Carol Rees Parrish, RD, MS, Series Editor

Nutrition Intervention for the Patient with Gastroparesis: An Update

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Gastroparesis, or delayed gastric emptying, has many origins. The clinical presentation may wax and wane depending on the underlying etiology. However, once a patient develops protracted nausea and vomiting, providing adequate nutrition, hydration and access to therapeutics such as prokinetics and antiemetics can present a unique challenge to clinicians. This article provides suggested guidelines to assess the nutritional status of patients with gastroparesis and strategies to treat the nutritional issues that arise in this patient population.

INTRODUCTION

Evaluation of nutritional status and the treatment of malnutrition are important factors in the management of patients with gastroparesis. Symptoms of gastroparesis (Table 1) may be severely debilitating and the resultant aberrations in nutritional status can be life threatening. Once a patient develops protracted nausea and vomiting, providing adequate nutrition, hydration and access to therapeutics such as prokinetics and antiemetics can present a unique challenge to clinicians.

Gastroparesis has many origins and its clinical presentation may wax and wane depending on the underlying etiology (see Table 2 for conditions associated with gastroparesis). Many patients (and some clinicians) assume that a diagnosis of gastroparesis means continuous clinical deterioration until an end-stage is reached. Research to date, however, supports that early nutrition support can reverse significant malnutrition while gastric function returns over time. In truth, many patients with refractory gastroparesis who initially require jejunal feeding tube placement for nutrition support often eventually eat again on their own (1–5).

(continued on page 33)

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(continued from page 29)

Table 1
Clinical Symptoms of Gastroparesis

- Decreased appetite / anorexia
- Nausea and vomiting
- Bloating
- Fullness (especially in the morning after an overnight fast)
- Early satiety
- Halitosis
- Post-prandial hypoglycemia, or fluctuating glucose levels in an otherwise well-controlled patient with diabetes mellitus

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Although prokinetic agents and antiemetics are front line therapy in the treatment of gastroparesis (6), the purpose of this article is to provide strategies to maintain or restore nutritional status in this patient population. There is a scarcity of clinical trials in the area of nutrition intervention for patients with gastroparesis. Review articles and textbooks are available, however, evidence-based nutrition recommendations are lacking. Most of the current dietary guidelines and restrictions have been developed from studies evaluating the effect of a single parameter on gastric emptying in normal subjects (7).

This article provides practical guidelines to assess the nutritional status of patients with gastroparesis and strategies to treat nutritional issues that arise in this patient population. More detailed reviews of all facets of gastroparesis are available elsewhere (8,9).

NUTRITION ASSESSMENT

The purpose of nutritional screening and evaluation in the patient with gastroparesis is to objectively distinguish the adequately nourished patient who can pursue further gastrointestinal (GI) evaluation and/or prokinetic trials, from a malnourished patient who requires immediate nutritional support.

Weight Change Over Time

Unintentional weight loss over time is probably the most important, noninvasive parameter to assess overall nutritional status in the patient with gastroparesis.

Table 2
Clinical Conditions Associated with Gastroparesis (53,54)

Mechanical obstruction
Duodenal ulcer
Pancreatic carcinoma or pseudocyst
Gastric carcinoma
Superior mesenteric artery syndrome

Metabolic/endocrine disorders
Diabetes Mellitus
Hypothyroidism
Hyperthyroidism
Hyperparathyroidism
Adrenal insufficiency (Addison's disease)

Acid-peptic disease
Gastric ulcer
Gastroesophageal reflux disease

Gastritis
Atrophic gastritis
Viral gastroenteritis

Post-gastric surgery
Vagotomy
Antrectomy
Subtotal gastrectomy
Roux-en-y gastrojejunostomy
Fundoplication

Disorders of gastric smooth muscle
Scleroderma
Polymyositis
Muscular dystrophy
Amyloidosis
Chronic idiopathic pseudoobstruction
Dermatomyositis
Systemic lupus erythematosus (SLE)

Psychogenic disorders
Anorexia
Bulimia
Depression

Neuropathic disorders
Parkinson's disease
Paraneoplastic syndrome
CNS disorders
High cervical cord lesions (C4 and above)

Table 3
Evaluation of Weight Change Over Time

$$\% \text{ Weight Change} = \frac{\text{Usual Weight} - \text{Actual Weight}^{*}}{\text{Usual Weight}} \times 100$$

	Significant Malnutrition	*Severe Malnutrition*
1 week	1%–2%	>2%
1 month	5%	>5%
3 months	7.5%	>7.5%
6 months	10%	>10%

*Compare the patient's UBW to their euvolemic current actual weight.
Adapted from Shopbell JM, Hopkins B, Shronts EP. Nutrition screening and assessment. In: Gottschlich M, ed. *The Science and Practice of Nutrition Support: A Case-Based Core Curriculum.* Dubuque, IA: Kendall/Hunt Publishing Company, 2001:119, with permission from the American Society for Parenteral and Enteral Nutrition (A.S.P.E.N.). A.S.P.E.N. does not endorse the use of this material in any form other than its entirety.

When an accurate weight can be obtained, this parameter is a simple, reliable indicator of nutritional status. An unintentional ≥7.5% loss of usual body weight over a three-month period signals significant malnutrition and should be a cause for concern.

It is important to compare a patient's current **actual** weight (AW) to their **usual** body weight (UBW) to determine nutritional risk and/or whether significant weight loss has occurred. To compare the patient's actual weight to an *ideal* body weight might either grossly over- or underestimate the true weight loss, and therefore the severity of malnutrition. Another essential principle is to assure that the patient's actual weight represents a "euvolemic" weight, neither dehydrated, nor edematous. As an example, a patient with diabetes mellitus (DM) who presents with vomiting, diarrhea and poor glucose control may have a falsely low actual weight due to dehydration. Failure to use a euvolemic actual weight might overestimate the amount of weight loss over time and suggest significant malnutrition instead of the fact that the patient is merely dehydrated. Finally, it is also imperative to remember that those patients who are clinically overweight or obese, yet have unintentionally lost a significant amount of weight over a short time interval, may carry the same risk profile as a chronically undernourished patient.

The time course of weight loss is also important. Table 3 demonstrates that both a 2% loss over 1 week and a ≥10% loss over 6 months both constitutes severe malnutrition, which is associated with increased morbidity and mortality (10). Beware of the hemodialysis patient who experiences serial drops in their target weight over time; 50% of patients on dialysis are a result of long-standing DM, which is commonly associated with gastroparesis.

Although unintentional weight loss over time is the best indicator of the severity of malnutrition, some investigators utilize weight alone as a measure of a patient's nutritional status. Patients may be deemed mal-

(continued on page 39)

Table 4
Risk of Associated Disease According to BMI and Waist Size

*BMI**	*Category*	*Waist less than or equal to 40 in. (men) or 35 in. (women)*	*Waist greater than 40 in. (men) or 35 in. (women)*
18.5 or less	Underweight	—-	N/A
18.5–24.9	Normal	—-	N/A
25.0–29.9	Overweight	Increased	High
30.0–34.9	Obese	High	Very High
35.0– 39.9	Obese	Very High	Very High
40 or greater	Extremely Obese	Extremely High	Extremely High

*These values may underestimate the degree of malnutrition in some patients. An overweight or obese patient may be malnourished if significant weight loss has occurred, but not fall into the category of malnutrition based on BMI alone.
Obtained from http://www.consumer.gov/weightloss/bmi.htm (Accessed 7-1-05).

