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Gastroparesis and Nutrition: The Art



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Gastroparesis, or delayed gastric emptying, is a complex disorder characterized by nausea, vomiting, early satiety, and abdominal discomfort. Complications of gastroparesis not only lead to increased morbidity, increased time spent in the hospital, and significant nutritional deficits, but also impact the overall quality of life of those affected. This article will review the anatomy and physiology of gastroparesis, discuss some of the treatment options available, and outline recommendations for nutritional monitoring and therapy for patients with gastroparesis.

INTRODUCTION

Gastroparesis (GP) is a chronic motility disorder of the stomach found in approximately 4% of the population (1). Hospitalizations with GP as the primary and secondary diagnoses more than doubled between 1995 and 2004 (2). Compared to hospitalizations for gastroesophageal reflux disease (GERD), gastric ulcers, gastritis, and nausea and vomiting, patients with GP had the longest duration of hospital stay and the highest or second highest total costs (2). By far, the greater burden, however, falls on the unfortunate patient who experiences GP. GP is frequently

accompanied by nausea, vomiting, abdominal pain and distension, as well as potentially life-threatening complications such as electrolyte imbalances, dehydration, malnutrition and poor glycemic control (if diabetes is present). In those with GP and diabetes mellitus (DM), the factors reported leading to hospitalizations were poor glycemic control, infection, medication non-compliance or intolerance, and adrenal insufficiency (3). This debilitating process alters one's ability to work, attend school, or carry out other normal daily activities. GP can affect the patient's emotional, mental, and social well being, as well as the body's ability to function normally—basically, it compromises life (4). The goal of this article is to help the clinician identify GP and provide suggestions to improve the nutritional status and overall quality of life of those who suffer from it.

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PREVALENCE AND ETIOLOGY

While most commonly associated with diabetes mellitus (DM), GP has many origins (see Table 1). The prevalence of GP is increasing—contributing to this statistic are the growing number of DM cases, the fact that those with DM are living longer, and the sheer volume of surgeries altering the vagus nerve (fundoplication, Bilroth I and II, gastric bypass surgery, heart-lung transplants, pancreatoduodenectomy, etc.). GP is a well-recognized complication of DM occurring in 25–55% of people with type 1 diabetes (DM1), 30% in type 2 diabetes (DM2), and approximately 40–50% of the hemodialysis population (5). Post-viral syndrome (23%) and idiopathic (36–68%) GP make up the bulk of the remaining cases.

DISORDERED GASTRIC EMPTYING

During a meal, the fundus serves as a reservoir and facilitates the chemical digestion of food into large particles. The antrum is mainly responsible for grinding, mixing, and trituration (process of reducing food ingested into particles <2 mm in size) (6). Nutrient emptying occurs at a rate of approximately 1–4 kcal/minute (60–240 kcal/hour) (7). Between meals, chyme is cleared from the stomach into the duodenum by the migrating motor complex (slow peristaltic waves that flush debris out of the stomach and move it along distally through the intestines until expelled in the stool).

SIGNS AND SYMPTOMS

Gastroparesis can occur in many settings with various symptoms as well as symptom severity. The clinical symptoms of GP include: decreased appetite or anorexia; nausea and vomiting; abdominal pain; bloating; fullness (especially in the morning after an overnight fast); early satiety; and halitosis. Recently, the following symptoms were identified as most prominent: nausea (92%), vomiting (84%), bloating (75%), early satiety (60%), and abdominal pain (46%) (8,9); however, severity of bloating was associated with an increased intensity of other gastroparesis symptoms, as well as a higher level of lower gut symptoms (10). These results suggest a possible association to extragastric dysmotility. Symptoms and severity of

Table 1.
Underlying Etiologies of Gastroparesis*

- Diabetes Mellitus
- Postsurgical gastroparesis
 - Fundoplication
 - Bariatric surgery
 - Bilroth I and II
 - Heart-lung transplants
 - Pancreaticoduodenectomy
- Neurological and Connective Tissue Disorders
 - Parkinson's disease
 - Multiple sclerosis
 - Amyloidosis
 - Scleroderma
 - Intestinal pseudoobstruction
- Viral-induced
- Idiopathic

*See reference 32 for a more complete list of etiologies.

disease may vary greatly from person to person and can wax and wane over time.

