Nutritional approaches for gastroparesis

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Patients with gastroparesis often have signs and symptoms including nausea, vomiting, epigastric discomfort, and early satiety, thus leading to inadequate food intake and a high risk of malnutrition. There is a considerable scarcity of data about nutritional strategies for gastroparesis, and current practices rely on extrapolated evidence. Some approaches include the modification of food composition, food consistency, and food volume in the context of delayed gastric emptying. If the patient is unable to consume adequate calories through a solid food diet, stepwise nutritional interventions could include the use of liquid meals, oral nutrition supplements, enteral nutrition, and parenteral nutrition. This Review discusses the role, rationale, and current evidence of diverse nutritional interventions in the management of gastroparesis.

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Introduction

Gastroparesis is a chronic motility disorder of delayed gastric emptying in the absence of mechanical obstruction. Common signs and symptoms include nausea, vomiting, early satiety, postprandial fullness, bloating, and abdominal discomfort, which can markedly affect quality of life and can lead to increased use of health care.[1–4] Patients with gastroparesis might also report a sense of loss and social isolation when their ability to eat with others changes.[5] Most cases of gastroparesis are idiopathic (50%), followed by diabetic gastroparesis (38%).[6] Other causes include medication-induced, post-surgical, viral, neurological, and autoimmune mechanisms.[1,7,8]

The pathophysiology of gastroparesis is incompletely understood and varies depending on the underlying cause. Healthy gastric motility is governed by a balance of excitatory and inhibitory signals, such as excitatory cholinergic innervation from the vagus nerve causing antral contraction and inhibitory nitrergic nerves inducing gastric accommodation and pyloric relaxation.[9] These excitatory and inhibitory neural signals are transmitted to the gastric smooth muscle cells through the interstitial cells of Cajal and possibly other fibroblast-like cells with pacemaker function, leading to coordinated contractions from the proximal stomach to the pylorus. Autonomic dysfunction, including disruptions to the vagal nerve, abnormalities in the enteric nervous system, and myopathic disorders, can all affect gastric motility. The pathophysiological changes that have been observed in gastroparesis include impaired fundal accommodation,[10] altered gastric myoelectrical activity,[11–14] diminished antral contractions,[15,16] and decreased pyloric compliance.[17–19] The exact mechanisms behind these motor disturbances are not clear. Studies have shown a decrease in the number of pacemaker cells, such as the interstitial cells of Cajal, in the stomachs of individuals with gastroparesis compared with in healthy individuals but this decrease does not necessarily correlate with symptom severity.[20–26]

The diagnosis of gastroparesis involves ruling out mechanical obstruction and confirming that gastric emptying is delayed. Scintigraphy is the gold standard for diagnosing delayed gastric emptying.[7,27] Treatments for gastroparesis include: dietary modification; optimisation of glycaemic control in patients with diabetes; pharmacological therapy, such as prokinetics; endoscopic therapy, such as gastric peroral endoscopic myotomy; gastric electrical stimulation; and surgery, including pyloroplasty or gastrectomy for people with refractory disease.[7,27,28] During surgery, such as for placement of a gastric electrical stimulator or jejunal tube, full thickness gastric or intestinal biopsies can be taken. Evaluation for the presence of interstitial cells of Cajal, ganglia, and fibrosis might better clarify gastrointestinal pathophysiology and predict treatment response.[23] Novel therapies, such as the use of intravenous immunoglobulins in autoimmune gastroparesis, are still under investigation and not yet approved for gastroparesis.[29–31]

Diet and other nutrition therapies are important considerations to address both symptoms and nutritional impairments that can result from gastroparesis. Nonetheless, the efficacy of nutritional interventions strongly relies on concurrent treatment measures to improve gastric function because the pathophysiology and symptoms of gastroparesis adversely influence tolerance to food intake. This Review discusses the role, rationale, and current evidence of diverse nutritional interventions in the management of gastroparesis.