(continued from page 34)

nourished based on a stable weight below normal, loss of an arbitrary amount of weight, or loss of a significant percentage of baseline weight. Commonly, ideal body weight for an individual is determined based on weight relative to height. The Body Mass Index (BMI) is determined as weight (kg)/ height $(m)^2$ (see Table 4 or go to: http://nhlbisupport.com/bmi/bmicalc.htm) (11). A BMI of 20 to 25 is considered to be normal. Most guidelines identify patients at nutritional risk if they:

- Are <80% of ideal weight
- Have a body mass index less than 20
- Have lost 5% of baseline weight or 5 pounds in one month
- Have lost 10 pounds or 10% of usual body weight in 6 months.

Diet History

A diet history can be very helpful in identifying patients who might benefit from nutrition support and to determine the level of nutrition support required. As an example, a patient who develops nausea while eating the usual, three large meals per day, may not require supplemental calories, but may benefit by simply instituting smaller, more frequent meals. In contrast, the patient with severe gastroparesis who has significant vomiting after the ingestion of water may require gastric decompression and enteral jejunal feeding to provide symptom relief, nutrients, fluids, and medications. When obtaining a diet history, be sure to evaluate for:

- Changes in appetite, nausea/vomiting/diarrhea.
- Problems chewing and/or swallowing which can affect their ability to ingest certain foods.
- The patient's typical daily dietary intake.
- The use of supplemental nutrition (oral, enteral or parenteral).
- Food intolerances or allergies.
- Use of supplements, such as vitamins, minerals, herbs or protein powders.
- Use of stool bulking agents or laxatives.
- Medications known to slow gastric emptying (Table 5).

Laboratory Data

Intolerance to various foods/food groups and malabsorption can lead to nutrient deficiencies, which can further aggravate clinical morbidity. Intolerances can often be managed with dietary manipulation and close nutrition follow-up. Nutrient deficiencies, particularly those resulting in anemia and metabolic bone disease, require ongoing monitoring and supplementation. Laboratory values are a useful adjunct in the initial evaluation and continued management, of the patient with gastroparesis. Initial assessment of a patient with gastroparesis should include:

- Glucose and glycosylated hemoglobin (HgbA1C) if the patient has DM
- Ferritin
- Vitamin B_{12}
- 25-OH vitamin D (particularly with longstanding gastroparesis or in the post- gastrectomy patient)

Table 5
Medications Known to Delay Gastric Emptying (6,53)

- Aluminum-containing antacids
- Anticholinergics
- Atropine
- Beta agonists
- Calcitonin
- Calcium channel blockers
- Dexfenfluramine
- Diphenhydramine
- Ethanol
- Glucagon
- Interleukin-1
- L-dopa
- Lithium
- Octreotide
- Ondansetron
- Narcotics
- Nicotine
- Potassium salts
- Progesterone
- Sucralfate
- Tricyclic antidepressants
- Selective serotonin reuptake inhibitors (SSRI)

Serum glucose and $HgbA_1C$. Glycemic control is critical in the management of diabetic gastroparesis. Hyperglycemia (>200 mg %) can cause transient gastroparesis in some patients and this delayed gastric emptying can respond quickly to normalization of serum glucose levels (12,13). Additionally, hyperglycemia has been shown to attenuate the prokinetic effect of erythromycin. Finally, hyperglycemia is a catabolic process, which ultimately thwarts the efforts of nutrition repletion. Glycemic control must therefore be carefully evaluated at the initial nutritional assessment and monitored regularly during the repletion process.

An elevated glycosylated hemoglobin at initial evaluation may suggest that improved glycemic control might attenuate the gastroparesis, and that the gastroparesis is

the likely culprit causing poor glycemic control. Subsequent monitoring of the glycosylated hemoglobin assures that optimal glucose control is maintained.

Ferritin. Iron-deficiency anemia is common in this patient population. The etiology is likely multifactorial. Serum iron levels may be marginal especially in menstruating women who are unable or prefer not to consume red meat. At our institution, we have anecdotally noted that many patients with gastroparesis report intolerance to red meat and voluntarily remove it from their diet.

Iron absorption is significantly enhanced by gastric acid. Reduced gastric acidity impairs the conversion of dietary ferric iron to the more absorbable ferrous form (14). Gastroparesis symptoms, especially concomitant acid reflux, are often treated with acid suppressive medications such as proton pump inhibitors. Vagotomy also reduces acid production, which is necessary for efficient iron absorption. Elevated gastric pH and poor motility can also increase the patient's risk of developing small bowel bacterial overgrowth, which can significantly decrease duodenal iron absorption. Iron deficiency should be carefully monitored in patients receiving jejunal enteral nutrition as the duodenum (primary site for iron absorption) is bypassed. Similarly, iron deficiency is also of particular concern in patients whose gastroparesis results from post-vagotomy syndrome, especially those with a gastrojejunostomy anastomosis (15). Low gastric acidity, combined with duodenal exclusion, results in impaired iron uptake. Iron deficiency may manifest more quickly following Billroth II than in Billroth I procedures, for this same reason (16). A decade following gastrectomy, iron deficiency is the most frequently reported nutrient deficiency (17).

A ferritin level is an accurate indicator of iron stores over time (18). However, ferritin is an acute phase reactant, therefore it should be checked in the non-acute setting (absence of infection, inflammation, etc.). Ferritin levels may be low even in the setting of a normal hematocrit, as the body utilizes iron stores while preserving hemoglobin and hematocrit. In one small study, 67% of patients with gastroparesis were found to have a low ferritin level despite low-normal hemoglobin and hematocrit levels (2).

Table 6
Guidelines for Iron Replacement in Adults (24)

1. 150–300 mg of elemental iron per day should be given in three divided doses.
2. Four to six months of oral iron therapy is needed to reverse uncomplicated iron deficiency anemia.
3. Sustained released preparations should not be crushed or chewed.
4. Absorption is enhanced when iron is taken on an empty stomach but GI intolerance may necessitate administration with food.
5. GI discomfort may be minimized with slow increase to goal dosage. Start with 1/4 to 1/2 dose two to four times daily if necessary; some replacement is better than none.
6. Do not take iron supplements within two hours of taking a dose of tetracycline or fluoroquinolone.
7. Drink liquid iron via a straw to minimize dental enamel stains.
8. Following Billroth II and total gastrectomy, sustained release or enteric-coated iron preparations may not be optimal as available iron is delivered past the duodenum following these surgeries.