In those with DM, delayed gastric emptying can create erratic glycemic control as a consequence of unpredictable nutrient delivery of food into the upper gut where it is absorbed. Hypoglycemia has resulted when insulin has been administered and gastric emptying of nutrients did not follow. Conversely, acceleration of gastric emptying of nutrients with prokinetic agents has been reported to cause early postprandial hyperglycemia (7). Fluctuating glucose control in an otherwise well-controlled patient should be a red flag for GP, especially if hypoglycemia occurs *after* meal ingestion.

DIAGNOSIS

Gastroparesis is diagnosed based on upper gastrointestinal symptoms and objective evidence of delayed gastric emptying. Mechanical obstruction should always be ruled out first—this includes gastric bezoars. Bezoars are retained concretions of indigestible material, the majority of which occur as a complication of previous gastric surgery or altered gastrointestinal motility in which there is a loss of nor-

Table 2.
Factors Influencing Results of Gastric Emptying Scintigraphy

Medications—Should be stopped 2–3 days prior to study (13):

Medications that Slow Gastric Emptying

- Anticholinergics
 - Atropine
 - Glycopyrrolate
 - Hyoscyamine
 - Scopolamine
- Anticholinergics antispasmodics
 - Bentyl
 - Donnatal
 - Levsin
 - Robinul
- Tricyclic antidepressants
 - Amitriptyline
 - Amoxapine
 - Desipramine (Norpramin)
 - Doxepin
 - Imipramine (Tofranil, Tofranil-PM)
 - Maprotiline
 - Nortriptyline (Pamelor)
 - Protriptyline (Vivactil)
 - Trimipramine (Surmontil)
- Narcotic analgesics
 - Natural opiates: morphine, codeine, thebaine
 - Semi-synthetic opioids: hydromorphone, hydrocodone, oxycodone, oxymorphone, desomorphine, nicomorphine, dipropanoylmorphine, benzylmorphine, ethylmorphine, buprenorphine

- Fully synthetic opioids: fentanyl, pethidine, methadone, tramadol, dextropropoxyphene
- Adrenergic agents
 - Clonidine
- Calcium channel blockers
 - Amlodipine (Norvasc)
 - Diltiazem (Cardizem LA, Tiazac)
 - Felodipine (Plendil)
 - Isradipine (DynaCirc CR)
 - Nicardipine (Cardene SR)
 - Nifedipine (Procardia, Procardia XL, Adalat CC)
 - Nisoldipine (Sular)
 - Verapamil (Calan, Verelan, Covera-HS)
- Anti-diabetic agents
 - Pramlintide (Symlin)
 - Exenatide (Byetta)
 - Liraglutide (Victoza)

Medications that Speed Gastric Emptying

- Prokinetic drugs
 - Metoclopramide (Reglan)
 - Cisapride (Propulsid)
 - Domperidone (Motilium)
 - Erythromycin
 - Azithromycin (Zithromax, Zmax)

Other Considerations that may alter results of the test

- Cigarette smoking (should be stopped the day of the test)
- Glucose >275 mg/dL (administer insulin, or reschedule study)
- Vomiting during the study

mal peristaltic activity, compromised pyloric function, or reduced gastric acidity. Anyone who forms a gastric bezoar, by definition, has GP and further gastric emptying studies may not be necessary.

Gastric-emptying scintigraphy (GES) is currently regarded as the gold standard for measuring the rate of gastric emptying over time. There are a number of factors that can alter gastric emptying and should be addressed prior to a GES, or taken into consideration when interpreting results (see Table 2). Solid-phase GES is used to document GP (liquid GES are typically not used because even in the setting of refractory GP, liquid emptying is preserved) (11). Consensus stan-

dards for GES have been published by the American Neurogastroenterology and Motility Society and the Society of Nuclear Medicine. The recommended radiolabeled meal consists of (12):

- 118 mL (4 oz.) of liquid egg whites (e.g., Eggbeaters® [ConAgra Foods®] or an equivalent generic liquid egg white)
- Two slices of toasted white bread
- 30 g of jam or jelly
- 120 mL of water

Gastric imaging begins at 0 hours and again at 1, 2, and 4 hours after meal ingestion. Delayed gastric

emptying is defined as 90% gastric retention at 1 hour, 60% at 2 hours, and 10% at 4 hours. In those patients who have a normal percentage of gastric retention after 2 hours, extend the GES up to 4 hours as 30% of those with delayed gastric emptying might be missed (13).

