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Nutritional Approaches to Chronic Nausea and Vomiting

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In addition to a relative lack of definitive diagnostics and effective therapies, maintenance of adequate nutritional intake can represent a significant challenge for patients with chronic nausea and vomiting. The strength and specificity of available dietary recommendations vary by underlying diagnosis, each of which has a tendency to overlap with others. The relevance of particular clinical distinctions (e.g. gastric emptying delay) is not yet certain, in light of which it may be the case that dietary recommendations for one patient category can be selectively applied to others with similar benefits. This brief review will consider the existing evidence basis for nutritional approaches to a variety of non-structural causes of chronic nausea and/or vomiting, including gastroparesis, chronic nausea and vomiting syndrome, functional dyspepsia, cyclic vomiting syndrome, and rumination syndrome.

INTRODUCTION

For a variety of reasons, chronic nausea and vomiting can be difficult complaints to manage clinically. In cases of severe or refractory symptoms, quality of life can be markedly diminished, which often corresponds with significant healthcare resource utilization.[1] Objective testing modalities beyond endoscopy and scintigraphy are also limited, leading to a sometimes frustrating lack of etiologic specificity and often empiric patterns of therapeutic decision-making.

Regardless of governing diagnosis, the role of nutrition in the setting of chronic nausea and vomiting can be vital. Given a tendency within this patient population toward postprandial symptom exacerbation, there is keen interest in potentially mitigating dietary strategies. Chronic nausea and vomiting also may limit the adequacy of nutritional intake, which can necessitate consideration of enteral or parenteral feeding alternatives. While several options exist for pharmacologic and mechanical intervention among patients with chronic nausea and vomiting, this review will focus on nutrition-based approaches to their longitudinal support.

Etiology and Nomenclature

Recognizing the diverse and often overlapping diagnostic categories for chronic nausea and vomiting is a useful preface to considering nutritional management

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in the face of these symptoms (Table 1). Gastroparesis, defined as a clinical syndrome of gastric retention with objective evidence of delayed gastric emptying, is a commonly suspected etiology. When gastric emptying time is normal, however, and mucosal abnormalities, mechanical gastric outlet obstruction, and metabolic disturbances have been excluded, a diagnosis often relies on clinical patterns. With its latest iteration of diagnostic criteria (Rome IV), the Rome Foundation offered a revised classification for this latter set of patients, combining "chronic idiopathic nausea" and "functional vomiting" into the single category of "chronic nausea and vomiting syndrome (CNVS)." CNVS is formally defined as bothersome nausea or episodic vomiting, either of which must occur at least once weekly, at the exclusion of regurgitation, rumination, disordered eating behaviors, and underlying structural or systemic processes.[2]

Some investigators have questioned the importance of distinguishing gastroparesis from chronic nausea and vomiting with a normal gastric emptying time (sometimes called "chronic unexplained nausea and vomiting" or "vomiting of unexplained etiology") given that the clinical presentation and management of these two entities are often strikingly similar.[3] Functional dyspepsia (FD), a related Rome IV diagnosis that emphasizes post-prandial pain or fullness as the primary complaint, also includes the possibility of co-morbid chronic nausea (though usually without significant vomiting). Alternative nomenclature, including "gastroparesis-like syndrome" and "gastric neuromuscular dysfunction," has been proposed to reflect the possibility that, along a spectrum of transit time measurements, patients with chronic nausea (with or without associated bloating, abdominal pain, subjective fullness, and early satiety) may reflect common pathophysiologic mechanisms.[4]

Other potential explanations for chronic nausea and vomiting, such as cyclic vomiting syndrome (CVS), abdominal migraine, and rumination syndrome, have distinct historical features, but are likewise limited by a lack of clear objective metrics. CVS is defined according to Rome IV clinical criteria as episodes of vomiting stereotyped by acute onset and short duration with absence of vomiting between episodes. On the basis of phenomenological similarities and associations with migraine headaches, abdominal migraine is hypothesized as a close relative of CVS, the former typified more prominently by pain than by nausea. Rumination syndrome is a behaviorally mediated disorder defined by Rome IV criteria as the persistent regurgitation of recently ingested food, often within 30 minutes of meal completion; regurgitation, in turn, is qualitatively distinguished from vomiting by its effortless quality and usual dissociation from prodromal retching.[2] Chronic nausea and vomiting complaints might also reflect disordered eating, excessive cannibis use, or dysmotility processes distal to the stomach (e.g. intestinal pseudo-obstruction).