Adapted with permission from Drug Facts and Comparisons www.efactsweb.com

Oral iron supplementation is the preferred method of replacement (19) and is available as ferrous sulfate, gluconate or fumarate. Optimal dosing is approximately 200 mg of elemental iron daily (18). Iron replacement therapy is typically administered three times daily, preferably 6 hours apart. The addition of very small amounts of vitamin C (25–50 mg) can enhance iron absorption (20). Patients can obtain this amount of vitamin C with 3 ounces of a vitamin C containing juice or beverage, either orally or via a feeding tube (given with the iron dose). Chewable or liquid iron is the preferred iron replacement in post-gastrectomy patients as solubilization of iron tablets may not be complete, resulting in poor absorption of the supplement (21).

Gastrointestinal side effects (nausea, abdominal pain, constipation or diarrhea) often decrease patient compliance to iron therapy. Advising patients to take iron with food can reduce these GI symptoms. Because the body increases its avidity for iron uptake (up to 20%–30%) in deficient states, even modest iron intake is better than none at all (18). Reducing the dosage or

(continued on page 42)

(continued from page 40)

Table 7
Elemental Iron Content of Various Iron Formulations (24)

Product	*Dose*	*Elemental Iron Content (mg)*	*Comments*
Tablets			
Ferrous Sulfate	325 mg	65	
Ferrous Gluconate	325 mg	36	
Ferrous Fumarate	325 mg	106	
Suspension			
Ferrous Sulfate	Elixir 220 mg/5 mL	44 mg/5 mL	May contain sorbitol
Ferrous Sulfate	Drops 75 mg/0.6 mL	15 mg/0.6 mL	May contain sorbitol
Feostat (ferrous fumarate)	100 mg/5 mL	33 mg/5 mL	Butterscotch flavor
Chewable tablets			
Feostat (ferrous fumarate)	100 mg	33	Chocolate flavor

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decreasing the frequency of administration may prevent patients from completely discontinuing their iron supplementation. Patients should also be encouraged to increase their intake of iron-rich foods. Meats (heme iron sources) are the most readily absorbed form of dietary iron.

Parenteral iron replacement is often unnecessary and should be reserved for patients with severe iron deficiency who cannot tolerate oral replacement. Parenteral replacement is expensive and its risks include anaphylactic shock. See Tables 6 and 7 for further information on iron supplementation.

Table 8
Guidelines for Vitamin B_{12} Supplementation (24)

Mild deficiency—over the counter oral vitamin B_{12} (500–1000 mcg/day).
- The amount absorbed decreases with higher doses.

Severe deficiency—vitamin B_{12} intramuscular (IM) or subcutaneous (SC) 100–200 mcg/month
- 1000 mcg dose is often used, however, the percentage of vitamin B_{12} retained decreases with larger doses.

Intranasal B_{12} should be limited to patients in remission following IM B_{12} injection.
- Recommended dose is 500 mcg once weekly.

Monitor B_{12} levels at baseline and then every 3 months until normalized, then annually.

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Vitamin B_{12}. Vitamin B_{12} deficiency is common in patients following partial or total gastrectomy. Intrinsic factor is synthesized in the stomach and is complexed to vitamin B_{12} to facilitate absorption in the terminal ileum. Reduced levels of intrinsic factor and gastric acid following gastrectomy impairs the cleavage of protein bound B_{12} resulting in little or no intestinal absorption. Bacterial overgrowth and reduced intake of vitamin B_{12} rich foods also contribute to a deficiency (22). The resulting anemia can be severe and often presents as a late complication of gastric resection. Lassitude, fatigue, chills, numbness in the extremities, dizziness and neurological symptoms are also common symptoms of vitamin B_{12} deficiency (23). Clinical features are non-specific and often absent in deficient patients. Baseline and periodic monitoring of vitamin B_{12} levels are therefore important.

Vitamin B_{12} supplements are available in oral, transnasal or intramuscular (IM) preparations. Following total gastrectomy, enteral vitamin B_{12} therapy will increase serum B_{12} levels (24). Symptom resolution is comparable in patients who receive enteral versus parenteral supplementation. Table 8 outlines guidelines for monitoring and replacing vitamin B_{12}. The decision to supplement B_{12} via oral, intranasal or intramuscular approach should be based on patient compliance. Table 9 provides a cost comparison for various vitamin B_{12} supplements.

25-OH vitamin D. The literature has shown that patients with gastrectomies (sub-total or total) can have accelerated bone loss, therefore increasing the risk for osteoporosis. A percentage of patients who undergo subtotal gastrectomy subsequently develop

Table 9
Cost Comparison of Vitamin B_{12} Supplements

B_{12} Formulation	*# Doses/ month*	*Average Cost Per Month**
Intranasal Nascobal®	4	$34.80 (500 mcg dose)
IM injection (cost of syringes not included)	1	$0.79 (1000 mcg dose)
Capsule	30	$0.76 (1000 mcg dose)

*Prices from Wal-Mart 2003
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gastroparesis. Osteopenia and osteomalacia are also not uncommon in this population (25). Low bone mineral density has been reported in 27%–44% of these patients (25), many of whom had normal serum calcium and alkaline phosphatase levels. Klein, et al found that vertebral compression fractures were three times as common in men who had undergone Billroth II surgery compared with controls (26). Age of the patient and bone mineral density (BMD) at the time of surgery play a significant role in the development of bone disease, independent of the type of gastric resection. The etiology of bone disease in this population is thought to be due to decreased intake of calcium, vitamin D and lactose-containing foods coupled with altered absorption and metabolism (14,26,27). Evaluation of 25-OH vitamin D levels (*not* 1, 25-OH_2 vitamin D) and bone mineral density may also be beneficial in patients with gastroparesis. Dual energy x-ray absorptiometry (DXA) provides an inexpensive, reproducible method to determine BMD (28). Given the frequency of bone disease in these patients, it is reasonable to monitor BMD (even in the setting of normal laboratory values), at baseline and then every one to two years. It is imperative to identify and treat high-risk patients early (i.e., young women with amenorrhea due to significant weight loss from debilitating gastroparesis) in order to reduce incident fractures.

Currently, there are no accepted calcium and vitamin D supplementation guidelines for gastroparesis or post-gastrectomy patients. Multivitamin/mineral tablets contain varying amounts of calcium and vitamin D; therefore additional supplementation is often required. Patients with bone disease are recommended to take 1500 mg calcium and 800 IU of vitamin D

(continued on page 47)

Table 10
Common Calcium Supplements

Calcium Carbonate: 40% elemental calcium

Brand	*Elemental Calcium (mg) per tablet*	*Approximate cost per tablet*	*Comments*
Tums®	200	$.02	
Extra Strength Tums®	300	$.04	
Oscal® 500	500	$.10	Also available with 200 IU vitamin D Also available in 250 mg dose
Caltrate® 600 Plus®	600	$.11	Contains 200 IU vitamin D plus additional minerals
Viactiv®	500	$.10	Contains 100 IU vitamin D and 40 mcg vitamin K Contains <0.5 g lactose per dose 20 calories per piece

Calcium Citrate: 21% elemental calcium (25)

Citrical®	200	$ .07	
Citrical® + D	315	$.11	Contains 200 IU vitamin D

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(continued from page 43)

daily (29). To maximize absorption, calcium should be administered in single doses no greater than 500 mg. Patients should also be encouraged to include calcium rich foods in their diet along with calcium supplements as tolerated (Table 10).