It is important to note that a discrepancy exists between patient symptoms and rates of gastric emptying (4). Furthermore, gastric emptying times have a high day-to-day variability of 12–13% (14). Several authors have reported that fullness, upper abdominal pain, and reduced hunger correlate better with delayed gastric emptying than nausea or vomiting (4,10). Other diagnostic techniques to assess gastric function are available and are discussed elsewhere (15).

TREATMENT OF GASTROPARESIS

The goals of treating GP are to:

1. Identify and rectify the underlying cause
2. Achieve glycemic control in those with DM
3. Relieve or control symptoms
4. Prevent or correct fluid, electrolyte, and nutritional deficiencies

Some patients achieve relief from symptoms with only minor adjustments in their eating habits; others may have persistent nausea and vomiting and have difficulty keeping even liquids down. Attention to medication dosing and scheduling (and, when necessary, discontinuing and trying another) is important to achieve symptom relief in these patients. It is very important to review all medications that may contribute to a delay in gastric emptying and discontinue or switch to alternative drug if possible (see Table 2). In addition, the clinician will need to enlist nutritional interventions that may range from simple oral dietary modification to enteral nutrition (EN) support, and on rare occasions, even parenteral nutrition (PN) support.

Achieving glucose control will help to reduce symptoms, improve gastric emptying, and improve efficacy of prokinetic agents (7). Improvement of glycemic control can be difficult given inconsistent food ingestion (and ability to keep it down) in some patients. As hemoglobin A1C is influenced by both fasting and postprandial glucose levels, it is important

that both be addressed. Post-prandial glycemia is affected by several factors: preprandial glycemia carbohydrate content of a meal, the rate of small intestinal delivery and absorption of nutrients, insulin and glucagon secretion, and peripheral insulin sensitivity. Hyperglycemia alone exceeding 180 mg% (7) aggravates GP (16,17).

MEDICATION OPTIONS

Prokinetic Agents

Prokinetic agents amplify contractility and peristalsis of the GI tract enhancing the movement of food and fluids distally. To achieve maximum clinical effectiveness, they should be given 30 minutes before meals. Symptom improvement is the best measure of success.

Metoclopramide (Reglan). Metoclopramide has both antiemetic and prokinetic actions and is widely used in the treatment of GP. It provides symptomatic relief as an antiemetic and accelerates gastric emptying. In 2009, the FDA issued a “black box” warning against due to the risk of tardive dyskinesia. Those at greatest risk include elderly people (70 years old) and those on long term therapy defined as >3 months. Due to its potential serious side-effects, it has been proposed that the following principles be considered when using metoclopramide (4):

1. Metoclopramide should be reserved for patients with documented GP (by symptoms and gastric emptying scintigraphy).
2. Because tardive dyskinesia might be reversible with discontinuation of metoclopramide, it should be prescribed for a trial period, and the lowest effective dose for the individual patient should be sought.
3. The liquid formulation might produce more predictable plasma drug levels and permit easier dose titration.
4. An alternative approach to above is to use an orally dissolvable formulation.

Despite frequent accusations, metoclopramide does not cause diarrhea—its prokinetic effects do not extend beyond the duodenum (15).

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Table 3.
Antiemetic Agents

- Phenothiazine derivatives (i.e., prochlorperazine): prochlorperazine (Compazine), trimethobenzamide (Tigan), promethazine (Phenergan)
- Serotonin 5-HT₃ receptor antagonists: ondansetron (Zofran) and granisetron (Kytril)
- Dopamine receptor antagonists (i.e., metoclopramide)
- Histamine H₁ receptor antagonists (i.e., diphenhydramine)
- Benzodiazepines (i.e., lorazepam)
- Anticholinergics (see Table 2)

Erythromycin. Erythromycin, an antibiotic of the macrolide group, at a low dose (125 mg) is an effective stimulator of gastric emptying in GP with symptom improvement reported in 43% of patients. Liquid suspension may prove beneficial in some as it is more rapidly and reliably absorbed. A higher dose of erythromycin can be associated with nausea, vomiting, abdominal cramps, diarrhea and prolongation of the QT interval and increases the concern for the development of antibiotic resistance (15). Hyperglycemia attenuates the prokinetic action of erythromycin (18,19).