Gastroparesis and Chronic Nausea and Vomiting Syndrome

Gastroparesis is usually thought to arise from autonomic nerve injury related to diabetes, surgery, or antecedent infections, though the largest disease subcategory remains idiopathic.[5] Aside from the need for blood glucose optimization in patients with diabetes, nutritional recommendations in gastroparesis tend not to be etiologically specific. Indeed, some practitioners regard these recommendations as broadly relevant enough to apply, at least in part, to patients with symptoms of gastroparesis and a normal gastric emptying time, though specific data are lacking regarding optimal nutritional strategies within this population.[6]

Dietary Symptom Management

American College of Gastroenterology (ACG) guidelines advocate dietary interventions as the first-line strategy for gastroparesis management (Table 2). Traditional dietary recommendations to minimize symptoms and maximize tolerance of oral intake include: small, frequent meals (≥4/day) given the tendency toward gastric retention; an emphasis on liquid nutritional sources given relative preservation of liquid emptying function in gastroparesis; restriction of excess fat intake with solid meals given its deleterious effects on stomach emptying; and restriction of fiber intake given the risk of bezoar formation.[7] The narrowness of these restrictions is often organized in a stepwise manner in accordance with symptom severity, such that patients are ideally graduated from thin liquids to tolerable solids as their symptoms improve.[8]

Despite the longstanding nature of these recommendations, they are largely rooted in physiologic models and expert opinion rather than direct, trial-based observation.[9] A recent study of 12 patients with gastroparesis demonstrated increased post-prandial symptom severity with high-fat versus low-fat meals

Table 1. Potential Etiologies for Chronic Nausea and/or Vomiting in the Absence of Mucosal, Structural, Metabolic or Central Nervous System Abnormalities

Differential Diagnosis	Distinguishing Features	Treatment Options
Gastroparesis	Delayed solid gastric emptying in the absence of mechanical obstruction	• Dietary modification • Jejunal feeding • Herbal therapies (e.g. fresh ginger, ginger extract) • Medications (prokinetics, antiemetics, neuromodulators) • Acupuncture/acupressure • Mechanical therapies in select cases (e.g. pyloromyotomy, Gastric electrical stimulation)
Chronic nausea and vomiting syndrome (CNVS) *(alternative/related nomenclature: gastric neuromuscular dysfunction, gastroparesis-like syndrome, chronic idiopathic nausea, functional vomiting)*	Symptoms at least once per week, duration greater than 3 months, normal gastric emptying time	• Dietary modification • Herbal therapies, medications (antiemetics, neuromodulators) • Acupuncture/acupressure
Functional dyspepsia (FD)	Post-prandial fullness or pain; usually absent vomiting	• Dietary modification (avoidance of trigger foods) • Medications (antiemetics, neuromodulators) • Herbal therapies (e.g. STW5 [Iberogast])
Abdominal migraine	Acute onset episodes; pain typically more prominent than nausea	• Dietary modification (anti-migraine diet) • Medications (abortive and prophylactic anti-migraine agents, antiemetics, NSAIDs) • Cognitive behavioral therapy
Cyclic vomiting syndrome (CVS)	Acute onset, qualitatively similar episodes; absence of vomiting between episodes	• Dietary modification (avoidance of trigger foods; avoidance of fasting in select cases) • Medications (tricyclic antidepressants, anti-migraine agents, antiemetics, NSAIDs)
Cannabinoid hyperemesis syndrome	Prolonged cannibis use;relief of vomiting with cannibis cessation	• Cannibis cessation
Rumination syndrome	Effortless regurgitation (usually within 30 minutes of meal completion); absence of retching	• Behavioral therapy (e.g. diaphragmatic breathing) • Bridging enteral supplementation if needed
Intestinal dysmotility	Association with lower gastrointestinal symptoms (e.g. gas, bloating, distension, altered bowel habits)	• Medications (prokinetics, antiemetics, laxatives, neuromodulators)
Small bowel bacterial overgrowth	Association with lower gastrointestinal symptoms; history of small bowel surgery, anatomic abnormalities, or recent antibiotic use; breath test results may support the diagnosis	• Dietary modification (FODMAP avoidance) • Antibiotics
Eating disorders	Distorted body image; self-induced purging behaviors	• Psychiatric specialty care