Malnutrition

The signs and symptoms associated with gastroparesis (ie, nausea, vomiting, early satiety, postprandial fullness, and abdominal discomfort) often lead to food aversion and restriction in oral intake of foods and fluids.[32,33] Malnutrition and dehydration are thus common complications in patients with moderate to severe gastroparesis. Dietary assessments of patients with gastroparesis often show diets deficient in calories, fat, protein, and several vitamins and minerals.[34,35] In a multicentre dietary survey of patients with diabetic or idiopathic gastroparesis, Parkman and colleagues[34] noted that a large proportion of patients (194 [64%] of 305) consumed diets with less than 60% of their daily energy requirements. Patients consumed 1·4 meals per day on average and more than half of patients had diets deficient in vitamin D (186 [61%] of 305), vitamin E (244 [80%]), vitamin K (170 [56%]), folate (208 [68%]), calcium (213 [70%]), iron (210 [69%]), magnesium (220 [72%]), and potassium (263 [86%]).

As well as reduced consumption of foods that are rich in iron, decreased gastric acidity from frequent use of proton-pump inhibitors means that patients with gastroparesis are at even greater risk for iron deficiency than are healthy individuals.[36,37] Gastric pH affects the absorption of dietary iron by decreasing the availability of absorbable ferrous iron. Patients with gastroparesis receiving jejunal enteral nutrition are particularly at risk of iron deficiency because iron is predominantly absorbed in the proximal small bowel.[37] Iron absorption is further inhibited by bacterial overgrowth from decreased gastrointestinal motility and decreased gastro-intestinal acidity, particularly in patients who have had a vagotomy.[14] Deficiency of vitamin B12 arises from low dietary intake, anatomical changes with loss of intrinsic factor after surgery, decreased gastric acidity, and bacterial overgrowth.[36–38] In healthy people, vitamin B12 is bound to intrinsic factor produced in the stomach and absorbed in the terminal ileum. High gastric pH in patients with gastroparesis inhibits cleavage of protein-bound vitamin B12 and subsequent binding to intrinsic factor. Decreased motility and decreased gastrointestinal acidity also promote bacterial overgrowth, which impair vitamin B12 absorption in the terminal ileum.[39] Patients who have undergone a gastrectomy are at further risk for vitamin B12 deficiency because of low intrinsic factor production.[38] Deficiency of vitamin D and calcium in gastroparesis can lead to metabolic bone disease. This deficiency mainly arises from decreased consumption of vitamin D, calcium, and foods rich in lactose.[36,37,40] Decreased absorption from postsurgical changes in anatomy can further exacerbate deficiency of vitamin D. Given the high risk of osteopenia or osteoporosis development, routine vitamin D screening is recommended as part of the management of gastroparesis, as well as a dual-energy x-ray absorptiometry scan at baseline and as needed thereafter.[8,36,37]

Nutrition screening

Given the high risk of malnutrition, nutrition screening is an important early step in the care of patients with gastroparesis. Although there are no specific screening tools validated for gastroparesis, several general screening tools can be adapted to this patient population. The Subjective Global Assessment is a widely used bedside screening tool that has been validated in several diseases, which gathers data about oral diet intake, unintentional weight loss, presence of gastrointestinal symptoms, functional capacity (eg, whether the individual can ambulate, is bedridden, etc), and physical findings.[41] A simpler derivative of the Subjective Global Assessment that consists of three questions is the Malnutrition Screening Tool, which relies on weight loss reported by patients, decreased oral intake due to poor appetite, and appearance of frailty or suboptimal weight.[42] The clinician should be vigilant about these three factors in patients with gastroparesis. Patients should be screened at the initial visit and routinely rescreened for risk of mal-nutrition, if malnutrition was not previously identified.

The European Society of Clinical Nutrition and Metabolism has defined two major criteria for diagnosing malnutrition: body-mass index less than 18·5 kg/m²; or unintentional weight loss (>10% of total weight for an indefinite period or >5% over the preceding 3 months) with a body-mass index of less than 20–22 kg/m² (depending on age) or fat-free-mass index less than 15–17 kg/m² (depending on sex).[43] A consensus statement by the American Society for Parenteral and Enteral Nutrition and the Academy of Nutrition and Dietetics did not formally define malnutrition risk, but used compromised intake or loss of body mass as sentinels of potential risk.[44] Specific criteria for malnutrition were nonetheless provided on the basis of the context of illness

(ie, acute illness or injury, chronic illness, or social or environmental circumstances). A diagnosis of malnutrition would be supported by recorded changes in nutritional intake, in weight, in body composition, in physical findings, and in functional status.