Domperidone (Motilium). Domperidone has similar effects on the upper gut to those of metoclopramide. An advantage of domperidone is that it does not readily cross the blood-brain barrier; hence, CNS side effects are minimal. The most notable side effects of domperidone include galactorrhea and amenorrhea. Although domperidone is not available in the U.S., the FDA has a program for physicians who would like to prescribe domperidone in refractory patients who have failed standard therapy.

Azithromycin (Zithromax, Zmax). Azithromycin, another macrolide similar to erythromycin, has fewer gastrointestinal side effects, improved compliance and a lower cardiac risk profile when compared to erythromycin. It has been demonstrated to accelerate gastric emptying in adult patients with chronic abdominal pain or suspected GP (20). Further work demonstrating safety and efficacy is warranted before this drug can be used in the clinical setting in patients with documented GP.

Antiemetics

Nausea and vomiting are two of the most disabling symptoms of GP. Antiemetic agents act in concert with prokinetic agents to provide symptomatic relief of nausea and vomiting (see Table 3). Too often these medications are ordered “prn” and, as such, are often not provided to the patient on a consistent basis. For a medication to be effective, it must reach its target—and to do so, it has to be taken by the patient. It may be necessary to schedule doses of these medications, at least for a time, to ensure efficacy.

Gastric Electrical Stimulation “Gastric Pacing”

Gastric electrical stimulation is emerging as a treatment option for drug-refractory diabetic or idiopathic GP (21). The surgically implanted “gastric pacemaker” device (Enterra™ —<http://www.medtronic.com/health-consumers/gastroparesis/device/index.htm>) entrains gastric myoelectric activity by means of electrodes implanted in the musculature of the gastric wall. The pacemaker attempts to restore effective gastric contractions and normal gastric emptying, as well as improve the symptoms of refractory GP. Gastric electrical stimulation has been reported to decrease GI symptoms and improve quality of life (15). Patients who have had the most favorable response to gastric electrical stimulation are those with DM reporting symptoms of nausea and/or vomiting, have not had an adequate response to antiemetic and prokinetic medications, and are not using narcotics. The most common complication is infection, resulting in device removal in 5–10% of patients (22). Symptomatic relief of GP in those using gastric electrical stimulation is not associated with significant improvement in actual gastric emptying at 6 months (23,24). Contraindications to using gastric electrical stimulation include dysmotility syndromes that involve the small intestine such as pseudoobstruction, progressive systemic sclerosis or previous gastric resection (25).

NUTRITIONAL ASSESSMENT

A thorough nutritional assessment is of primary importance in determining the appropriate plan of care for the patient with GP. Not only will it help identify the dietary modifications needed, but the degree of

malnutrition in order to distinguish between patients for whom dietary modifications are indicated versus those in whom an escalation to EN support may be warranted.

Unintentional weight loss over time is one of the most clinically useful markers of a declining nutritional status. A current, *euvolemic* weight should be compared to the patient's usual weight. Comparing weight to an ideal body weight may overestimate or underestimate the degree of nutritional risk. Various factors must be considered when evaluating the degree of weight loss over time. Patients with ongoing nausea and vomiting are at risk for dehydration. Dehydration will make weight loss appear greater than the actual amount lost (as well as put patients at risk for a variety of other complications). Therefore, weight should be evaluated after patients are rehydrated. Patients on hemodialysis due to DM are at high risk for GP, however, weight loss over time may be difficult to monitor in these patients due to fluid issues. Symptoms of nausea and vomiting are often present, but may be attributed to the underlying disease, the hemodialysis treatment, or other co-morbidities (26). Be on the lookout for a gradual decline in a patient's target weight over time and investigate complaints of nausea, vomiting, or morning fullness in this population.

A diet history can help determine what interventions may be helpful for an individual patient as well as identify eating habits that may be aggravating their GP symptoms. For example, a patient consuming a diet extremely high in fiber or fat may benefit from different food choices, whereas a patient who consumes 2–3 large meals each day may do well on a regimen of smaller, frequent meals. Factors to consider in a diet history include:

- Frequency, patterns and description of symptoms
- Types of foods tolerated/not tolerated
- Size and frequency of meals
- 3–5 day diet record
- Prior nutrition interventions (imposed by health care professional or self-imposed?)
- Use of any kind of supplements such as vitamins and mineral supplements, protein powders, liquid nutritional drinks, probiotics, fiber supplements, or herbal supplements