Anti-resorptive agents (calcium, vitamin D, calcitonin and bisphosphonates) and bone-formation agents (recombinant hormone PTH) may all be considered to treat bone loss.

Albumin, prealbumin and/or transferrin were often used in the past as markers of a patient's nutritional status. Today we know that levels of these proteins can be significantly altered by a multitude of factors, thus eliminating their validity as markers of nutritional status (30).

Albumin. Serum albumin levels are a poor measure of a patient's nutritional status and they can be especially misleading in patients with gastroparesis. Patients may have significant malnutrition, yet maintain intact visceral protein stores (31). Serum albumin levels can also appear to be normal due to extravasation of albumin from the interstitium into the vascular space with starvation. Low serum albumin levels can be caused by many conditions other than malnutrition such as urinary wasting due to nephrotic syndrome; it is also a negative acute phase reactant. See Table 11 for other factors that alter serum albumin levels.

Prealbumin. In healthy individuals, prealbumin levels decline during periods of decreased intake and normalize within two days upon resumption of nutrition. Thus prealbumin is primarily utilized to monitor the effectiveness of nutritional intervention. Prealbumin levels can decrease with hyperglycemia or in other catabolic states (e.g. inflammation/infection). Patients with poorly controlled DM may have low values that do not reflect the patient's true nutritional status; in addition, prealbumin is lost via the kidney in the setting of nephritic syndrome, common in patients with long-standing DM. Other factors that affect prealbumin levels are presented in Table 12.

Transferrin. In the past, serum transferrin levels were used to assess visceral protein status. Due to the many factors that affect these levels and the eight-day half-life,

Table 11
Factors Affecting Serum Albumin Levels

Increased in:
- Dehydration
- Marasmus
- Blood transfusion
- Exogenous albumin

Decreased in:
- Overhydration/ascites/eclampsia
- Hepatic failure
- Inflammation/infection/metabolic stress
- Nephrotic syndrome
- Protein-losing states
- Burns
- Trauma/postoperative states
- Kwashiorkor
- Collagen diseases
- Cancer
- Corticosteroid use
- Bedrest
- Pregnancy
- Zinc deficiency

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Table 12
Factors Affecting Serum Prealbumin Levels

Increased in:
- Severe renal failure
- Corticosteroid use
- Oral contraceptive use

Decreased in:
- Acute catabolic states
- Post-surgery
- Liver disease/hepatitis
- Infection/stress/inflammation
- Dialysis
- Hyperthyroidism
- Sudden demand for protein synthesis
- Nephrotic syndrome
- Significant hyperglycemia (catabolic state)
- Pregnancy

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Table 13
Factors Affecting Serum Transferrin Levels

Increased in:
- Iron deficiency
- Dehydration
- Pregnancy (third trimester)
- Oral contraception/ Estrogens
- Chronic blood loss
- Hepatitis
- Hypoxia
- Chronic renal failure

Decreased in:
- Pernicious anemia (B_{12} deficiency)
- Anemia of chronic disease
- Folate deficiency anemia
- Overhydration
- Chronic infection
- Iron overload/iron dextran therapy
- Acute catabolic states
- Uremia
- Nephrotic syndrome (permeability of glomerulus)
- Severe liver disease/hepatic congestion
- Kwashiorkor
- Age
- Zinc deficiency
- Corticosteroids
- Cancer
- Protein

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serum transferrin is not a good indicator of protein status. See Table 13 for factors that affect transferrin levels.

KEY FACTORS AFFECTING GASTRIC EMPTYING

The key factors affecting gastric emptying are presented in order of clinical importance below. These factors are reported to slow gastric emptying, however, their clinical significance has yet to be proven in prospective, randomized, controlled trials. Physicians and dietitians may successfully manipulate them in an effort to improve gastric emptying.

The patient's diet history will help determine which factors may be integral to dietary tolerance for a particular patient. As an example, if a patient with gastroparesis has better tolerance of liquids than solids, convert the vast majority of nutrients and calories to a more liquid form. Provide medications, specifically prokinetics, in a liquid form also. It may behoove the clinician to start prokinetic therapy and change only one or two parameters at a time. This technique often allows patients to tolerate their diet and medications, and ultimately restore nutritional status. Avoid overzealous intervention of all of these factors at one time (i.e. "the shotgun approach") because it often results in unnecessary limitations of the patient's dietary and caloric needs, which can further aggravate the nutritional decline.

Volume

The primary influence on gastric emptying is volume—the greater the volume, the slower the emptying. Early satiety is one of the hallmarks of gastroparesis. Because larger volumes slow emptying, smaller, more frequent meals may enable patients to tolerate their diet and achieve adequate caloric intake (32).

Liquids Versus Solids

Functionally, the stomach can discern between liquids and solids. In normal subjects, antral peristaltic waves occurring three times per minute can deliver approximately 30 mL to the small bowel with each contraction. Patients with gastroparesis will often have preserved emptying of liquids even when they have a clinically documented delay in the emptying of solids. Liquids empty by gravity and do not require antral contraction to leave the stomach (32). Liquids, even those that are highly caloric, will empty from the stomach. Pureed foods become liquified after mixing with saliva and gastric secretions, and may be more easily tolerated than solid foods. A trial diet primarily of pureed food or liquids can be designed to meet a patient's nutritional requirements.

Patients with gastroparesis often report increased fullness and bloating with subsequent meals over the course of the day. As a result, patients often avoid eating later in the day and this can exacerbate malnutrition. Transitioning to more liquid calories towards the end of the day may be useful to alleviate these symptoms while continuing to provide appropriate nutrition.

(continued on page 50)

(continued from page 48)

Medications

Many medications can delay gastric emptying, and their use can significantly exacerbate idiopathic or diabetic gastroparesis (Table 5). The patient's medication list should be reviewed regularly and avoid prescribing drugs that aggravate gastroparesis.

Hyperglycemia

Hyperglycemia (glucose >200 mg %) is known to worsen the symptoms of gastroparesis. In the patient with DM, it is not often clear whether the gastroparesis is negatively affecting the glucose control or vice versa. Wide fluctuations in blood glucose impair gastric emptying more so than continuously elevated glucose levels. In addition, hyperglycemia can diminish the prokinetic effects of erythromycin (33,34). Maintenance of glycemic control is imperative to maximize utilization of the nutrition that is provided.