Nutrition assessment

Once screened to be at risk for malnutrition, patients with gastroparesis should be referred to a registered dietitian to undergo formal nutrition assessment (panel 1). The components of a nutrition assessment include a review of the patient's symptoms, food intake, nutrition history, medical history, surgical history, social habits, reported changes in body and weight composition, physical examination focused on nutrition, diagnostic procedures, and laboratory tests.[45] The nutrition assessment is key to understanding drivers and trends in the nutritional status of the patient, and to guiding the plan for nutritional interventions. For instance, the patient's understanding of their condition, personal challenges, and readiness to change eating behaviours are important considerations when designing a nutritional treatment plan. The plan of care could include education about nutrition concepts related to gastroparesis and on the rationale for the plan, counselling to provide the patient with strategies for implementation, and adjusting the diet or nutrition regimens to overcome obstacles as anticipated or as they arise.

Oral diets

Research about the use of solid food diets in gastroparesis is scarce and most guideline recommendations and clinical practices have relied on extrapolated evidence. For instance, the premise that fats decelerate gastric emptying has led to the common recommendation that patients with gastroparesis should consume a diet that is low in fat. Similarly, the knowledge that volume and particle size influence gastric emptying time fuels the recommendation to eat smaller but more frequent meals. These nutritional approaches for gastroparesis consider how food composition, food consistency, or food volume might influence gastric emptying (panel 2) Optimisation of the oral diet needs to be accompanied by concurrent optimisation of medical, endoscopic, or surgical treatments, or a combination, for gastroparesis to improve outcomes.

Food composition

Dietary recommendations for gastroparesis have generally recommended reduction of fibres, fats, and refined carbohydrates on the basis of their theoretical effects on symptoms in the setting of altered gastric emptying (table). In a survey of 45 patients with idiopathic gastroparesis on their tolerance to specific foods, the following were observed to most aggravate their symptoms: fried chicken, sausage, bacon, roast beef, oranges, cabbage, peppers, onions, lettuce, broccoli, orange juice, and tomato juice.[32]

Panel 2: Recommendations and rationale for oral food intake for gastroparesis

Food composition
- Patients should limit foods that are high in fibre because fibre is less easily digested and might empty the stomach more slowly than other macronutrients, thus aggravating symptoms. Some fibres are fermentable prebiotics that can increase gas production, particularly in patients with intestinal dysmotility or small intestine bacterial overgrowth, or both.
- Patients with gastroparesis are at risk of phytobezoar formation so it is recommended that foods that contribute to formation are avoided.

Food consistency
- Food should be mashed, chopped, ground, or blended to reduce particle size because small particles of food might empty the stomach more readily than large particles.
- If the patient is unable to tolerate meals of solid foods, liquid meals or oral nutrition supplements of high calorific value can be consumed. This is recommended because liquids empty the stomach more readily than solids.
- Patients should chew food well because small particles empty the stomach more readily than large particles.

Food volume
- Frequent meals of small volume should be consumed because this could reduce risk of intragastric pressure.

Body position
- After meals, patients should sit, stand, or walk because food empties the stomach more rapidly when sitting or standing than when lying supine after meals.

The predominant qualities attributed to these foods by the investigators were described as fatty, acidic, spicy, and roughage-based. Foods that were better tolerated included salmon, white fish, gluten-free foods, white rice, applesauce, potatoes, popsicles, ginger ale, and tea. These foods were primarily described as bland, sweet, salty, and starchy. This small study provides some insight into the dietary tolerances of patients with gastroparesis, although in the absence of larger studies, empirical trial and error is a major component of dietary recommendations.

Dietary fibre

Fibre reduction is a common recommendation for patients with gastroparesis. Dietary fibres are indigestible carbohydrates found only in plant-based foods. Fibre has traditionally been classified by its solubility (soluble or insoluble), although classifications in 2001 considered fermentability and viscosity.[46] Soluble fibres with moderate fermentability and viscosity include guar gum, inulin, fructo-oligosaccharides, galacto-oligosaccharides, and pectin. Insoluble fibres are typically non-fermentable and include cellulose, hemicellulose, and lignin. The consumption of a diet that is high in fibre has the potential to slow gastric emptying, leading to recommendations for patients with gastroparesis to consume a diet low in fibre.[47,48] From our experience, tolerance to fibre can vary between patients and the degree of fibre reduction should be personalised for optimal tolerance. Besides the aggravation of upper