- Any food allergies or intolerances
- Information on dentition, any trouble chewing or swallowing food
- Medication list
 - Some medications are known to slow gastric emptying (see Table 2)
 - May need set doses as opposed to “as needed” or “prn” orders of prokinetics and antiemetics

Patients who have had a prolonged poor nutritional intake, or who have experienced a significant weight loss, are at risk for multiple nutrient deficiencies. In a recent survey of patients with GP, many nutrient deficits were documented (27). Many gastrointestinal surgical procedures put patients at risk for nutrient deficiencies due to the resulting alterations in anatomy. GP may also result from vagal nerve interruption in many of these settings (examples include: partial gastrectomy, Roux en Y anastomosis, Whipple procedure, etc.) and may further alter normal nutrient absorption, putting patients at further risk. Laboratory values can be useful in identifying certain nutritional deficits and should include:

- Vitamin D (25-OH vitamin D)
- Serum B12/ methylmalonic acid
- Ferritin (during non-acute phase)
- RBC Folate
- Glycosylated hemoglobin (if DM present)

Keep in mind that, although these are nutrients of special concern in patients with GP, many patients are at risk for general, overall nutrient deficiency. Treatment with a therapeutic vitamin /mineral supplement (2–4 weeks) may be beneficial in patients with malnutrition or known poor nutritional intake. A chewable or liquid supplement may be better tolerated than the tablet form in some patients; for others, a smaller dose (such as one-half tab, BID), may work better.

An elevated serum folate may be suggestive (but not diagnostic) of small bowel bacterial overgrowth (SBBO) in patients with generalized gastrointestinal dysmotility. Bacteria in the small bowel synthesize folate; this folate is then absorbed into the bloodstream and elevated serum levels can result (28). Those with very poor intake who are not taking a vitamin supplement are more suspect. More information on SBBO is available elsewhere (29).

Poor glucose control, especially wide swings in blood glucose levels, can exacerbate GP. In addition, hyperglycemia is a catabolic state that will thwart any efforts to improve nutritional status. Glycemic control can be monitored by patient glycemic records and a periodic glycosylated hemoglobin (HbA1C) level.

Albumin and prealbumin are not accurate markers of nutritional status and should not be used to identify or rule out malnutrition (30,31). Albumin may remain normal in patients with prolonged and severe malnutrition due to an adaptation of visceral protein stores and extravasation of fluid into the interstitium. Albumin levels are affected by factors such as infection, illness, volume overload or dehydration. Prealbumin may be affected by renal failure, use of corticosteroids, stress or illness. Both albumin and prealbumin are more indicative of an inflammatory state and severity of illness rather than the degree of malnutrition (32).

Finally, one aspect that is often overlooked is a patient's normal bowel routine. Constipation can worsen the symptoms of GP, and chronic constipation may be a sign of a more generalized intestinal dysmotility. Keep in mind, that there is often a tendency to expect daily bowel movements when, in fact, a patient's baseline habits may be different than this. Knowing a patient's baseline can change the expectations of the healthcare team and can be helpful in the event enteral feedings are initiated. It may also identify those in need of intervention, or perhaps in need of their normal bowel regimen continued during hospitalization (too often patients are put on colace nightly when they use miralax BID at home). Avoid the temptation to treat constipation with fiber—in those with small bowel dysmotility or chronic small bowel bacterial overgrowth, fiber can aggravate constipation and increase bloating and abdominal distension (33).

NUTRITION INTERVENTION

Unfortunately, there are no randomized, prospective trials available to guide clinicians in developing a nutritional care plan for the patient with GP. Many of the studies to date are small trials or observational studies involving a single meal or food, in patients with varying symptoms and different etiologies for their GP (34–40). Hence, the clinician is left with

Table 4.
Summary of Oral Diet Recommendations for Patients with Gastroparesis

- Decrease volume of meals / eat smaller, more frequent meals throughout the day
- Use more liquid calories
 - May need to switch to liquid calories over the course of the day as fullness worsens
 - If solids are not tolerated, consider a trial of a pureed/liquid diet
- Chew foods well
- Sit up for 1–2 hours after a meal
- For patients with diabetes, control blood glucose levels
- Decrease fiber in the diet (see Table 5)
 - May delay gastric emptying
 - May lead to bezoar formation
- Evaluate fat intake
 - Fat in liquid form is often tolerated
 - Fat is a good source of calories and should only be limited after other measures have been exhausted or if intake of solid fat is excessive

physiologic presumption and clinical experience until better data is forthcoming.