Table 14
High Fiber Foods/Medications and Foods Associated with Bezoar Formation*

High Fiber Foods

- **Legumes/Dried Beans**. Refried beans, baked beans, black-eyed peas, lentils, black, pinto, northern, fava, navy, kidney, and 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.** Dried fruits (apricots, dates, figs,* prunes, raisins), blackberries* blueberries* raspberries* strawberries* oranges, apples* kiwi, coconuts* persimmons*
- **Vegetables.** Green peas, broccoli, Brussels sprouts* green beans* corn* potato peels* sauerkraut* tomato skins*

High Fiber Medications/Bulking Agents

- Acacia fiber
- Benefiber
- Citrucel
- FiberChoice
- Fibercon
- Konsyl
- Metamucil
- Perdiem

*Foods Associated with Bezoar Formation
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Fiber

Fiber, particularly pectin, can slow gastric emptying. Fiber is also poorly digestible and increases a patient's risk of forming a bezoar (35). Although the avoidance of high fiber foods is recommended in patients with gastroparesis, what is not known is the type of fiber and the quantity of fiber that should be withheld. Over-the-counter fiber/bulking laxatives (Table 14) should probably be discontinued (36). Fiber-containing jejunal enteral feedings are generally well tolerated, unless small bowel bacterial overgrowth is present as unabsorbable fiber may theoretically aggravate symptoms due to fermentation.

Fat

Fat is a potent inhibitor of gastric emptying (32), however, many patients are not affected by dietary fat if it is present in liquid form (e.g. whole milk, milkshakes, nutritional supplements, etc.) Avoid manipulating fat calories as a first line endeavor because fat in liquid form is often well tolerated (and pleasurable), and it provides high-density calories in a smaller volume.

Osmolality

Osmolality has been shown to slow gastric emptying in various study populations, however it is overrated in terms of its clinical significance (37). Most patients with gastroparesis can easily tolerate a clear liquid diet. The standard clear liquid diet has an osmolality range from 500–1200 mOsm (isotonic = 300 mOsm). Sherbet has an osmolality of approximately 1225 mOsm; juices are 700–950 mOsm. Liquid metoclopramide, a commonly used prokinetic agent in this patient population, has an osmolality of 5400 mOsm. Overall, osmolality is believed to be a non-issue (versus volume or fiber) to manipulate when attempting to nourish these patients.

IMPORTANT FACTORS THAT CAN AFFECT ORAL INTAKE IN THE PATIENT WITH GASTROPARESIS

Nausea and Vomiting

Treatment of nausea and vomiting is paramount to providing successful oral nutrition and in preventing electrolyte and acid-base abnormalities. Antiemetics and

(continued on page 52)

(continued from page 50)

prokinetic agents are the mainstay of treatment in gastroparesis. When administering these useful medications, regular, scheduled dosing and the use of liquid preparations provide the most consistent symptom control. Scheduled dosing (versus "as needed" dosing) assures that continuous, consistent levels of medication are provided to the patient. Provision of liquid forms of these drugs in patients with delayed gastric emptying also assures more accurate and consistent drug delivery as liquids empty readily via gravity without the need for antral peristalsis. From a clinical perspective, if the patient does not respond to this therapy, intravenous medications, or more distal enteral delivery will be required. In the patient who requires gastric decompression concurrent with jejunal nutrient infusion, all medications should be delivered via the jejunal port to promote maximal absorption. Tablet or pill forms of medications should be crushed into a fine powder with careful flushing of the tube before and after dosing to keep the jejunal tube patent.

Note: Medications should be checked from a pharmacological standpoint to assure that they may be crushed and that they are efficacious when delivered into the jejunum.

Small Bowel Bacterial Overgrowth

Patients with gastroparesis are at high risk of developing small bowel bacterial overgrowth (SBBO). Peristalsis and normal gastric acid production typically prevents the colonization of bacteria within the small bowel. In the setting of gastric dysmotility, bacteria colonize the relatively sterile small bowel. Because many patients with gastroparesis are concurrently treated with potent acid inhibitors to reduce reflux symptoms, the resultant hypochlorhydria also significantly predisposes these patients to develop SBBO.

Bacterial colonization of the small bowel results in a mucosal inflammatory process that impairs nutrient absorption. Intestinal bacteria also compete with the host for available nutrients. They deconjugate bile salts, altering micelle formation, which results in fat malabsorption. Bacterial metabolism also produces short chain fatty acids that are poorly absorbed in the small bowel. These decrease luminal pH, hindering intestinal enzymes and increasing the overall osmotic load. The endpoint is increased gut transit, maldigestion and malabsorption.

Symptoms of SBBO include gas, bloating, abdominal distension, nausea, diarrhea, weight loss and an overall decline in nutritional status. These symptoms may mimic those of gastroparesis, thus it is imperative to consider SBBO in every patient with gastroparesis (38,39). Enterally provided antibiotics are generally the treatment of choice. The most frequently utilized antibiotics are metronidazole, ciprofloxacin, amoxicillin/clavulanate or doxycycline. However, rifaximin is being used with increasing frequency (40). For persistent SBBO, monthly rotating antibiotics may be necessary.

Ileal Brake

The ileal brake is the primary inhibitory feedback mechanism that acts to control the transit of a meal through the gastrointestinal tract. This distal gut inhibitory feedback system slows the speed of gastrointestinal transit in response to a meal. Nutrients, particularly fatty acids, are believed to be the main activator of the ileal brake. Teleologically, this gut "traffic brake" regulates the speed of luminal peristalsis in the GI tract to maximize the absorption of nutrients (41). Clinically, the ileal brake may play a role in increased GI symptoms that develop when jejunal tube feedings are initiated. Exacerbation of nausea and vomiting may be attributed to stimulation of the ileal brake by fats that have escaped more proximal absorption in the small bowel. This phenomenon should not be confused with intolerance to jejunal feeding or abnormal small bowel dysmotility. It is conceivable that SBBO can aggravate the ileal brake due to the resultant malabsorption. Treatment should be aimed at slowing the rate of tube feeding for a short period of time and treating SBBO if present.

Bezoar Formation

Bezoars are retained concretions of indigestible foreign material that accumulate in the stomach. Patients with altered gastrointestinal anatomy and/or motility are at increased risk for developing bezoars, thus patients with gastroparesis are particularly predisposed

(continued on page 55)

(continued from page 52)

toward this complication (35). Clinicians should be mindful of bezoar formation because when present, the symptoms often mimic that of gastroparesis. Patients may present with early satiety, nausea and vomiting. Failure to recognize the presence of a bezoar may further compromise the patient's nutritional status.

Phytobezoars are composed of nondigestible food material including cellulose, hemicellulose, lignin, and fruit tannins (leucoanthocyanins and catechins). These are found in celery, pumpkins, grapes, prunes, raisins, and persimmons. In high concentrations, fruit tannins may form a coagulum upon exposure to an acidic environment initiating the formation of a phytobezoar. Some medications can also cause pharmacobezoars. Common culprits include: cholestyramine, sucralfate, enteric-coated aspirin, antacids (e.g. aluminum hydroxide) (42) and bulk forming laxatives (Table 14). Additionally, extended-release nifedipine or verapamil can cause bezoars as the tablets are coated with poorly digestible cellulose.

