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Gastroenterological Society of Australia Position Statement on the Assessment and Management of Idiopathic Gastroparesis

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ABSTRACT

Background: Idiopathic gastroparesis (IGP) treatment guidelines have to date focused on delayed gastric emptying as the cause of the associated symptoms of postprandial nausea, vomiting, early satiety, fullness, and pain. However the diagnostic hallmark—delayed gastric emptying—correlates poorly with symptoms, and the efficacy of treatments targeting gastric emptying is low. Clinically, there is substantial overlap with functional gastroduodenal disorders and eating disorders. As a result, current international guidelines provide limited guidance with low consensus. Recent advances support reconceptualizing IGP as a sensorimotor disorder, on a spectrum with functional dyspepsia, arising within the biopsychosocial model of disease.

Objective: To provide clear guidance to clinicians on the assessment and management of IGP, as a sensorimotor disorder, using a multidisciplinary framework.

Methods: Following formal review of the literature, a national multidisciplinary working group devised 20 consensus statements with a grade of evidence and strength of recommendation using a modified Delphi approach, and a suggested treatment algorithm. External review was obtained via patient representation, expert review, and public comment.

Conclusion: A novel treatment algorithm and 20 consensus statements are provided to guide clinicians in the multidisciplinary assessment and management of IGP as a sensorimotor disorder, alongside its overlapping comorbidities. We call for review of the current definition of IGP and a shift in research efforts to identify novel therapeutic targets.

The recommendations in this position statement represent the best available evidence at the time of compilation and are intended to be used only as a guide. Clinical decision making must be determined by the individual circumstances of each patient and is the responsibility of the treating clinician(s).

1 | Introduction

Gastroparesis has historically been defined as a condition presenting with the cardinal upper gastrointestinal symptoms of postprandial nausea, vomiting, early satiety, fullness, and bloating due to delayed gastric emptying in the absence of mechanical obstruction. Subtypes include idiopathic, diabetic, and postsurgical gastroparesis. This position statement refers specifically to idiopathic gastroparesis (IGP), where no cause can be identified using traditional diagnostic techniques. Global epidemiological data for IGP are lacking, and the population prevalence of asymptomatic delayed gastric emptying is unknown. Although considered a rare disease, IGP appears to be increasing in Western populations [1]. When severe, the individual and socioeconomic impacts are high. Available treatments targeting gastric emptying are often ineffective, and there have been few recent therapeutic developments. This is largely due to the historic emphasis on delayed gastric emptying as the cause of symptoms. This preconception has contributed to bias in study design, interpretation and therapeutic approach [2].

Although not a new concept, there is now increasing acceptance that IGP is a sensorimotor disease on a spectrum with functional gastroduodenal disorders. Functional dyspepsia and gastroparesis have been shown to be clinically indistinguishable [3] and there is substantial diagnostic overlap with other disorders of gut–brain interaction (DGBI) and eating disorders, particularly chronic nausea vomiting syndrome and rumination syndrome. Although there may be an academic argument to delineate IGP from functional dyspepsia based on cardinal symptoms—with nausea and vomiting more strongly associated with IGP, and postprandial early satiety and pain more strongly associated with functional dyspepsia [4, 5]—this somewhat arbitrary distinction will lead to ongoing limitations in research and clinical care.

This shift in concept is timely. In Western societies, presentations with gastroparesis-like syndromes are increasing in younger people, in the context of multisystem diagnoses of uncertain significance, persistent pain, eating disorders and marked psychosocial vulnerabilities. In turn, there is increased demand for invasive treatment modalities including nutrition support for IGP, with high risk of iatrogenic harm and economic burden. Patient expectations are increasingly shaped by health information obtained from the internet, most of which is not medically endorsed, and the impact of social media on abnormal illness behavior is substantial [6].

International guidelines from European [5] and North American societies [7] and the Rome Foundation [8] acknowledge these challenges surrounding IGP, including our limited understanding of symptom pathogenesis, poor correlation of symptoms with gastric emptying, overlapping diagnoses and lack of effective therapies. Despite this, the neurogastroenterology community has failed to progress past the historic focus on IGP as a motor disorder.

Accordingly, we present the first Australian position statement on the assessment and management of IGP as a sensorimotor disorder, including multidisciplinary treatment approaches for common overlapping disorders. Following formal literature review, our working group aimed to provide guidance that is highly clinically relevant, with clear consensus recommendations, with the aim of supporting all clinicians to improve the lives of patients living with this challenging disorder.

2 | Methodology

This position statement was conceived in the October 2024 Gastroenterological Society of Australia (GESA) Luminal Faculty Committee meeting. Working group invitations were sent to clinicians and academics nationally with expertise in gastroparesis, DGBI, and eating disorders, aiming for equal and diverse representation across specialities and viewpoints. A final working group of 12 members was formed representing gastroenterology, dietetics, psychology, and psychiatry. Patient and public involvement was provided by representatives from the GESA Luminal Faculty patient advocacy group, which included lived-experience and family and carer members. The working group drafted the document, with sections allocated according to expertise. Authors undertook a formal review of the literature using MEDLINE, EMBASE, PubMed, CINAHL, and PsycINFO databases with university research-librarian input. Inclusion criteria were peer-reviewed articles reporting studies in adults, published in English between January 1985 and January 2025.