	Examples of foods to include	Examples of foods to limit
Meats, poultry, and fish	Lean (eg, beef, pork, chicken or turkey without skin, and grilled or steamed fish or shellfish)	Fried or fatty (eg, beef, pork, chicken, turkey, fish, and shellfish)
Dairy, dairy alternatives, and eggs	Whole-fat dairy in liquid or semiliquid form (eg, milk, milkshakes, and yogurt), low-fat dairy (eg, cheeses, cottage cheese, and cream cheese), non-dairy milks (eg, almond, coconut, and rice), eggs (eg, scrambled eggs, hard-boiled eggs, and egg whites)	Fried cheese, creamed eggs, and fried eggs
Grains	Breads of low fibre (eg, sourdough and white), cereals (eg, cornflakes, cream of wheat, cream of rice, and oatmeal), graham crackers, noodles, pancakes, pastas, pretzels, white rice, flour tortilla, and waffles	Whole grains or grains covered with nuts and seeds (eg, whole wheat), barley, bran, bulgur, high fibre cereals, granola, quinoa, brown rice, and corn tortilla
Fruits and vegetables	Canned fruits or vegetables, banana, peeled fruits (eg, apples and pears), cantaloupe, honeydew, watermelon, fruit or vegetable sauces with no pulp or skins, peeled and cooked vegetables (eg, beetroot, carrots, potatoes, and squash)	Fruit or vegetable skins, dried fruits, coconut, berries, figs, grapes, grapefruit, persimmons, prunes, oranges, alfalfa or bean sprouts, artichokes, asparagus, broccoli, brussel sprouts, cabbage, cauliflower, celery, corn, cucumbers, eggplant, lettuce, mushrooms, and okra
Vegetables, legumes, and nuts	Tofu and creamy nut butters (eg, peanut or almond butter)	Beans, chickpeas, lentils, and crunchy nut butters
Beverages	Fruit and vegetable juices or smoothies with no pulp or skins, coffee, sport drinks, tea, and plain water	Prune juice, sparkling water, and sodas

Tolerance can vary among patients. The diet and portions of foods should be individualised with nutrition education and counselling by a registered dietitian. This is a sample of recommended foods and not an exhaustive list.

Table: List of recommended foods for gastroparesis

gastrointestinal symptoms from food retention in the stomach, an additional concern with high fibre intake is the formation of phytobezoars.[49] Phytobezoars are composed of the skins, seeds, and leaves of plant-based foods that accumulate in the stomach over time, so patients with gastroparesis might need to avoid particular types of foods with fibre. Foods prone to phytobezoar formation include fruits, such as apples, berries, coconut, figs, oranges, persimmons; and vegetables, such as brussels sprouts, celeries, green beans, and potato peels.

Moreover, some fermentable fibres are considered to be prebiotics that could lead to gaseous abdominal distention and discomfort in the presence of bacterial overgrowth in the small intestine, a condition common in patients with gastroparesis. In a small study published in abstract form by Hsu and colleagues,[50] eight patients with gastroparesis were given two consecutive diets for 5 days per diet: low fibre and normal fat, and normal fibre and low fat. Consuming a low-fibre and normal-fat diet reduced nausea, vomiting, abdominal fullness, and early satiety, whereas consuming a normal-fibre and low-fat diet did not improve symptoms.

Fat

In addition to reducing fibre intake, patients with gastroparesis are often counselled to reduce their fat intake because fats might delay gastric emptying.[51,52] However, there is little evidence to support or refute this recommendation for gastroparesis. The aforementioned abstract by Hsu and colleagues noted that a normal-fat and low-fibre diet improved symptoms, but the low-fat and normal-fibre diet was ineffective.[50] These observations suggest that fat might not be the crucial macronutrient to avoid, although the findings are confounded

by differing fibre content. We do not know whether fat and fibre reduction would provide greater benefit than fibre reduction alone. Paradoxically, an early study of eight healthy male volunteers noted that consuming a diet high in fat for 14 days led to accelerated gastric emptying of a test meal that was high in fat, suggesting that adaptation might occur after continual fat consumption.[53] In a 2015 study of 12 patients with idiopathic or diabetic gastroparesis, participants received four different types of meals (solid meal high in fat, liquid meal high in fat, solid meal low in fat, liquid meal low in fat) in a randomised order on four separate days.[54] The solid diet that was high in fat provoked significantly higher total gastrointestinal symptom scores than the other diets did, although the total scores were otherwise similar in the other diets. Although the total symptom scores were elevated during the 4 h postprandial observation, scores returned to baseline by 3 h for all diets except the solid diet that was high in fat. This observation indicates that solid meals that are high in fat could aggravate symptoms more than other forms of meals, although this study could suggest that a high fat content and solid food consistency do not necessarily provoke symptoms by themselves. Overall, the current amount of evidence is low and unable to clarify the benefit of fat reduction in isolation for gastroparesis.