Treatment of gastric phytobezoars includes enzymatic therapies such as papain or cellulose or lavage with or without endoscopic therapy to mechanically disrupt the bezoar. Patients with gastroparesis also benefit from long-term prokinetic therapy to treat and prevent bezoar formation. Metoclopramide (43), erythromycin or domperidone may be efficacious given their effect on gastrointestinal motility. Avoiding high fiber foods (especially citrus fruits and raw vegetables) is necessary to prevent recurrent phytobezoar formation (Table 14).

NUTRITION SUPPORT

Oral Nutrition Guidelines

Patients, dietitians and clinicians fundamentally prefer to provide nutrition by the oral route. If the patient's nutritional status and symptoms allow, then a trial of oral nutrition is indicated.

Ask the patient to keep a food diary to provide a more objective determination of actual oral intake (we generally do not recommend a specific caloric intake per day but rather look at overall trends over the ensuing 1–2 weeks). During this time, we look for improvements in caloric intake and gastrointestinal symptoms. At the start, it is prudent for the physician, patient and dietitian to define a target weight goal and the time period over which this weight must be reached. If the patient begins to gain weight, we stay the course and continue the oral nutrition trial. If they fail to gain weight or continue to lose weight, we proceed to enteral nutrition support. Table 15 summarizes an approach to oral nutrition support in the patient with gastroparesis. In addition, oral dietary guidelines can be found at: http://www.healthsystem.virginia.edu/internet/digestive-health/nutrition.cfm. Look under patient education materials and find three different diets for patients with gastroparesis: gastroparesis, gastroparesis and diabetes mellitus, and gastroparesis and kidney disease.

Enteral Nutrition (EN)

When significant malnutrition or poor control of gastroparesis symptoms prevails, a trial of enteral nutrition should be initiated. Enteral nutrition can keep patients out of the hospital, reducing their risk of nosocomial infection while concomitantly allowing for provision of nutrients, hydration and medication. EN is less expensive and is associated with fewer infectious complications than total parenteral nutrition (TPN) (44). It is also less labor intensive for the patient and the caregiver in the home setting. See Table 16 for criteria for enteral nutrition support.

Patients may be resistant to the idea of enteral nutrition, especially regarding nasal or endoscopically placed tubes. We stress to patients that:

- Enteral access provides reliable delivery of nutrition and hydration, as well as medications.
- Enteral access provides better delivery and thus more consistent absorption of prokinetic and antiemetic medications.
- More consistent delivery of nutrients enhances glucose control.
- Enteral nutrition is optimal in that it utilizes the gut.
- Even endoscopically placed tubes can be easily removed when symptoms resolve and they return to an oral diet.
- Our goal is to ultimately get the patient back on the road to oral feedings.
- If necessary, gastric venting can alleviate many of the debilitating symptoms of gastroparesis.

Table 15
Summary of Oral Nutrition Intervention in the Patient with Gastroparesis

1. Decrease the volume of meals.
   - Advise patients to eat smaller, more frequent meals.
2. Use more liquid calories.
   - If solid foods cause increased symptoms, begin with a liquid/pureed diet to promote gastric emptying.
   - If symptoms increase over the course of the day, try solid food meals in the morning, switching to more liquid meals later in the day.
   - Chew foods well.
   - Suggest that the patient sit up during and for 1–2 hours after meals.
3. Glucose control
   - If gastroparesis is a result of diabetes mellitus, maximize glucose control.
   - Monitor the need to change the timing of, or the overall requirements for insulin in order to have consistent delivery of nutrients with optimal total calories ingested.
   - Expect an increase in insulin requirements as improved symptom control will likely result in an increase in total calories ingested.
   - In general, dietary restrictions (e.g. diabetic or heart healthy diets) should be lifted during the trial.
4. Medications
   - Prokinetics and antiemetics should be given in regular scheduled doses (rather than "as needed" doses) and may be best tolerated in liquid form.
   - Avoid use of medications that affect gastric motility if possible (Table 5).
   - Review and delete any "unnecessary" meds (they can always be added back later).
5. Fat
   - Fat in liquids should be tolerated; implement #1–4 above before restricting.
6. Fiber
   - Fiber can be fermented in a "slow" gut by bacteria potentially causing gas, cramping and bloating, and can ultimately aggravate gastroparesis.
   - If bezoar formation is a concern, the patient should avoid the following high-fiber foods and medications:
     – Oranges, persimmons, coconuts, berries, green beans, figs, apples, sauerkraut, Brussels sprouts, potato peels, and legumes.
     – Fiber supplements such as: Metamucil, Perdiem, Benefiber, Fibercon, Citrucel, etc.
7. Treat bacterial overgrowth if suspected/symptomatic.
8. Monitor and replace as needed: Iron, vitamin B_{12}, vitamin D, and calcium.
   - If the patient is significantly malnourished, a daily standard vitamin/mineral elixir can be used for one month or longer or until stores are replete.
   - If patient has gastric intolerance to iron, try smaller doses; some is better than none. Liquid iron may be a better choice in some patients. Consider giving iron with vitamin C.

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Enteral Access

The type of enteral access required depends on patient/physician preference, estimated time that EN will be needed, and the ability of the patient to tolerate endoscopy or surgery for enteral tube placement.

In the ideal situation, patients should be given a short-term (48 hour) trial of nasojejunal feeding prior to endoscopically or surgically placed enteral access. This allows clinicians to determine if the patient will tolerate small bowel feedings. The major drawback of the nasojejunal tube is that it can migrate back into the stomach or become dislodged during a bout of emesis. Some clinicians favor the use of nasogastric/jejunal (NG-J) tubes, which allow gastric venting concomitantly with jejunal feedings. In our clinical experience, patient acceptance of these tubes is poor because of discomfort due to their large outer diameter. Multiple nasoenteric tube replacements with the burden of reinserting tubes along with radiation risks from repeated fluoroscopy should prompt placement of more permanent endoscopic or surgical enteral access. Endoscopically placed feeding tubes do not require general anesthesia and they are typically associated with lower costs when compared to surgically placed tubes.

If the patient tolerates jejunal feeding, endoscopic, laparoscopic or surgical jejunostomy may be the best approach. Oftentimes, patients with gastroparesis also benefit from concurrent gastric venting to prevent nausea and vomiting. There is no consensus regarding the need for gastric venting or the method by which it is accomplished. Percutaneous endoscopic gastrostomy-

(continued on page 58)

(continued from page 56)

Table 16
Criteria to Determine Candidacy for Enteral Nutrition

- Severe weight loss, e.g. unintentional weight loss >5%–10% of UBW over 3–6 months respectively.
- Repeated hospitalizations for refractory gastroparesis requiring intravenous hydration and/or medication delivery.
- Patient would benefit from gastric decompression.
- Patient has maintained usual body weight, but experiences significant clinical manifestations such as:
  - Diabetic ketoacidosis
  - Cyclic nausea and vomiting
  - Overall poor quality of life due to gastroparesis symptoms.
- Inability to meet weight goals set by physician, dietitian and patient.