External review of the initial draft was sought broadly from experts both nationally and internationally in gastrointestinal surgery, radiology, mental health, eating disorders, general and pediatric gastroenterology, and intestinal failure (see Acknowledgements). Feedback was incorporated through multiple revisions before 20 consensus statements were drafted. The Grading of Recommendations, Assessment, Development and Evaluation (GRADE) process was applied to the 20 statements using a standard template to assess the quality of evidence as high, moderate, low, or very low [9], and the strength of recommendation as strong or conditional. The quality of evidence was deemed not applicable for statements considered good practice points by consensus. Where the quality of supporting evidence was low or very low for poorly researched question, a strong recommendation may still be appropriate. The references included in this document represent only the most critical of the articles reviewed.

A modified Delphi approach was applied to the 20 statements with grading by the working group members via an anonymous online survey in September 2025. A 4-point Likert scale (strongly agree, agree, disagree, strongly disagree) was used. Consensus was deemed ≥ 85% agreement or strong agreement, with 80%–84% agreement deemed borderline endorsement. Only one round of voting was required, with no major revisions to statements or grading; no statements were removed (see Table 1). Disputes were included as discussion points, with reference to the Appraisal of Guidelines for Research and Evaluation (AGREE) reporting checklist. The document was presented for public comment at the World Congress of Gastroenterology@Australian Gastroenterology Week 2025 in Melbourne, Australia, September 2025. Feedback was incorporated before finalization. An extended version

TABLE 1 | Summary of consensus statements.

No.	Statement	Endorsed	Quality of evidence[a]	Strength of recommendation[a]	Agreement[b]
1	Idiopathic gastroparesis is a sensorimotor disorder. There is substantial overlap with functional gastroduodenal disorders and eating disorders.	Yes	Low	Strong	**100%** SA: 100%
2	A comprehensive medical, surgical, and psychosocial history is needed, including psychological comorbidity and nutritional assessment.	Yes	NA	Consensus	**100%** SA: 100%
3	Co-assessment by a clinician specializing in eating disorders is recommended for all patients with disordered eating behavior, due to the high comorbid prevalence of disordered eating and eating disorders.	Yes	Low	Strong	**100%** SA: 75% A: 25%
4	Initial work-up should include all tests indicated in the clinical context to identify structural gastrointestinal and systemic diseases.	Yes	NA	Consensus	**92%** SA: 67% A: 25% D: 8%
5	The rate of gastric emptying correlates poorly with symptoms and assesses only one aspect of idiopathic gastroparesis. Sensory abnormalities are not measured by available tests. Gastric emptying studies must be considered only one part of a broad clinical assessment.	Yes	Moderate	Strong	**100%** SA: 67% A: 33%
6	The recommended nuclear scintigraphy test should include a standardized low-fat egg-based meal or a validated variant, with greater than 10% gastric retention at 4 h considered abnormal.	Yes	Low	Strong	**100%** SA: 42% A: 58%
7	When modifiable factors are present, a repeat gastric emptying study should be considered 3–12 months after an abnormal result, following optimisation of all reversible factors, to improve validity.	Borderline	Very low	Conditional	**84%** SA: 17% A: 67% D: 17%
8	Routine assessment for vascular compression syndromes, hypermobility spectrum disorders, mast cell disorders, autonomic dysfunction, and microbial dysbiosis is not recommended. If suspected, subspecialist input is recommended to guide appropriate testing and interpretation of test results in the clinical context.	Yes	Low	Strong	**100%** SA: 58% A: 42%
9	All patients with gastroparesis should undergo a comprehensive nutritional assessment by a gastrointestinal dietitian at diagnosis and as clinically needed thereafter.	Yes	Low	Strong	**100%** SA: 75% A: 25%
10	Dietary therapy should prioritize oral nutritional rehabilitation, with the aim of improving symptoms whenever possible while not compromising nutritional status.	Yes	Low	Strong	**100%** SA: 83% A: 17%

(Continues)

TABLE 1 | (Continued)

No.	Statement	Endorsed	Quality of evidence[a]	Strength of recommendation[a]	Agreement[b]
11	Temporary nasogastric tube feeding should only be considered where there is malnutrition, with ongoing weight loss, and medical instability, despite intensive oral nutritional support.	Yes	Low	Strong	**100%** SA: 58% A: 42%
12	The decision to initiate long-term enteral tube feeding should be made only with formal multidisciplinary team consultation.	Yes	NA	Consensus	**100%** SA: 75% A: 25%
13	Long-term enteral tube feeding should be avoided where possible. It has not been shown to consistently improve global symptoms or nutritional status and carries increased risk of iatrogenic harm.	Yes	Low	Strong	**100%** SA: 75% A: 25%
14	There is no evidence supporting parenteral nutrition in gastroparesis and, given the risk of complications, it should be avoided.	Yes	Low	Strong	**92%** SA: 67% A: 25% D: 8%
15	Limited evidence supports a trial of prokinetic therapy in idiopathic gastroparesis, whereas the use of antiemetics is largely empirical. Metoclopramide or domperidone is recommended first-line treatment.	Yes	Low	Conditional	**100%** SA: 50% A: 50%
16	Neuromodulators are under-researched in idiopathic gastroparesis, though evidence-based in disorders of gut–brain interaction. Given the overlap in functional gastroduodenal symptoms, neuromodulators are recommended adjunctive treatment, with choice of agent targeting the predominant gastrointestinal symptoms.	Yes	Low	Conditional	**100%** SA: 75% A: 25%
17	Cannabinoids slow gastric emptying but, paradoxically, may improve symptoms of gastroparesis, including satiation. There is insufficient evidence to recommend their use.	Yes	Low	Conditional	**100%** SA: 50% A: 50%
18	Mental health clinicians are recommended core members of the multidisciplinary care team for all individuals with idiopathic gastroparesis and significant psychosocial or psychiatric comorbidity.	Yes	Low	Strong	**100%** SA: 75% A: 25%
19	Evidence-based psychological interventions for overlapping disorders, such as disorders of gut–brain interaction and persistent pain disorders, should be provided early in the treatment of idiopathic gastroparesis.	Yes	Low	Strong	**100%** SA: 83% A: 17%