Carbohydrates

Simple carbohydrates have also been implicated in gastroparesis. In patients with diabetic gastroparesis, glycaemic control is a crucial consideration in meal planning. Intake of carbohydrates of appropriate portions should be consistent in all meals to optimise glycaemic control. However, whether appropriate intake

of carbohydrates in patients without diabetes has any practical clinical effect is unclear. Although liquids containing dextrose have been shown to empty the stomach more slowly than saline does even in healthy individuals,[55] this effect could be more related to the osmolarity or energy density of the meal than the presence of dextrose per se.[56–58] Consistent with the hypothesis that energy density (rather than intrinsic properties of the macronutrient type) influences gastric emptying, protein consumption also appears to have a dose-dependent effect on gastric retention.[59] Therefore, there is insufficient evidence for carbohydrate restriction in patients with gastroparesis but without diabetes.

Food consistency

Mechanical and chemical digestion in the stomach breaks down food particles before emptying into the duodenum. The stomach empties small digestible particles faster than large particles.[60] In the only randomised controlled trial thus far of a dietary intervention for diabetic gastroparesis, 56 participants either followed an intervention diet with small particle size ("food should be easy to mash with a fork into small particle size") or a conventional diet recommended to patients with diabetes.[61] Over 20 weeks, participants who were given the intervention diet had a reduction in nausea, vomiting, fullness, bloating, lower abdominal pain, heartburn, and regurgitation, but not upper abdominal pain. Participants who were given the control diet had no change in symptoms. These findings occurred despite the participants who were given the intervention diet having a higher mean fat intake (67 g per day vs 57 g per day, p=0·034) and no difference in fibre intake compared with participants on the control diet. Patients with gastroparesis could therefore benefit from strategies to decrease particle size, such as chewing food well, selecting foods that can be mechanically reduced with a fork, and grinding food with a blender. If the patient continues to show intolerance to solid foods and is unable to reach nutritional goals, liquid meals can be introduced as an oral nutrition supplement or as puréed foods (eg, blended smoothies and soups) to augment caloric intake. Liquids are generally better tolerated in gastroparesis, as they begin to exit the stomach soon after consumption, whereas solids initially accumulate in the fundus, presumably for trituration and reduction of particle size to occur.[55] Oral nutrition supplements are premade liquids or powders formulated to provide a supplemental and dense source of calories or protein, or both. Oral nutrition supplements have traditionally been used to improve caloric intake and stabilise weight in patients who are malnourished, although formulations specific for diseases are available.[62–64] For gastroparesis, oral nutrition supplement mostly serves as a supplemental source of calories and protein; there is otherwise an absence of evidence directly evaluating its role specific to gastroparesis. From our experience, oral nutrition supplement with small sips throughout the day has been generally well tolerated in patients with gastroparesis.

Food volume

In healthy individuals, large meal volumes accelerate gastric emptying because of increased intragastric pressure.[58] However, patients with gastroparesis might potentially benefit from small and frequent meals. Meal consumption can traditionally involve three meals per day, whereas small frequent meals can involve six to ten meals per day.[65] The rationale, composition, timing, and frequency of meals and the challenges of implementation, should be discussed with the patient. Selection of foods that are dense in calories allows for consumption of small meal volumes while preserving daily calorie intake. A caveat is that energy dense meals might counterproductively decelerate gastric emptying, thus highlighting the need to individualise the balance between size, frequency, and content of meals.[57,59]

Body position

Body position after meals has also been shown to influence gastric emptying. In a small study of eight healthy volunteers, the combination of sitting and standing (walking as desired) led to significantly faster gastric emptying times than did standing, sitting, or being in the supine position alone.[66] Standing or sitting after meals also led to significantly faster gastric emptying times than being in the supine position did.