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and with solid meals versus liquid meals.[10] A slightly larger study by the same group inventoried specific foods in a cohort of 45 patients with gastroparesis, identifying a trend toward fatty, spicy, acidic and roughage-based foods as reliable symptom triggers, while bland, sweet, salty, and starchy foods were comparatively well tolerated.[11]

Relatively newer strategies for oral nutrition in patients with gastroparesis include the small particle size diet, which emphasizes food that is easily mechanically processed to the consistency of a mashed potato. Within this framework, easily digestible foods include, for example, avocados, processed cheese, and other foods that can be pureed, mixed, or cooked to the consistency of mashed potatoes. Poorly digestible foods, by contrast, include seeds, grains, and fibrous, unpeeled fruits and vegetables. A randomized controlled trial of the small particle size diet among patients with diabetic gastroparesis demonstrated significant reductions in the severity of nausea/vomiting symptoms (as well as postprandial fullness and bloating) relative to a traditional diabetic diet. These symptom reductions were noted despite a significantly higher amount of fat in the intervention diet, suggesting that, with further study or in particular patient subsets, some nutritional recommendations may take priority over others.[12]

Other investigators have considered the utility of reducing fermentable carbohydrate loads (e.g. fermentable oligo-, di-, monosaccharides and polyols; FODMAPs) among patients with gastroparesis, particularly in light of the benefits such diets yield in the context of irritable bowel syndrome.[13] Retrospective, questionnaire-based analysis has suggested an association between high FODMAP intake in gastroparesis and increased abdominal pain and reduced quality of life, though no significant trend was noted with respect to nausea and vomiting.[14]

Table 3 offers more targeted recommendations based on the broad strategies outlined above (but should be supplemented with other published materials and consultation with a dietitian for the purposes of patient counseling). Dietary strategies may also include the consumption of herbal compounds with previously demonstrated antiemetic properties. While ginger preparations have not been rigorously studied in the context of gastroparesis or CNVS, they have demonstrated benefit in chemotherapy-induced nausea and hyperemesis gravidarum, with a putative mechanism related to accelerated gastric emptying and increased antral contractions.[15-16] Researchers have tended to study ginger as a powdered extract in dose ranges of 250-1,000 mg, though anecdotal benefits have also been reported from a variety of commonly available ginger products, including raw and crystallized ginger as well as ginger ale.[11] A separate compound of nine herbal extracts called STW5 (marketed from Germany as Iberogast) has demonstrated the ability to promote antral contractions and increase proximal gastric volume, suggesting a possibly beneficial role in gastroparesis, though it has not been formally studied in this population.[17]

Table 2. Established and Emerging Oral Nutritional Strategies in Gastroparesis

Established Dietary Recommendations
Multiple (4 or more) small-volume meals per day
Restrict fat in solid meals
Restrict dietary fiber
Relative emphasis on liquid nutritional sources
Blood glucose optimization in diabetic patients
Emerging Dietary Considerations
Small particle size diet
Poorly fermentable carbohydrate (i.e. low FODMAP) diet
Herbal therapies (e.g. ginger)

Nutritional Support

The propensity toward malnutrition among patients with gastroparesis is well established with regard to overall caloric intake as well as vitamins and minerals. Data suggest that particularly common micronutrient deficiencies include iron, folate, thiamine, calcium, magnesium, phosphorus, zinc, and Vitamins B_{12}, C, D, E, and K.[18-19] Importantly, overweight status precludes neither the diagnosis of gastroparesis nor the possibility of malnutrition, as recent research indicates that weight gain may be due to significantly reduced energy expenditure in this population relative to healthy controls.[20]