(Continues)

TABLE 1 | (Continued)

No.	Statement	Endorsed	Quality of evidence[a]	Strength of recommendation[a]	Agreement[b]
20	There is insufficient evidence to recommend intrapyloric botulinum toxin injection, surgical pyloroplasty, gastric electrical stimulation, or gastric peroral endoscopic myotomy in medically refractory idiopathic gastroparesis. These therapies should only be trialed following multidisciplinary team consensus.	Yes	Low	Conditional	**92%** SA: 50% A: 42% D: 8%

Abbreviations: A = agree; D = disagree; NA = not applicable; SA = strongly agree.

[a]Quality of evidence and strength of recommendation were rated according to Grading of Recommendations Assessment, Development and Evaluation (GRADE). Quality of evidence was deemed not applicable for statements considered good practice points by consensus, where a literature search was not relevant.

[b]Agreement was rated using a modified Delphi consensus approach. Statements were endorsed when ≥ 85% of working group members agreed or strongly agreed and deemed borderline when 80%–84% agreed or strongly agreed. Percentages are rounded to the nearest whole number.

is available at https://www.gesa.org.au/resources/position-statements/. This position statement was endorsed by the Australasian Neurogastroenterology and Motility Association, Australia New Zealand Academy for Eating Disorders, Australasian Society of Parenteral and Enteral Nutrition, Australasian Association of Nuclear Medicine Specialists, and New Zealand Society of Gastroenterology.

3 | Assessment

3.1 | Overview

Recent advances have shown the symptom pathogenesis in IGP to be much more complex than delayed gastric emptying alone. Abnormal gastric accommodation and contractility, gastric arrhythmias, pyloric dysfunction, small bowel dysmotility, and visceral hypersensitivity have all been documented [10], highlighting that IGP is better conceptualized as a sensorimotor disorder. There is substantial diagnostic overlap with functional gastroduodenal disorders and eating disorders, and limited pathophysiological information is provided by available testing. As a result, suspected IGP requires a comprehensive biopsychosocial assessment.

3.2 | Clinical Assessment

A complete medical, nutritional, and psychosocial history is essential. All prescription and non-prescription medications should be reviewed, particularly opioid, anticholinergic, antimuscarinic, antispasmodic, antipsychotic and centrally acting agents, weight loss agents, cannabinoids, and illicit substances, as these may alter gastric emptying or symptoms. If cessation is not feasible, the limitations of motility testing must be explained.

Time should be allowed to explore past and present psychological and neurodevelopmental comorbidities and perpetuating factors including adverse life events, with trauma-informed, neurodiversity-affirming care where relevant. This may be performed by the physician or a collaborating mental health specialist and provides an opportunity to build trust, dispel stigma, and correct misinformation. Advanced communication training and mental health supervision may benefit clinicians.

Eating disorders, DGBI, and delayed gastric emptying frequently coexist. Studies have shown 20%–80% of patients with an eating disorder may have delayed gastric emptying [11], and up to 98% experience functional gastrointestinal symptoms [12]. Delayed emptying has been demonstrated in both restrictive and binge-purge eating patterns at similar rates, not reliant on body mass index. Disordered eating behavior may pre-date IGP, or develop in attempt to minimize the symptoms of IGP, or they may co-develop in a bidirectional manner. A thorough review of this topic is provided elsewhere [13]. The diagnostic overlap between avoidant restrictive food intake disorder (ARFID), and DGBI with restricted oral intake in particular requires future attention [14]. Co-assessment by a clinician with expertise in eating disorders is strongly recommended for all patients with IGP and disordered eating behavior, where "disordered eating behavior" is used here to encompasses symptoms that may represent either disordered eating or a formal eating disorder.

Statement 1. IGP is a sensorimotor disorder. There is substantial overlap with functional gastroduodenal disorders and eating disorders. (Low quality of evidence; Strong recommendation).

Statement 2. A comprehensive medical, surgical, and psychosocial history is needed, including psychological comorbidity and nutritional assessment. (Quality of evidence not applicable; Consensus recommendation).

Statement 3. Co-assessment by a clinician specializing in eating disorders is recommended for all patients with disordered eating behavior, due to the high comorbid prevalence of disordered eating and eating disorders. (Low quality of evidence; Strong recommendation).