Enteral nutrition

Enteral nutrition is indicated when a patient with gastroparesis shows ongoing intolerance to the oral diet despite medical and nutritional interventions (panel 3).

Types of feeding tube

In contrast to the nasogastric tube, a postpyloric feeding tube is recommended for patients with refractory gastroparesis to allow delivery of the formula directly into the small bowel, bypassing the stomach. Options for feeding tubes in the short term include the nasoduodenal or nasojejunal tubes (figure 1). These tubes can be placed at the bedside and are reserved for short-term use because of the increased risk of sinus infections from long-term use. Although prone to clogging and migration back into the stomach, particularly in patients with persistent vomiting, these tubes should be considered as a first-line option to assess tolerance to enteral nutrition before more invasive placement of a long-term feeding tube. Feeding tubes for intermediate or long-term use include gastrojejunostomy tubes or jejunostomy tubes. Gastrojejunostomy tubes enter the abdomen via the stomach, course through the pylorus into the small bowel, and end in the jejunum. Gastrojejunostomy and jejunostomy tubes can be placed endoscopically as percutaneous endoscopic gastrostomy-jejunostomy tubes or direct percutaneous endoscopic jejunostomy tubes. These tubes can alternatively be placed

Figure 1: Routes of access for types of jejunal tube

via radiological guidance by interventional radiology or via surgery. During surgical placement of jejunostomy tubes, full thickness biopsies can be concurrently obtained to aid diagnosis and guide treatment considerations.[23] Options for placement vary according to institutional expertise.

Long-term use of enteric tubes is generally considered to be safe, although uncommon complications can arise. Beyond the risks associated with the procedural placement of the enteric tubes, longitudinal complications related to the tubes can include clogging, dislodgment, malfunction, tip migration, buried bumper syndrome, stoma leakage, and site infections.[67–69] Similar to the nasoenteric tubes, the small-bore jejunal tips of gastrostomy-jejunostomy tubes are at risk of clogging and migrating back into the stomach. Jejunostomy tubes, however, are inserted directly through the abdominal wall into the jejunum. Although jejunostomy tubes are similarly at risk of clogging because of their small diameter, they do not migrate into the stomach. Unlike with other tubes, jejunal volvulus can occur in less than or equal to 1% of patients with jejunostomy tubes, when their fixed position serves as a fulcrum for twisting of the jejunum.[70]

Stoma-related complications that can affect all abdomino-enteric tubes include buried bumper syndrome, leakage, and infections at the stoma site. Buried bumper syndrome stems from excess pressure between the external tube bolster and internal bumper that leads to pressure necrosis, ulcer, oedema, and the mechanical effect of the bumper being buried into the gastrointestinal tissue.[71] Prevention of 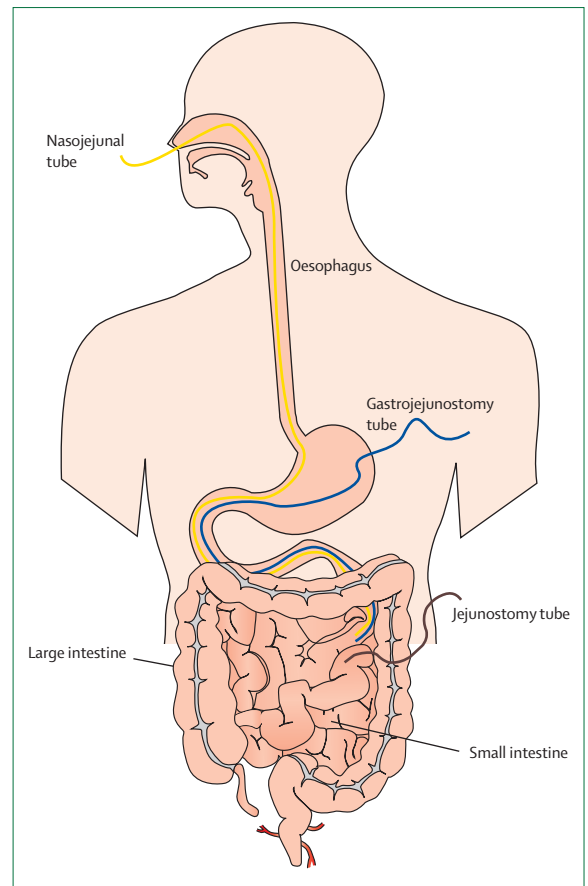stoma site complications requires periodic evaluation that the tube is not too tight (beginning several days after initial tube placement) and concurrent stoma site assessments.