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jejunostomy (also known as Jet-PEG or PEG-J) is beneficial in that it can provide gastric decompression with concurrent infusion of jejunal feedings utilizing only one abdominal insertion site. Some clinicians prefer a separate percutaneous endoscopic gastrostomy (PEG) for gastric venting plus a percutaneous endoscopic jejunostomy (PEJ) for enteral nutrition. Those advocating the latter option claim that the gastric venting is insufficient with the "tube within a tube" approach. They also describe excessive retrograde migration of the jejunal tube (J-tube) to the stomach with the PEG-J, thus increasing the risk for aspiration pneumonia. Other experts refute the benefit of gastric venting asserting that it may delay the recovery of gastric motility. Clinical trials are greatly needed in this area to determine if one technique is better than others with regard to patient preference, symptom improvement and complications.

At our institution, we prefer the PEG-J approach. Newer tubes have larger lumens that provide adequate gastric tube (G-tube) venting and consistent jejunal feeding without excessive clogging of the tube. J-tubes are also longer in length, allowing for more distal placement and significantly less retrograde migration. One percutaneous site theoretically results in fewer total site infections and less enteral/wound drainage than two separate abdominal sites. The University of Virginia's Digestive Health Center of Excellence utilizes a 24 French (Fr) Wilson Cook PEG tube with a 12 Fr J-tube passed fluoroscopically beyond the ligament of Treitz. Anecdotally, patients who have a PEG placed in the mid- to lower antrum facing the pylorus experience easier placement of the j-arm and less migration of the j-arm back into the stomach.

Direct percutaneous endoscopic jejunostomy (PEJ) (45) and surgical jejunostomy tubes do not allow for gastric venting and are used less often than PEG-J tubes. Care must be taken to avoid distal placement of a surgical jejunostomy, which can result in malabsorption due to a short bowel syndrome-like state. Surgical J-tubes also often have internal balloons, which if over-inflated, can obstruct the small bowel.

Fluoroscopically placed or computer tomography (CT) guided gastric tubes should be avoided. Their small diameter (12–16 Fr) makes them significantly more prone to clogging and they do not allow placement of a J-tube for jejunal feeding. If endoscopic tube placement cannot be performed, surgical jejunostomy with or without gastrostomy is preferred.

Initiating Feeding After Tube Placement

If the patient is significantly malnourished, special care should be taken to start nutrition support at the lower end of the calorie range at 20–25 kcal per kilogram of actual, euvolemic weight to avoid refeeding syndrome (46). Overfeeding the diabetic patient can also aggravate glucose control and hence nutritional rehabilitation.

The following are the UVAHS Nutrition Support Team protocols for initiating EN in patients with gastroparesis after feeding tube placement (Table 17):

- Keep patient NPO during initiation of tube feeding until tolerance is established: Avoids clouding the issue of gastric intolerance versus tube feeding intolerance.
- If feeding tube trial is desired prior to permanent access, soft, small bore feeding tubes, nasojejunal (not nasoduodenal), orojejunal: radiographic confirmation of tube placement followed by immediate use.
- PEJ or PEG with jejunal extension (PEG-J): Tube feeding may begin immediately via the jejunal port. There is no need to check for residual volumes with jejunal tubes, as there is no "reservoir" to collect tube feeding. In patients who continue to vomit, the gastric port can be placed to gravity drainage (e.g. leg bag or standard urinary drainage bag). If the gastric output is greater that 500 mL over a twenty-four hour

Table 17
Protocol for Initiation of Enteral Nutrition Following PEG-J Placement

1. NPO except 8 ounces of ice chips per day
2. Enteral nutrition to start via the J-port upon return to the unit per Nutrition Support Team recommendations
3. G-port to gravity for the first 24 hours after PEG-J placed
   - If <500 mL drainage, clamp G-port and monitor for nausea/vomiting
   - If >500 mL drainage, continue gravity drainage and assess need for jejunal re-infusion
4. Start liquid PPI via J-port q HS same day PEG-J was placed
5. Check gastric pH (via G-port) Day #2 (or after patient has received 3 doses of PPI
6. If patient is hyperglycemic on nocturnal tube feedings, add accucheks at 1800, 2200 and 0600.

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period, patients should re-infuse the gastric contents (see reinfusion section) via the jejunostomy.

- Surgically placed tubes: Initiation and advancement per the surgeon.

Formula Selection

Many practitioners believe that small bowel feeding requires special enteral formulas; however the majority of patients tolerate standard, polymeric formulas. In general, we use the least expensive formula that meets their individual needs.

A fiber containing formula may be useful due to its benefits on gut integrity. However, if the patient has concomitant small bowel dysmotility, indigestible fiber may aggravate symptoms if bacterial overgrowth is a chronic problem (47,48). For jejunally fed patients, the remaining absorptive area of the small bowel combined with intact pancreatic enzyme and bile salt secretions, further supports the use of standard formulas (consider the patient who has undergone a total gastrectomy that is discharged eating a regular diet). In patients with pancreatic exocrine insufficiency, powdered pancreatic enzymes can be added to enteral feedings or dosed periodically during infusion.

For patients with DM, there are no data to warrant the use of the more expensive diabetic formulas (49). Formulas to control glucose levels have not been shown by clinical trials to be efficacious or cost effective. One longitudinal study demonstrated no difference in $HgbA_1C$ levels when utilizing a standard, polymeric versus a lower carbohydrate formula in a nursing home population (50). The studies that have been done are small, of short duration, mostly in relatively healthy, non-hospitalized subjects, or in acute head injury patients without a documented history of DM. If glucose control is suboptimal and insulin therapy is undesirable, then it may be worthwhile to try one of the lower carbohydrate formulas.

Patients with end-stage renal disease are allowed a reasonable level of potassium, sodium, phosphorus, and volume. Often these patients can tolerate standard products and still stay within the guidelines of their renal diet prescription.

In the rare patient that can tolerate gastric tube feedings, volume is the most important factor. Changing to a calorically dense formula will provide more calories at a lower flow rate. Decreasing the total volume needed to meet nutrient needs may be all that is needed to allow continued gastric feeding.

Delivery Methods

Jejunal feeding commits the patient to cycled pump or gravity infusion typically over 8 to 14 hours, depending on symptoms. The small bowel is volume sensitive and does not tolerate intermittent bolus feeding to any appreciable extent. Enteral feedings do not need to be diluted, and the flow rate can be increased from 125 mL to 160 mL per hour or greater if tolerated.