3.3 | Initial Investigations

Structural gastrointestinal abnormalities including mechanical gastric outlet obstruction should be excluded initially with upper gastrointestinal endoscopy. Alarm features should prompt consideration of urgent endoscopy and cross-sectional imaging. Biopsies for eosinophils and mast cells are not recommended in the absence of relevant reference intervals [15]. Radiological investigation may include abdominal imaging with consideration of small bowel and biliary tract imaging for pain-predominant presentations, and central nervous system imaging for persistent unexplained nausea or focal neurology.

Basic blood tests should include measurement of hemoglobin, electrolytes, blood glucose, coeliac serology, thyroid function tests, haematinics, and macro- and micronutrient screens. Assessment of alternative systemic causes, such as neurological, rheumatological, and endocrine disorders, is determined by the clinical context.

The "test and treat" approach to *Helicobacter pylori* eradication has not been studied in IGP. Given the overlap with functional dyspepsia, we support offering individualized eradication following discussion of the limited anticipated symptom benefit and antimicrobial resistance considerations.

Statement 4. Initial work-up should include all tests indicated in the clinical context to identify structural gastrointestinal and systemic diseases. (Quality of evidence not applicable; Consensus recommendation).

3.4 | Measurement of Gastric Emptying

By definition, the diagnosis of IGP requires measurement of gastric emptying. However, gastric emptying is a composite endpoint and dynamic process which is constantly responding to intrinsic and extrinsic factors, and even in the highest-quality studies symptom correlation is weak [16] and gastric emptying rate is highly variable over time [3], questioning the pivotal role of this test. Current guidelines recommend 4-h gastric emptying scintigraphy using a standardized egg-based solid test meal, with greater than 10% retention at 4 h deemed abnormal [8]. The percentage retained does not stratify symptom severity or predict treatment response [17]. ^{13}C-breath testing is an accepted alternative. Solid and mixed liquid–solid meal variants may be offered based on allergy or cultural preferences if validated reference ranges are available. Higher-calorie mixed-composition meals may be more physiological [8] but are not yet protocolised. In Australia, the lack of a commercially available test meal limits its standardization and comparability, a gap requiring future attention [18].

Confounding factors must be optimized before performing gastric emptying studies, including medications and non-prescription substances, malnutrition, and disordered eating behaviors. Potential confounders should be clearly documented to assist interpretation of the test in the clinical context. Where modifiable factors are present, an abnormal gastric emptying study may be repeated within 3–12 months, after optimization. However, this achieved only borderline consensus (see Statement 7) and was a major discussion point within the working group, due to the fundamental limitations of gastric emptying measurement.

Gastric emptying assesses only one pathophysiological mechanism identified in IGP. As a sensorimotor disorder, tests of gastroduodenal sensorimotor function may be standardized as routine tests with clinically relevant reference ranges in future but remain research tools in most centers at present. These include gastric magnetic resonance imaging, ultrasound, barostat, nutrient drink challenge, antroduodenal manometry, wireless capsules, pyloric distensibility, and body surface gastric mapping electrogastrography [19]. Retained gastric contents at endoscopy or prolonged retention of a contrast meal may suggest delayed gastric emptying but are not sufficiently specific for diagnostic use.

Statement 5. The rate of gastric emptying correlates poorly with symptoms and measures only one aspect of IGP. Sensory abnormalities are not measured by available tests. Gastric emptying studies must be considered only one part of a broad clinical assessment. (Moderate quality of evidence; Strong recommendation).

Statement 6. The recommended nuclear scintigraphy test should include a standardized low-fat egg-based meal or a validated variant, with greater than 10% gastric retention at 4 h considered abnormal. (Low quality of evidence; Strong recommendation).

Statement 7. When modifiable factors are present, a repeat gastric emptying study should be considered 3–12 months after an abnormal result, following optimization of all reversible factors, to improve validity. (Very low quality of evidence; Conditional recommendation; borderline endorsement).

3.5 | Further Investigations

There is insufficient evidence to support a causative association or routine testing for vascular compression syndromes, hypermobility spectrum disorders, mast cell activation syndrome, autonomic dysfunction, small intestinal bacterial overgrowth, or microbial dysbiosis in patients with gastroparesis. Subspeciality input is required if these disorders are suspected.

Statement 8. Routine assessment for vascular compression syndromes, hypermobility spectrum disorders, mast cell disorders, autonomic dysfunction and microbial dysbiosis is not recommended. If suspected, subspecialist input is recommended to guide appropriate testing and interpretation of test results in the clinical context. (Low quality of evidence; Strong recommendation).

4 | Management

4.1 | Overview

Targeting gastric emptying alone has proven ineffective. Acknowledging that IGP is a sensorimotor disorder should prompt consideration of using the symptom-based therapies established in functional gastroduodenal disorders as adjuncts, within the biopsychosocial model of care (see Figure 1). In complex cases, formal multidisciplinary team (MDT) input is recommended, via consultation or peer support, and should include medical, surgical, nutrition, and mental health clinicians with expertise in gastroduodenal dysfunction. As few treatments targeting visceral hypersensitivity have been studied specifically in IGP, recommendations are extrapolated from key overlapping disorders, as appropriate.