A possible benefit of gastrostomy tubes with a jejunal extension is the ability to vent the stomach for alleviation of nausea and vomiting, while allowing enteral formula to be administered through the jejunal extension. Potential risks of frequent gastric venting include dehydration, hypochloraemic metabolic acidosis, and imbalances in electrolytes.

Type of enteral formula

Enteral formulas include oral nutrition supplements, which come in liquid or powder form, and specialised formulas that are intended for administration through a feeding tube. Enteral formulas are classified as polymeric (ie, intact macronutrients), semi-elemental (ie, peptide based), or elemental (ie, amino-acid based).[72] Formulas containing fibre are generally not recommended for patients with gastroparesis because of potential aggravation of symptoms. A polymeric formula that does not contain fibre can be initiated in patients with gastroparesis. Diabetic formulas are often used to assist with the management of glycaemic levels in patients with

hyperglycaemia or diabetic gastroparesis; however, the efficacy of diabetic formulas in this role or in other conditions has not been shown. Moreover, diabetic formulas contain fructo-oligosaccharides as a source of fibre and might not be tolerated well by some patients. Blended formulas consisting of foods and liquids that have been mechanically puréed, have gained popularity in patients who need enteral nutrition because of the perception that they include ingredients that are considered to be more natural than those used in synthetic formulas.[73] Additional benefits include improved tolerance (low risk of developing symptoms, such as nausea and vomiting) versus synthetic formula, the ability to eat the same foods as family members, and lower costs. Blended formulas can be prepared at home, although commercial products containing animal-based and plant-based foods exist on the market. Despite the assortment of options for enteral nutrition formula that is available, there are no clear first-line choices other than considering a polymeric formula that is low in fibre or a diabetic formula for patients with diabetic gastroparesis.

Initiation of enteral nutrition

Because the provision of enteral nutrition can be fairly burdensome, such as delivery via enteric feeding tube, ample discussion with the patient and caregivers should be held before initiation of enteral nutrition. Ongoing education showing successful use of the enteric feeding tube is also important. An enteral pump would be required to administer the formula through the jejunostomy tube. As a test to distinguish intolerance to the enteral nutrition formula from intolerance to the oral diet, the clinician could temporarily stop the oral intake of patients at the initiation of enteral nutrition. In hospital, patients are often initiated on a continuous regimen over 24 h at a low rate of delivery, followed by a gradual increase toward a goal rate that balances increased independence from the enteral regimen, tolerance, and the ability to still meet daily nutrient needs. From the our experience, patients can be discharged while still on the 24 h regimen and can progress towards a cyclic regimen at home. Patients who require insulin therapy will need to be managed much more closely by a multidisciplinary team. This strategy of progression at home would nonetheless require close monitoring and adjustments as needed. In particular, the oral diet can be continued as tolerated, although it would be important to differentiate between gastrointestinal symptoms arising from oral intake and from enteral nutrition. In a cohort of 36 patients who required enteral nutrition and were placed on so-called gastric rest (where no food is consumed while exclusively relying on feeding by tube) for 3 months, 17 (47%) patients were able to return to oral intake with a gradual stepwise increase in consumed calories and were able to wean off enteral nutrition.[74] All other patients were unable to tolerate progressively increasing oral intake and thus continued to receive nutrition support. The efficacy of gastric rest is

unproven and requires further investigation before consideration for broader application.