Oral Diet Suggestions

The diet modifications listed in this section may be helpful for the patient with GP. Keep in mind, these are not based on clinical trials or prospective randomized trials—as mentioned above, such trials are not available. Instead, the following recommendations are based on the limited data that is available as well as the author's clinical experience. See Table 4 for a summary of these suggestions (also www.ginutrition.virginia.edu under patient education materials for several different diets available for those with GP).

Smaller, More Frequent Meals

Large volumes of food are known to decrease gastric emptying (41) and may also increase gastric reflux. Patients often complain of early satiety and may feel

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better with smaller amounts of food at each sitting. If the volume of food at each meal is decreased, it will be necessary for patients to eat more often to meet their calorie and protein requirements. Six or more small meals per day may be needed for patients to take in enough food to meet their nutritional needs.

Liquids versus Solids

Liquid emptying is often preserved in patients with GP, even when solid emptying is impaired. Some patients may be able to tolerate some solids in the morning, but may need to progress to more liquid meals as the day progresses and the feeling of fullness increases. Using more liquid calories in the diet may help patients meet their nutritional needs, especially during exacerbations. Despite a higher calorie (fat) content, such liquids are often tolerated. Pureed foods may also be an option for patients having difficulty tolerating solid foods.

Decreasing Fiber

High fiber foods can delay gastric emptying and lead to early satiety in those with GP. Patients with severe dysmotility are at risk for bezoars and certain high fiber foods may be more likely to precipitate bezoar formation (see Table 5) (42). In those patients at risk for small bowel bacterial overgrowth (such as those taking proton pump inhibitors or patients with a generalized dysmotility), fiber may also exacerbate abdominal distension, gas, bloating, reflux and diarrhea. More information on bezoars is available elsewhere (42).

Fat

Patients with GP are often instructed to restrict fat in the diet. Solid meals that are high in fat may lead to a decrease in gastric emptying and discomfort in many patients. However, to eliminate fat completely from the diet takes away a significant calorie source and will require patients to eat larger volumes of food to meet their caloric needs. Patients may have difficulty taking in enough food, putting them at further nutritional risk. Clinically, we have found that fat-containing liquids are often well tolerated and can be a valuable source of nutrition for the patient with GP.

Table 5.
Foods and Medications Associated with Bezoar Formation

High Fiber Foods

- Legumes/Dried Beans (refried beans, baked beans, black-eyed peas, lentils, black, pinto, northern, fava, navy, kidney, garbanzo beans, soy beans)
- Bran/Whole Grain Cereals (bran cereals, Grape-Nuts®, shredded wheat type, granolas)
- Nuts and Seeds (pumpkin seeds, soy nuts, chunky nut butters)
- Fruits (apples, blackberries, blueberries, raspberries, strawberries, oranges, kiwi, coconuts, figs, persimmons)
- Dried fruits (apricots, dates, figs, prunes, raisins)
- Vegetables (green beans and peas, broccoli, Brussels sprouts, corn, potato peels, sauerkraut, tomato skins)
- Popcorn

High Fiber Medications/Bulking Agents

Examples include: polycarbophil (Fibercon®); inulin (FiberChoice®); methylcellulose (Benefiber®, Citrucel®); psyllium (Konsyl®, Metamucil®, Perdiem Fiber)

Other Suggestions

Patients with GP may also benefit from chewing foods well. A major function of the stomach is to grind food into smaller particles to enhance digestion. This function may be impaired or lost in the patient with GP, therefore, a focus on chewing foods well may help to compensate. Positioning may also play a role in helping patients tolerate an oral diet. Sitting upright can enhance emptying and patients may try sitting up for 1–2 hours after a meal.

See Table 4 for a summary of the diet interventions listed above. In addition, extensive dietary guidelines that may be beneficial in this population can be accessed at the University of Virginia Health System website: www.ginutrition.virginia.edu under patient education materials. Diets available include those for GP, gastric reflux, and others.

NUTRITION SUPPORT

Patients unable to maintain a healthy weight despite oral diet modifications are candidates for specialized nutrition support. We recommend setting a defined target weight—if a patient is unable to achieve this, nutri-

tion support should be discussed. Other indications for nutrition support include frequent hospitalizations for dehydration or diabetic ketoacidosis (DKA), refractory nausea/vomiting, the need for gastric decompression, or the inability to take or tolerate medications by mouth. For some patients, the burden of eating and the symptoms that follow necessitates the initiation of nutrition support to improve overall quality of life.