4.2 | Nutritional Management

4.2.1 | Nutritional Assessment and Monitoring

Gastroparesis presents unique nutritional challenges. All patients require a comprehensive nutritional assessment by a gastrointestinal-focussed dietitian at diagnosis and as clinically indicated thereafter, including evaluation of macro- and micronutrient intake, eating behaviors, body image, food beliefs, previous dietary interventions, and related quality of life. Patients with gastroparesis have high rates of micronutrient deficiencies, including vitamin D (61%), vitamin E (80%), folate (68%), calcium (70%), iron (69%), magnesium (72%), and potassium (86%) [20]. Dietary interventions should be assessed using validated tools, including body composition measurements, micronutrient assessments, and symptom surveys. When disordered eating behavior is identified, co-management with an eating disorder service is recommended.

Statement 9. All patients with gastroparesis should undergo a comprehensive nutritional assessment by a gastrointestinal dietitian at diagnosis and as clinically needed thereafter. (Low quality of evidence; Strong recommendation).

4.2.2 | Dietary Interventions

The majority of patients with IGP can and should be managed with oral nutritional rehabilitation. Up to 60% of patients respond symptomatically to dietary therapy combined with prokinetic medication [21]. Dietary therapy should be prescribed by a gastrointestinal dietitian and may be tailored to the patient's symptoms, although meeting nutritional requirements should always maintain priority over symptom management. Figure 2 provides a suggested decision-making framework for selecting dietary interventions based on nutritional status and symptoms, with sample meal plans provided in Appendix 1. Appendix 2 summarizes the dietary approaches studied in gastroparesis.

Support to maintain smaller, frequent meals (6–10 meals daily) is recommended [22], well-chewed or blended, as part of a strategy termed "effortful eating." Oral nutritional supplements are recommended when oral intake is inadequate. Remaining upright for 1–2 h after meals may be helpful. A low-fat diet is not of proven benefit in gastroparesis [23], and is contraindicated in individuals with malnutrition.

Dietary strategies established in functional dyspepsia may be carefully considered. A low-FODMAP (fermentable oligosaccharides, disaccharides, monosaccharides, and polyols) diet has some evidence for reducing epigastric symptoms, early satiety, bloating, and abdominal pain in functional dyspepsia [24] and may be trialed for a period of 6 weeks, before reintroducing food groups. Restrictive dietary approaches are contraindicated where malnutrition or disordered eating behaviors are present.

Constipation should be well managed in patients with IGP using standard treatments [25]. Although it is difficult to separate cause and effect in chronic constipation and delayed gastric emptying, regular bowel clearance is expected to reduce overlapping symptoms and may optimize gastrointestinal motility [26, 27].

Statement 10. Dietary therapy should prioritize oral nutritional rehabilitation, with the aim of improving symptoms whenever possible while not compromising nutritional status. (Low quality of evidence; Strong recommendation).

4.2.3 | Artificial Nutritional Support Considerations

The initiation of enteral tube feeding (ETF) must be carefully considered and approached with caution. For patients with medical instability from severe malnutrition who require immediate intervention, temporary nasogastric tube feeding may be considered short-term until medically stabilized. Nasogastric feeding is recommended over post-pyloric feeding, as gastric emptying correlates poorly with symptoms [28]. Long-term ETF should only be initiated with tertiary MDT input (see Table 2), which should address any overlapping DGBI, persistent pain, and psychiatric comorbidities. Long-term ETF does not appear to consistently relieve symptoms or nutritional status (see Appendix 3) and carries the risk of perpetuating food avoidance and iatrogenic harm [21]. Long-term ETF should be reserved only for those at medical risk due to severe malnutrition after all reasonable non-invasive approaches have been attempted with MDT support, and with ongoing planned reviews to support oral rehabilitation and ETF removal whenever possible.

There is no evidence supporting the use of parenteral nutrition in patients with gastroparesis and, given the risk of complications, it should be avoided. Parenteral nutrition is associated with a significantly higher risk of infectious complications than other nutritional approaches, without long-term survival benefit [29]. If ETF is not tolerated because of symptoms, intensive