Parenteral nutrition

Parenteral nutrition bypasses the gastrointestinal tract and delivers nutrients and electrolytes directly into the veins. Parenteral nutrition is rarely needed for patients with gastroparesis and should be reserved for patients who did not respond to a trial of enteral nutrition or who do not have enteral access, particularly given the high risk of complications (eg, infections associated with catheters, thrombosis, metabolic bone disease, and liver disease).[75,76] Compared with parenteral nutrition, potential benefits of enteral nutrition include delivery of nutrients in a physiological manner, modulation of gut health and immunity, fewer complications, and lower costs.[77–79] Before the initiation of parenteral nutrition, discussions with the patient and caregivers are needed about the indications, risks, benefits, and implications of parenteral nutrition. Training on sterile technique and appropriate administration of parenteral nutrition is crucial because there is a high risk of infections associated with catheters. The risk of re-feeding syndrome should be considered in patients with malnutrition who are going to have parenteral nutrition. If patients are identified to be at risk for re-feeding syndrome, the parenteral regimen should be initially formulated at a low volume to reduce the risk of fluid overload, with modest provision of carbohydrates, and with sufficient provision of potassium, phosphorous, and magnesium; the serum concentrations of these elements might decline with re-feeding. Parenteral nutrition is often initiated as a continuous regimen and transitioned to a cyclical regimen, when appropriate, for the home setting. Monitoring involves appropriate catheter care and routine review of laboratory tests. Planning for a transition to enteral nutrition or oral diet, or both, should continue during optimisation of medical or surgical management (figure 2). Discontinuation of parenteral nutrition generally involves a gradual weaning process that is tailored to the individual, while tolerance to enteral diet or oral diet, or both, is established.
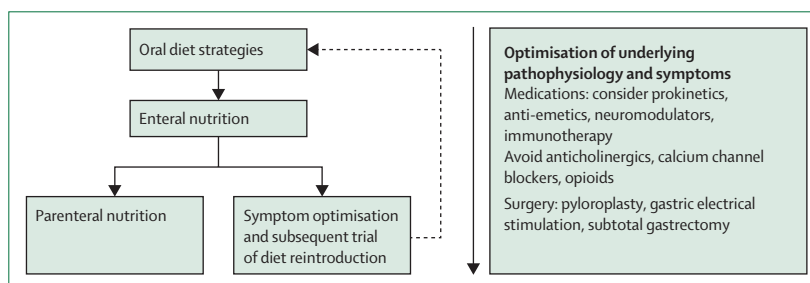
Figure 2: **Nutritional treatment pathway for patients with gastroparesis**
Enteral nutrition should be considered for patients with gastroparesis who do not respond to dietary approaches. Approximately half of these patients might not tolerate enteral nutrition and might therefore require parenteral nutrition. Optimisation of underlying pathophysiology and symptoms should be considered alongside the nutritional treatment pathway.

Search strategy and selection criteria

PubMed was searched for articles published from inception until Oct 29, 2019, with the following search string: "diet" or "nutrition" or "food" or "tube feed" or "supplement"; and "gastroparesis" or ("stomach" and ["delay" or "slow" or "dysmotility"]). Two reviewers screened the titles and abstracts of the 1006 articles retrieved for consideration in this Review. For each article, one of the reviewers had to be a nutrition specialist (BNL or NDS). Conflicts in screening decisions were adjudicated by discussion with the lead author (BNL). Inclusion criteria included studies or review articles focused on both nutrition and gastric emptying in humans. Review articles on gastroparesis published within the past 10 years that did not specifically focus on nutrition management were also included. Review articles that were not written in English language were excluded from consideration. The title and abstract screening phase excluded 913 articles and yielded 93 articles for full text review. Relevant references within included full-text articles and independent searches were also considered as needed.

Conclusion

The symptoms of gastroparesis often provoke suboptimal eating patterns and a high risk of malnutrition in patients with the disease. Attention to and improvement of nutritional status are therefore crucial and best done in tandem with optimisation of pharmacological, endoscopic, or surgical therapies, or a combination, to improve gastric function and emptying. Research into nutritional strategies for gastroparesis is scarce and the overall quality of evidence is poor. Nonetheless, there are strategies available to use (eg, modification of food composition, food consistency, and food volume) based on indirect evidence of factors that might influence gastric emptying. Because patients' tolerance can vary over time according to medical optimisation of their gastroparesis, the composition of their oral diet (ie, the relative proportions of solid and liquid food in their meals) will similarly require periodic reassessments and adjustments. If the patient is unable to maintain adequate caloric intake by mouth despite best efforts at nutritional and pharmacological optimisation, initiation of enteral nutrition should be considered. Most patients who are advanced to enteral nutrition appear to tolerate it well. Otherwise, parenteral nutrition would be an option for those whose gastrointestinal tract persistently rejects nutrition. Although not all patients with gastroparesis will be able to tolerate oral intake, at the least, various nutritional therapies are available to prevent and treat malnutrition in patients with this potentially debilitating condition.

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