Among patients in whom oral feeding is deemed inadequate, enteral supplementation is preferred to parenteral supplementation due to the former's relative

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Table 3. Sample Recommendations Drawn from Broad Principles Regarding Oral Nutrition in Patients with Gastroparesis and Gastroparesis-Like Syndrome

Multiple small-volume meals per day[i]	• 4-6 meals/day or more • Decreasing meal size should correspond with increasing meal frequency to maintain adequate daily caloric intake
Restriction of dietary fat[i]	• Fat-containing liquids (e.g. nutritional supplements, milkshakes) are often well-tolerated • Favor lean meats or eggs • Avoid fried foods, fatty meats, creamy sauces, and peanut butter
Restriction of dietary fiber[i]	• White rice, refined white flour, refined cereals and cream of wheat are favored over whole wheat products • Avoid seeds, nuts, dried or fresh fruits, and legumes • Remove skins before cooking vegetables
Emphasis on liquid nutrition[i]	• Purees of solid foods (strained and thinned with milk or broth if necessary) can facilitate tolerance • Evaporated milk, protein powder, instant breakfast, and ice cream can be added to liquids for supplemental calories • Among patients restricted to thin liquids, nectars, juices, and sweetened beverages are preferable to water given caloric content
Small particle size diet[ii]	• Favored foods are those easily rendered to the consistency of mashed potatoes (e.g. asparagus tips, tomato paste, mixed pepper/onion, fruit puree, ripe bananas, watermelon, processed cheese, mashed eggs, baked fish) • Avoid poorly digestible foods (e.g. fibrous vegetables/meats, roughage, citrus fruit, nuts and seeds, whole grain breads, scrambled eggs, smoked fish) • An easily digestible consistency may be more important than fat avoidance for some patients
Avoidance of fermentable carbohydrates[iii]	• FODMAP exclusion diet is structured by elimination and challenge phases for IBS, but there is much less data for gastroparesis/chronic nausea • Foods high in poorly fermentable carbohydrates (FODMAPs) include those with lactose (e.g. milk, ice cream, soft cheeses), fructose (e.g. apples, pears, cherries, asparagus, sugar snap peas, honey, high-fructose corn syrup), fructans (e.g. wheat, rye, barley, peaches, chicory, onions, garlic), galactans (e.g. beans, legumes, chickpeas), and polyols (e.g. apples, apricot, avocados, blackberries, plums, prunes, cauliflower, mushrooms, sorbitol, xylitol) • Due to the complexity of this diet, consultation with a dietitian is recommended prior to initiation

Adapted from:

[i]Parrish CR. Nutritional considerations in the patient with gastroparesis. Gastroenterol Clin N Am. 2015 Mar;44(1):83-95;

[ii]Olausson EA, Storsrud S, Grundin H, et al. A small particle size diet reduces upper gastrointestinal symptoms in patients with diabetic gastroparesis: a randomized controlled trial. Am J Gastroenterol. 2014 Mar;109(3):375-85;

[iii]Gibson PR, Shepherd SJ. Food choice as a key management strategy for functional gastrointestinal symptoms. Am J Gastroenterol. 2012 May; 107(5):657-66.

safety, lower costs, and ease of use. Common thresholds for considering the initiation of enteral therapy include: unintentional, progressive weight loss (e.g. greater than 5-10% over 3-6 months, or consistently below agreed upon goals); frequent hospitalizations for dehydration or metabolic disarray; an inability to reliably take oral medications; and an otherwise unsustainably low quality of life or failure to thrive. The vast majority of patients will tolerate standard enteral formulations, though hospitalization is usually required for initiation of feedings, particularly among patients with labile blood glucose control or acutely severe symptoms.[21]