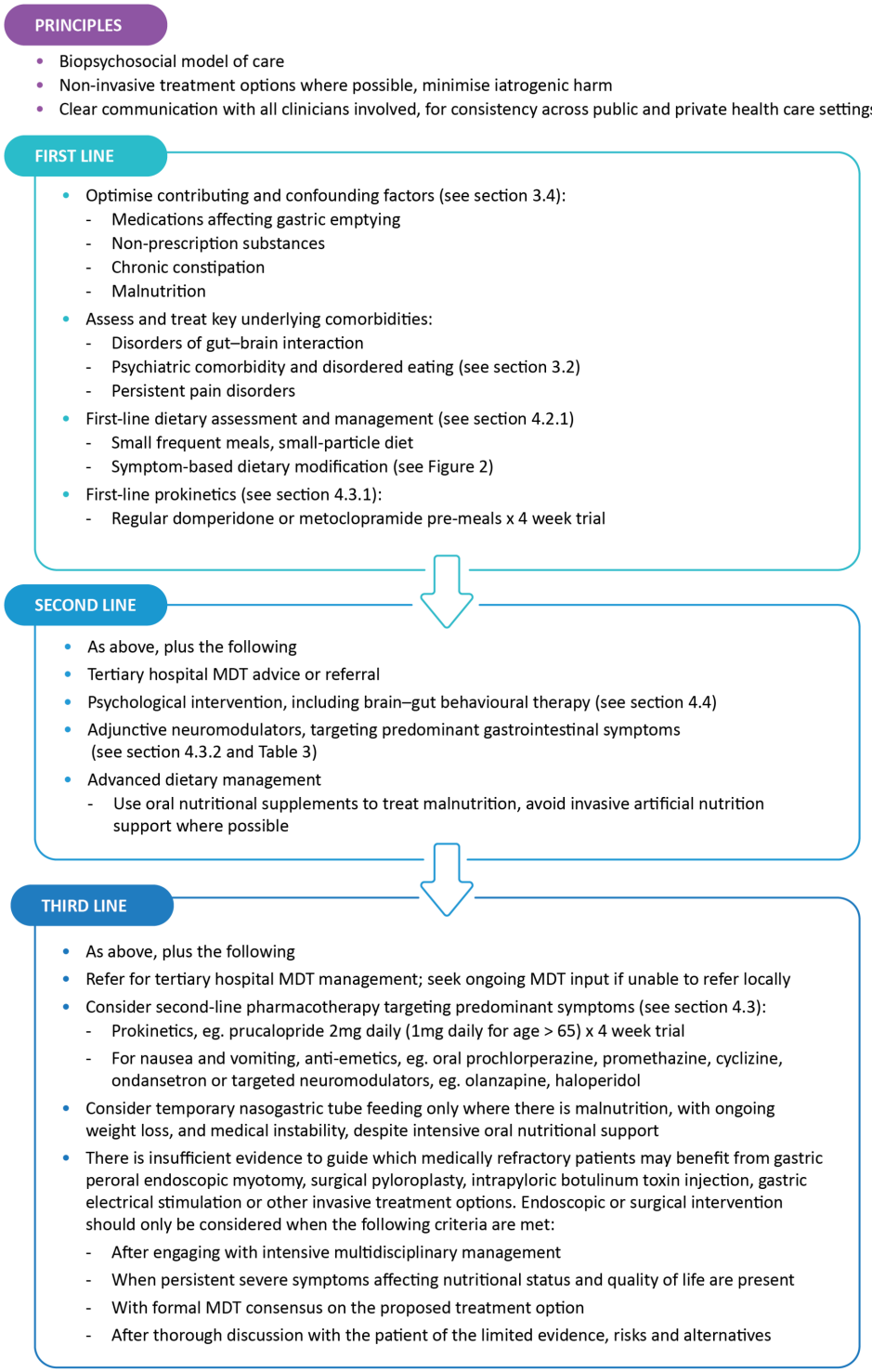

FIGURE 1 | Treatment algorithm for idiopathic gastroparesis. MDT = multidisciplinary team.

multidisciplinary management of the associated DGBI is recommended, rather than escalation to parenteral nutrition.

Statement 11. Temporary nasogastric tube feeding should only be considered where there is malnutrition, with ongoing weight loss, and medical instability, despite intensive oral nutritional support. (Low quality of evidence; Strong recommendation).

Statement 12. The decision to initiate long-term ETF should be made only with formal multidisciplinary team consultation. (Quality of evidence not applicable; Consensus recommendation).

Statement 13. Long-term ETF should be avoided where possible. It has not been shown to consistently improve global symptoms or nutritional status and carries increased risk of iatrogenic harm. (Low quality of evidence; Strong recommendation).