Most patients with GP who require nutrition support are candidates for enteral nutrition (EN). Compared with parenteral nutrition (PN), EN is associated with fewer infections and complications, is less expensive, and is less labor intensive for nursing staff and caregivers. There are times when PN may be required due to a dysmotility that extends beyond the stomach into the small bowel or colon. PN may also be indicated in a severely malnourished patient that is not tolerating EN, or is frequently NPO night after night for procedures and tests.

The optimal route for EN and type of enteral access for a patient with GP remains a source of debate. There are vented and non-vented options available. “Vented” indicates that there is a separate gastric port to drain gastric secretions; this may be helpful to relieve GP symptoms in some patients (although careful monitoring is needed if this route is chosen—see section on gastric venting below). Non-vented tubes do not offer such an option. There is little evidence to support any one route; practices vary depending on institutional preference and protocols. Options for enteral access include:

Non-vented

- Gastric tube or percutaneous endoscopic gastrostomy (PEG)
- Nasogastric, nasoduodenal or nasojejunal
- Direct percutaneous endoscopic jejunostomy (PEJ)
- Surgical or laparoscopic “J” tube

Vented

- Separate G and J tubes
- PEG with a jejunal extension (often called Jet-PEG or PEG/J)
- Nasogastric-jejunal tube; examples include:
 - Dobbhoff™ Naso-Jejunal Feeding and Gastric Decompression Tube-Covidien
 - Compat Stay-Put Nasojejunal Feeding Tube—Nestle

- Jejunal Feeding/Gastric Decompression Tube—Bard
- Silicone Gastro-Duodenal Levin Tube—Vygon, Alibaba, 3T-Medical

A trial of EN using a temporary feeding tube (such as a nasoenteric tube) may be desirable prior to the placement of a more permanent tube; however, such a trial is not always possible or practical. Placement of nasoenteric tubes by fluoroscopy is expensive, and patients that vomit frequently may very well dislodge a recently placed nasoenteric tube.

When EN is required, many patients may benefit from a PEG with a jejunal extension (often called a PEG-J or Jet-PEG). This will allow for gastric decompression to relieve nausea and vomiting while at the same time allowing for jejunal feeding and medication delivery. The tube used for the jejunal extension should be 12Fr to avoid frequent clogging, especially if the tube will be used to deliver medications. A more in-depth discussion on various enteral access techniques is available elsewhere (32).

Enteral Nutrition Initiation

When EN is initiated a step-wise approach allows differentiation between symptoms caused by the underlying GP versus those caused by enteral feeding; it also allows clinicians to establish a medication regimen to achieve glycemic control in those with DM. Our practice is to make patients strict NPO for 48 hours during EN initiation. This avoids blaming persistent symptoms of GP on enteral feeding intolerance, when it is the oral intake that is the culprit. Patients are typically initiated on a nocturnal EN infusion to mimic the home discharge plan. If DM is present, blood glucose monitoring every 4 hours *during* EN infusion for the first 48 hours helps to determine if an oral/enteral medication or insulin regimen is needed early on to maximize glycemic control. Beginning with a nocturnal infusion initially will eliminate the step of controlling blood glucose levels on a continuous regimen only to transition to nocturnal feedings prior to discharge and starting over with insulin management.

Most patients will tolerate a standard, polymeric enteral feeding. Specialized diabetic formulas for those with DM have not demonstrated an outcome

benefit to date (43). Fiber-containing formulas may cause abdominal discomfort in patients with GP and intestinal dysmotility (see section on fiber in the oral diet section above). Renal formulas are often not necessary for those with underlying renal disease/on hemodialysis as the potassium delivered in the volume needed on ≤ 2000 calories is usually within the range allowed on most renal diets (60–80 mEq/day).

ENTERAL FEEDING CHALLENGES

Refeeding

Patients who have experienced a significant weight loss or have been unable to tolerate food for a prolonged period, are at risk for refeeding syndrome (RS). RS can lead to decreased serum levels of potassium, phosphorous, and magnesium due to an intracellular

shift of glucose and electrolytes in response to endogenous or exogenous insulin. EN should be initiated slowly (15–25 calories per kilogram) and serum electrolyte levels should be monitored daily as nutrition support is initiated. Severely malnourished patients should also be provided with thiamine prior to feeding and for 3–5 days after feeding starts to prevent Wernicke's encephalopathy (44).