ACG guidelines for gastroparesis management recommend a trial of nasojejunal feedings prior to placement of a percutaneous feeding tube terminating in the jejunum in order to bypass the stomach. Keeping a jejunal tube in an appropriate position can be a challenge, however, particularly in patients with persistent vomiting. Anecdotal strategies to avoid displacement include minimizing the pre-pyloric distance traversed by the tube and locating its tip as distally as possible, though even with optimal initial positioning, endoscopic or fluoroscopic replacement may become necessary. Direct jejunostomy placement can also mitigate the likelihood of tube displacement but is relatively more technically challenging and precludes the possibility of gastric venting.[7] Little data is available regarding preferred enteral supplementation strategies in symptomatic patients with a normal gastric emptying time. In practice it may be more difficult to make the case for jejunal tube placement in patients without a formal diagnosis of gastroparesis, though cohort studies acknowledge the potential need for enteral feeding in this population as well.[22]

Other Causes of Chronic Nausea and Vomiting

Functional Dyspepsia

Similar food-based symptom triggers (fried, fatty, spicy foods, along with carbonated beverages) have been identified in the gastroparesis and FD populations.[23] While the mechanistic relationship between these two conditions is not yet definitively established, intriguing arguments have been made regarding the likely interplay among dietary fat, gastrointestinal signaling hormones, and aberrant gastroduodenal bolus transit giving rise to distressing visceral sensations in FD.[24-25] These hypotheses may have relevance for other chronic nausea syndromes as well. Recently published ACG guidelines do not recommend routine use of complementary and alternative modalities such as herbal preparations for FD, citing insufficient evidence.[26]

Cyclic Vomiting Syndrome (CVS) and Abdominal Migraine

While CVS manifestations can be marked by significant heterogeneity, diet plays a significant role in a subset of patients with this disorder. Potential triggers of stereotyped vomiting episodes can include prolonged fasting and catabolism associated with interceding illness, in which case prophylactic nutritional recommendations include supplemental carbohydrates between meals, before exercise, and at bedtime. Specific food-related triggers sometimes attributed to CVS include chocolate, cheese, caffeine, and monosodium glutamate (MSG), which, once identified in a given patient, should be avoided.[27] Such recommendations are sometimes broadened to exclude all foods generally implicated in migraine provocation (including citrus, pork, shellfish, game, gravies, yeast extract, and alcohol), attesting to the putative clinical proximity of CVS and abdominal migraine.[28] Interest in the dietary management of these conditions spans several decades; a low-oxalate diet (e.g. avoidance of carrots, onions, rhubarb, spinach, chocolate, and tea) was once advised in the 1970s for CVS and abdominal migraine, which at the time were deemed synonymous.[29]

Rumination Syndrome

While the therapeutic mainstay for rumination syndrome is behavioral, many experts advocate a multidisciplinary team of providers, particularly in severe cases. As with chronic nausea and vomiting of any etiology, the perspective of a dietitian can be quite valuable for accurately assessing caloric deficits and goals. The use of temporary enteral feeding modalities (e.g. nasogastric or nasoduodenal tubes) can also help meet patients' nutritional needs while targeted behavioral interventions are being pursued.[30]

CONCLUSION

Formalized dietary recommendations for patients with chronic nausea and vomiting hinge on diagnostic categories whose boundaries have been subject to ongoing revision. The clinical relevance of gastric emptying delay, in particular, has been called into question, suggesting that oral feeding recommendations for gastroparesis may be at least partially applicable to symptomatic patients with normal scintigraphic

results. Enteral supplementation is preferred when oral nutrition is deemed inadequate, with post-pyloric feedings perhaps easier to rationalize in the setting of documented gastroparesis. Particular foods have been identified as typical symptom triggers in gastroparesis, FD, CVS, and abdominal migraine, and avoidance of these foods can be considered on an empiric basis. Areas ripe for further study include the precedence of dietary content versus consistency in the management of gastroparesis; optimal thresholds and locations for enteral feeding in various patient subsets; and any relevant distinctions between gastroparesis, FD, and CNVS that might impact individual strategies of dietary optimization. ■

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