dysfunction and mood disorders, specifically depression. While this overlap can be related to the simple fact that additional disorders may tax the mood of the individual, we may also want to consider a hormonal role as well. Also, dysregulation of the gut microbiota, which can occur with functional disorders and motility issues, may reduce the availability of tryptophan. This may impact serotonin levels in the hippocampus [35], [38]. Treating the gut through dietary strategies, nervous system approaches, and manual treatments in visceral manipulation may be beneficial for these patients.

Dysfunction in the gut and the muscles involved in eating (jaw, throat, etc.) may lead to disordered eating or fear of eating [3]. If these issues are left unaddressed, this has the potential to progress to an eating disorder in some patients. It is important to understand that a driving force in this population can be physical dysfunction and/or fear avoidance from pain or reactions. This underlying fear must be addressed when treating an eating disorder in this population. Notably, patients that have disordered eating due to pain, nausea, and vomiting caused by dysautonomia may receive the inappropriate diagnosis of an eating disorder, which has a completely different treatment approach than what would be needed for someone who's eating is limited by a dysfunctional GI system. This misdiagnosis can lead to significant frustration for the patient who is given the wrong or inadequate treatment for their very real physical issue. On the other hand, the more stress or fear of discomfort one has, the more dysfunctional the GI system can become, so it is difficult to completely separate psychological from physical complaints. It is therefore very important to listen to the patient, not to make assumptions, and to consider the complex nature of GI symptoms. More information can be found in "Chapter 11: Psychological and Behavioral Presentation."

Red Flags in the GI System

While GI symptoms are very common for the HSD/EDS population (as well as the general population), it is important to watch out for red flags that would require referral to a specialist for specific and more immediate treatment. Two diagnoses that are more commonly seen symptomatically in HSD/EDS than the general population are abdominal angioedema (or hereditary abdominal angioedema) and median arcuate ligament syndrome (MALS). If either of these issues are suspected, the patient should see a specialist as soon as possible.

Abdominal Angioedema

Abdominal angioedema is an allergic reaction that occurs in the gut and surrounding tissue. This can cause localized temporary swelling that can affect all layers of the skin or the walls of the hollow organs, acute pain, typically non-pitting edema, and/or diarrhea. This swelling is seen in the respiratory system, GI tract, and oropharynx (area of the throat). The severity ranges from mild, with nausea and vomiting with pain, to life threatening episodes if the respiratory system is severely impacted. Attacks can be severe and acute, or may develop into a chronic condition with recurrent,

moderate abdominal pain that is crampy and colicky. Hereditary abdominal angioedema (HAE) presents similarly and can be genetically tested for. The key feature that should cause suspicion is the swelling noted that occurs with these reactions [30].

Median Arcuate Ligament Syndrome (MALS)

MALS is more of an anatomical issue, where the median arcuate ligament compresses the celiac complex, which can include the celiac artery, a major branch of the aorta that delivers blood to the stomach, liver, and other organs. The median arcuate ligament is formed at the base of the diaphragm between the left and right crus of the diaphragm. The ligament creates the top of the arch for the aortic hiatus behind it. The aorta passes through this aortic hiatus along with the azygos vein and the thoracic duct, the largest lymphatic vessel in the body. Dysfunction at the median arcuate ligament may not only compress these contents within the aortic hiatus, but can also compress the celiac plexus as well (Figure 6.8) as the local nerves in this area. Compression may also occur at the celiac plexus, located just under the aortic hiatus, due to dysfunction of the median arcuate ligament, in relation to the diaphragm from MALS. The psoas muscle (psoas major) shares fascia that is continuous with the median arcuate ligament. Dysfunction within this muscle may also contribute to issues at the ligament itself, or vice versa.

Some patients may have fluid retention within the abdominal space. This typically does not have the feel of fatty tissue, but increased pressure from the lymphatic fluid that may be backed up in this area. We recommend our practitioners consider this area of the median arcuate ligament, diaphragm, lymphatic vessels, and psoas muscles as a beneficial area to investigate for more efficient treatment of abdominal and lymphatic issues.

While this compression occurs in about 10-25% of the general population [14], in a few people it can cause severe symptoms. MALS seems to be problematic within the HSD/EDS community. Symptoms usually arise after eating, when putting the body in certain positions, or after exercise. Symptoms can include stomach pain and/or pressure, especially after eating, chest pain and/or pressure, nausea, diarrhea and/or constipation, bloating, vomiting, weight loss, radiating pain into the torso or back, and dysautonomia symptoms. Pain is classically in the upper abdomen after eating. It is also important to note the relationship of these structures with the diaphragm and psoas muscle as noted previously. Referral to a GI specialist should be made as soon as possible for this condition. The therapist may be able to address diaphragmatic restrictions, psoas dysfunction, or other organ and myofascial adhesions that may impact the overall symptoms as well. Much more detailed information on MALS, and conditions that present similar to "MALS," along with interventional strategies that have anecdotally worked for these authors will be provided in Volume 2.

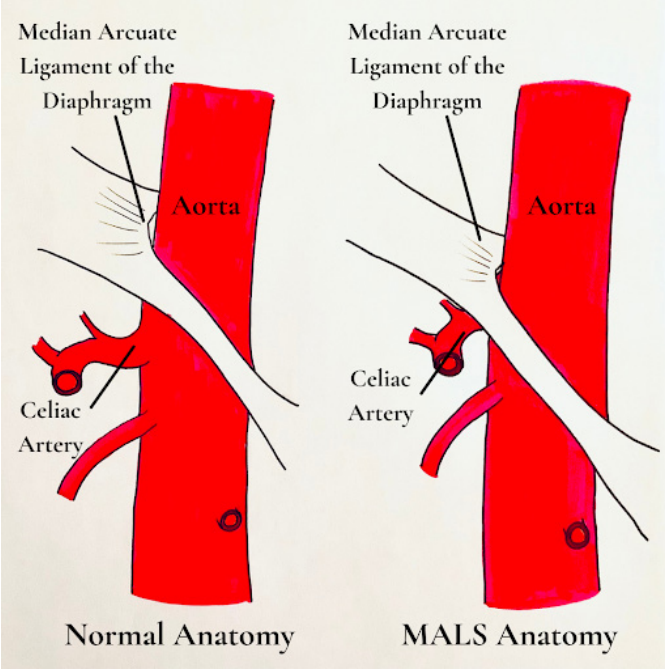

Figure 6.9 Figure of normal anatomy on the left as the aorta passes through the diaphragm and potential compression on the right seen in MALS of the celiac artery by the median arcuate ligament.