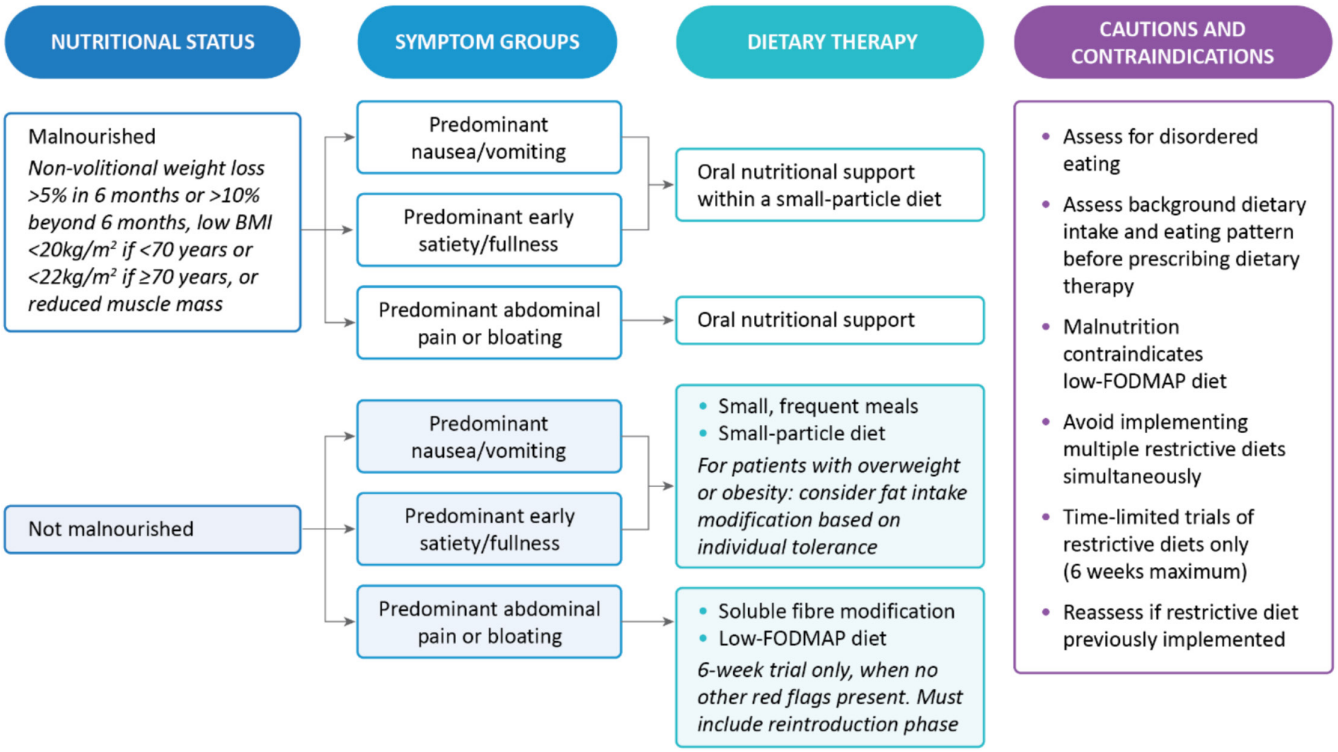

FIGURE 2 | Decision framework for nutritional recommendations in idiopathic gastroparesis. This framework guides individualized nutritional management based on a patients nutritional status and predominant symptoms. See Appendix 1 for small-particle and texture-modified meal plans. BMI = body mass index; FODMAP = fermentable oligosaccharides, disaccharides, monosaccharides, and polyols.

Statement 14. There is no evidence supporting parenteral nutrition in gastroparesis and, given the risk of complications, it should be avoided. (Low quality of evidence; Strong recommendation).

4.3 | Pharmacotherapy

4.3.1 | Prokinetics and Antiemetics

Minimal research has been undertaken on prokinetics specific to IGP, and it is uncertain whether outcomes from functional dyspepsia and diabetic gastroparesis can be generalized to IGP. A network meta-analysis of 29 trials in patients with gastroparesis of any etiology indicated symptom benefit over placebo for dopamine antagonists [30]. A separate meta-analysis of 29 trials of prokinetics in patients with functional dyspepsia indicated global symptom benefit [31].

Metoclopramide and domperidone are the only prokinetics approved for use for gastroparesis in Australia. Only one of four placebo-controlled trials of metoclopramide in gastroparesis included patients with IGP; it showed symptomatic improvement after 3 weeks [32]. Adverse effects may include acute dystonia, prolonged QT interval, tardive dyskinesia, hyperprolactinaemia, and galactorrhoea. Domperidone does not cross the blood–brain barrier, reducing neurological side effects. Despite favorable evidence in diabetic gastroparesis, only one of six placebo-controlled trials of domperidone included patients with IGP, but it did show symptom benefit [33].

Use of prucalopride and erythromycin for patients with IGP is off-label. Erythromycin improved gastric emptying and symptoms in IGP short-term in one uncontrolled study; tachyphylaxis limits its long-term utility [34]. Prucalopride, which is approved in severe constipation and devoid of cardiac effects, reduced symptoms and improved gastric emptying compared with placebo in a 4-week double-blind crossover study predominantly involving patients with IGP [35]. Cisapride, a 5-HT_4 receptor agonist, was withdrawn due to cardiac arrhythmias.

Antiemetics such as phenothiazines (e.g., prochlorperazine) and antihistamines (e.g., promethazine, cyclizine) are used empirically in IGP. Intravenous administration of cyclizine can induce euphoria and dependence [36]. Haloperidol was found to be superior to placebo for treating nausea in emergency presentations of patients with gastroparesis [37]. The 5-HT_3 receptor antagonist granisetron reduced nausea and vomiting in open-label IGP studies [38]. Although a study found that the NK-1 receptor antagonist aprepitant reduced "gastroparesis-like" nausea and vomiting compared with placebo, it did not satisfy the primary outcome [39].

Statement 15. Limited evidence supports a trial of prokinetic therapy in IGP, whereas the use of antiemetics is largely empirical. Metoclopramide or domperidone is recommended first-line treatment. (Low quality of evidence; Conditional recommendation).

4.3.2 | Pharmacological Neuromodulation

Though widely used, few studies have formally assessed neuromodulator medications in patients with IGP. The

TABLE 2 | Multidisciplinary team decision-making principles for nasogastric feeding.

Principle	Description
Assessment	Comprehensive medical, nutritional, and psychosocial assessment should be completed to assess for coexisting structural, psychosocial, and psychiatric contributors, including disordered eating behavior
Indication	ETF should be considered only for patients who are severely malnourished with ongoing objective weight loss despite MDT-guided oral nutritional rehabilitation
Symptom management	ETF is indicated for nutritional support in patients with severe malnutrition, not primarily for symptom relief
Risk–benefit assessment	The risks of ETF (including perpetuation of disordered eating patterns, difficulty weaning and complications) must be weighed against potential benefits in an individualized assessment
Exit strategy	A clear exit strategy with defined nutritional goals should be established before initiating ETF
Weight considerations	For patients with high body weight who have experienced significant recent weight loss (> 10% within 6 months), the risk of malnutrition complications versus risks of invasive intervention must be carefully balanced

Abbreviations: ETF = enteral tube feeding; MDT = multidisciplinary team.

only placebo-controlled randomized controlled trial (RCT), Nortriptyline for Idiopathic Gastroparesis (NORIG) published 2013, found no difference in the primary outcome of 50% reduction in symptom scores with escalating nortriptyline 10–75 mg versus placebo over 12–15 weeks. Treatment escalation and completion was limited in both groups due to medication intolerance [40]. A strong placebo response with short follow-up intervals and small completion numbers were limitations; further studies are needed.