Patients with diabetic ketoacidosis (DKA) are especially at risk for RS due to the catabolic process. When insulin is provided to control DKA, electrolytes along with glucose shift into the cells. Extremely low serum electrolyte levels may result and electrolyte replacement is often required, sometimes for several days (45,46).

Nausea/Vomiting/Bloating

It is not unusual for patients with GP to experience an increase in nausea with the initiation of EN. EN is rarely the cause. Hyperglycemia may precipitate nausea and aggravate delayed gastric emptying; this may be an issue until a stable insulin regimen can be achieved to control blood glucose levels during EN. As mentioned earlier, making patients NPO during the initiation of the EN regimen is advisable. Small bowel bacterial overgrowth has been shown to also play a role in symptoms (47).

Diarrhea

Although frequently blamed for diarrhea in the EN-fed patient, enteral formula is rarely, if ever the "cause." Diarrhea can be multifactorial in a patient with GP (due to longstanding diabetes for one; unappreciated celiac disease for another). Clinicians should target the list of medications first and look for elixirs and liquids. The association with EN is frequently presumed because medications are often changed to liquid form and delivered via the feeding tube once enteral access is achieved. Many liquid, syrup or elixir medications contain sugar alcohols, such as sorbitol, which can precipitate diarrhea. Frequent offenders include: acetaminophen elixir, guaifenesin syrup, neotrophos powder (48). It is also

(continued on page 40)

Table 6.
Managing Problems Associated with EN Initiation and Delivery

- Refeeding Syndrome
 - Initiate EN slowly in patients at risk (15–25 calories per kilogram)
 - Monitor electrolytes regularly and replace as needed
 - Accelerated in those with resolving DKA
- Persistent Nausea and Vomiting
 - NPO status during EN initiation
 - Optimize glucose control
 - 'Venting' from gastric tube, if gastric access available
 - Optimize antiemetics and prokinetics—scheduled doses vs. "prn"
 - Consider effects of 'ileal brake' or small bowel bacterial overgrowth
- Diarrhea
 - Medications—especially liquid
 - Rule out C. Diff or other causes (small bowel bacterial overgrowth, pancreatic insufficiency, celiac disease—especially those with DM1)
- Poor glucose control
 - When possible, initiate feeding according to home/maintenance schedule so medications only need to be adjusted once
 - Monitor glucose levels every 4 hours during EN infusion and adjust insulin regimen as needed

(continued from page 38)

important to discontinue standing orders for laxatives that frequently get ordered on admission.

Gastric Venting and Reinfusion

Patients who experience persistent vomiting which results in recurrent admissions for dehydration, may benefit from a gastric venting port with their jejunal feeding tube. The use of a PEG/J tube has proved successful in keeping such patients out of the hospital (49). The gastric port allows venting of gastric secretions, while the jejunal port allows both feeding and maintenance of hydration. However, monitoring these patients closely is very important as they can become dehydrated with a severe metabolic alkalosis if vented secretions are not replaced.

For patients that vent from the gastric port and have an output of >500 mL per day, reinfusion may be an option. Reinfusing gastric secretions—a rich source of fluid and electrolytes—may help to prevent dehydration and electrolyte abnormalities. A detailed article is available describing this practice (50). Table 6 summarizes trouble-shooting for those on EN.

Table 7. Resources

- University of Virginia Health System GI Nutrition Website: www.ginutrition.virginia.edu Find patient education materials including:
 - Gastroparesis
 - Short version
 - Long version
 - Renal
 - Diabetes
 - Spanish Version (Dieta Para Gastroparesia)
- Other Websites:
 - Gastroparesis and Dysmotilities Association <http://digestivedistress.com>
 - International Foundation for Functional Gastrointestinal Disorders (IFFGD) <http://www.aboutgimotility.org/>
 - American Association of Gastrointestinal Motility Disorders www.agmd-gimotility.org

SUMMARY

Gastrointestinal symptoms are common in patients with DM and significantly impair quality of life. Gastroparesis is one of the more debilitating complications associated with both DM1 and DM2. Identifying and treating both of these issues will go a long way to improve the overall well-being of these patients. A list of further resources is available in Table 7. ■

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