Hydration

Patients that are enterally fed do not get enough hydration with tube feeding alone, thus it is imperative to provide supplemental water. As a rule of thumb, most tube feeding products are approximately 80% water. Current recommendations suggest that patients receive 1 mL of fluid (combined tube feeding and supplemental water) for every kcal of tube feeding. Patients typically require at least 1800–2000 mL of fluid each day. Thus if a patient is taking in only 1500 kcal of tube feeding per day (80% = 1200 mL), we provide an extra 500–700 mL of water to ensure they receive adequate

Table 18
Enteral Nutrition Summary and Problem-Solving Suggestions for the Patient with Gastroparesis

1. If needed, use enteral (rather than parenteral) nutrition; preferably jejunal feedings.
2. Use standard, polymeric formulas.
3. If there is a high suspicion of oral intake, we ask our patients to tell us what, rather than "if", they are eating and drinking on a daily basis.
4. Reiterate periods of strict NPO status (except for a few ice chips) during initiation of EN to avoid confusing oral intolerance with EN intolerance, until tube feeding tolerance is established.
5. If the patient experiences increased nausea/vomiting after initiating EN, try decreasing the rate of infusion for a few days (this could be from the ileal brake).
6. Maximize the use of liquid prokinetic agents and anti-emetics with delivery via the J-tube (scheduled versus "prn" dosing and liquid preparations may be beneficial). Gastric delivery of medications in a patient requiring gastric decompression will be ineffective.
7. Consider enteral treatment of bacterial overgrowth utilizing antibiotics given via the J-tube.
8. If bacterial overgrowth is a chronic problem, try a non-fiber-containing formula.
9. Check tube placement—specifically for backward migration of a J-tube into the stomach. Seek to reposition the tube.
10. If nausea and vomiting increase after the placement of a surgical J-tube and ileus is ruled out, a fluoroscopic study with oral contrast (swallowed, not via the J-tube) can determine if the internal balloon is obstructing the lumen. Decreasing the volume of saline in the balloon will alleviate the mechanical obstruction.
11. Use a more calorically dense formula to decrease total volume of EN required to meet calorie needs. Note: be sure to increase hydration with more calorically dense tube feeding formulas.
12. Maximize glucose control (glucose levels >200 mg/dl may aggravate gastroparesis).

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hydration over the course of the day. In patients who take in more than 1800 kcal of tube feeding, we adjust to provide an additional 1 mL of water for every 1 kcal over 1800. Patients with DM are at increased risk of dehydration, hence an additional 200–400 mL of water during the day may prove beneficial.

Because water is hypotonic, it is generally tolerated in bolus infusions (even in the jejunum) or it can be mixed with TF. Water can be delivered via syringe, gravity drip or pump. One convenient way to ensure adequate water delivery is to recommend that patients measure their daily water allotment every morning into a quart or similar container. Instruct them to use the water from this container over the course of the day; emphasize that **ALL** of it needs to be used before bedtime. They can infuse water through their enteral tube, use it as medication flushes, or drink it (if tolerated). Advise them to take in more water if they are thirsty, if their urine appears concentrated or if their urine output declines.

Re-infusion of Gastric Output

Gastric venting can be extremely beneficial to the patient with gastroparesis by decreasing nausea and preventing most episodes of vomiting. Patients with intractable nausea and vomiting can place their G-tube to gravity drainage using a leg bag or a standard urinary collection bag. If their G-tube output is greater than 500 mL over 24 hours, dehydration and metabolic disarray (hypochloremic, hypokalemic metabolic alkalosis) can occur if the fluid and electrolytes are not replaced. Replacement can be accomplished using a saline solution given via the jejunal port (e.g. 1/2 normal saline = 3/4 teaspoon salt in a liter of water replaced 1:1 for losses—this is in addition to their water and medication flushes!!). A more physiologic method involves re-infusion of the gastric secretions.

To further minimize the volume of gastric output, it is often beneficial to treat patients with acid reducing agents such as the proton pump inhibitors (PPI): omeprazole, lansoprazole, pantoprazole, etc., which also act to decrease the sheer volume of gastric secretions. Liquid PPI preparations are available; however, most PPIs in pills or capsule form may also be made into liquid forms that can be given via the enteral tube. Physicians should be cautioned, however that continued inhibition of gastric acid may increase the patient's risk to develop small bowel bacterial overgrowth.

Problem Solving

True intolerance of jejunal tube feeding is uncommon in patients with gastroparesis. More often, the side

(continued on page 63)

(continued from page 60)

Table 19
Preparation to Prevent Thrombophlebitis Associated with Peripheral Parenteral Nutrition (51)

Add directly to each liter of intravenous PPN solution:
- Hydrocortisone, 15 mg
- Heparin, 1500 units
- AND place transdermal nitroglycerin (NTG) patch, 0.1 mg/hour proximal to catheter

effects associated with enteral nutrition can be explained physiologically and are relatively simple to resolve. In some patients, the ileal brake may be the culprit (38,39,41). In our experience, this feedback inhibition generally resolves within a few weeks. Proximal migration of the J-tube can also result in a sudden increase in vomiting in the patient who has previously tolerated small bowel feedings. If the J-tube is not at, or beyond the ligament of Treitz, tube feeding can reflux back into the stomach. Surgical jejunostomy tubes can also cause nausea and vomiting due to partial mechanical obstruction caused by over-inflation of the internal balloon. A barium fluoroscopic study, orally or via the g-port, can confirm the obstruction; minor deflation of the balloon generally resolves the problem. Finally, it is imperative to ascertain if the nausea and vomiting are resulting from covert oral intake of foods. See Table 18 for summary and problem-solving guidelines for enteral intervention.

Total Parenteral Nutrition (TPN)

Total parenteral nutrition is rarely necessary for the patient with gastroparesis. TPN should be considered a last resort in patients who have a functional GI tract distal to the stomach. If TPN is required, close clinical and laboratory monitoring is imperative to prevent metabolic disarray and significant complications. Transition to enteral nutrition should be undertaken when clinically feasible and should be a priority.

Peripheral parenteral nutrition (PPN) is often considered to be easier than TPN, primarily because it is delivered via a peripheral vein. Its use, however, is limited by the sensitivity of peripheral veins to hypertonic solutions (51,52). The primary complication associated with PPN is thrombophlebitis (Table 19). This limits the caloric density of fluids that may be used and limits the length of time that PPN can be given. Thus, it is often impossible to meet full calorie and protein needs with PPN.

Table 20
Other Resources

GI Motility Web Sites
- Gastroparesis and Dysmotilities Association (GPDA): http://digestivedistress.com
- American Motility Society www.motilitysociety.org
- Association of Gastrointestinal Motility Disorders, Inc. www.agmd-gimotility.org
- Cyclic Vomiting Association www.cvsaonline.org
- International Foundation for Functional Gastrointestinal Disorders www.iffgd.org

Nutrition Support Web Sites
- American Society of Parenteral and Enteral Nutrition (ASPEN) www.nutritioncare.org
- Oley Foundation (Support Organization for patients on home nutrition support) www.oley.org

CONCLUSION

Gastroparesis can be very debilitating. Nausea and vomiting impact the patient's quality-of-life and can result in significant medical problems, most notably malnutrition. Accurate nutrition assessment is vital in the initial evaluation of a patient with gastroparesis as malnutrition contributes to significant morbidity and mortality in this patient population. Providing nutrition support, assuring excellent glucose control and treating nutrient deficiencies can be extremely challenging in the patient with gastroparesis. Nutrition intervention can decrease symptoms, replenish nutrient stores and improve an individual's overall quality of life. As with any chronic condition, support groups provide an invaluable resource for these patients. Table 20 provides a list of websites available for patients with gastroparesis or other gastroin-

testinal dysmotility disorders and for those patients who require nutrition support. ■

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(continued from page 64)

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