Amitriptyline has shown symptom benefit in functional dyspepsia. One RCT ($n = 292$) included 21% with delayed gastric emptying. Amitriptyline 50 mg daily over 12 weeks reduced functional dyspepsia threefold compared with placebo, though patients with delayed gastric emptying were less likely to respond. Neither nortriptyline nor amitriptyline worsened gastric emptying delay [41, 42].

An open-label trial of mirtazapine 15 mg daily in 30 patients with IGP showed improvements in nausea, vomiting and appetite at 2 and 4 weeks. 20% of participants ceased due to adverse effects [43]. Another 8-week RCT showed improvement in postprandial symptoms of functional dyspepsia, but gastric emptying was not measured [44].

The Buspirone for Early Satiety and Symptoms of Gastroparesis (BESST) trial compared 4 weeks of buspirone with placebo in 96 patients with moderate to severe gastrointestinal symptoms, of whom 50% had delayed gastric emptying. Despite no global GCSI score benefit, there was modest improvement in bloating scores, irrespective of gastric emptying time [45].

The atypical antipsychotics olanzapine and quetiapine are used as adjunctive therapy for functional nausea, and serotonin–noradrenaline reuptake inhibitors for unexplained pain, though neither group has been studied in IGP.

Our working group recommends the use of neuromodulators in patients with IGP as second-line therapy (see Figure 1). In the absence of IGP-specific trials, or a primary psychiatric indication to guide therapy, choice of neuromodulator should be based on the patient's predominant gastrointestinal symptoms, with reference to the Rome Foundation 2018 report (Table 3) [46].

Statement 16. Neuromodulators are under-researched in IGP, though evidence-based in DGBI. Given the overlap in functional gastroduodenal symptoms, neuromodulators are recommended adjunctive treatment, with choice of agent targeting the predominant gastrointestinal symptoms. (Low quality of evidence; Conditional recommendation).

4.3.3 | Cannabinoids

A single placebo-controlled RCT of cannabidiol in 44 patients with gastroparesis found a reduction in global GCSI score and vomiting episodes despite slower gastric emptying [47]. Large epidemiological studies indicate higher health care utilization with cannabinoids [48], and their use remains controversial.

Statement 17. Cannabinoids slow gastric emptying but, paradoxically, may improve symptoms of gastroparesis, including satiation. There is insufficient evidence to recommend their use. (Low quality of evidence; Conditional recommendation).

4.4 | Psychological Interventions

There is a paucity of research into psychological interventions in gastroparesis, due to the research emphasis on motility. Only one study has been published, in postsurgical gastroparesis. This study found that psychosocial support, plus music and massage therapy, and family psychoeducation, improved mood and residual gastric volume compared with standard medical care. This is notable given the apparent "structural" pathophysiology underlying symptoms in post-surgical gastroparesis is perceived to be higher than in IGP. The IGP overlap with functional dyspepsia suggests that brain-gut interventions which are established in DGBI should be considered in IGP [49]. In DGBI, multidisciplinary treatment that includes mental health clinicians has been shown to improve patient-reported outcomes and cost-effectiveness [50]. However, brain-gut behavioral therapies are under-researched and underused even in DGBI.

There is, however, a broader indication for mental health support in IGP. Gastroparesis is associated with significant

TABLE 3 | Summary of gut–brain neuromodulatory medications[a].

Drug class, drug	Mode of action	Actions on GI sensorimotor function		Relevance to symptom control	Side effects
TCA					
• Amitriptyline • Imipramine • Desipramine • Nortriptyline	• Presynaptic SRI and NRI. • Antagonism/inhibition of multiple post-synaptic (5-HT2, 5-HT3, H1, muscarinic-1, α1) and presynaptic (α2) receptors.	Motility: slow GI transit, largely related to their anticholinergic and noradrenergic properties	Sensitivity: limited and inconsistent evidence that TCAs decrease visceral sensitivity	• Pain reduction. • Best documented for IBS, but also FD (EPS). • Potential usefulness in all FGIDs where pain is a prominent feature. • Side effect profile can be useful in order to reduce diarrhea and improve sleep.	• Drowsiness, • Dry mouth, • Constipation, • Sexual dysfunction, • Arrhythmias, and • Weight gain
SSRI					
• Citalopram, • Escitalopram, • Fluoxetine, • Paroxetine, • Sertraline	• Presynaptic SRI.	Motility: enhancement of gastric and small bowel propulsive motility	Sensitivity: no major impact on visceral sensitivity in healthy subjects or patients with FGIDs	• Treatment of associated anxiety, phobic features, and OCD in FGIDs.	• Agitation, • Diarrhea, • Insomnia, • Night sweats, • Headache, • Weight loss, and • Sexual dysfunction.
SNRI					
• Duloxetine, • Milnacipran, • Venlafaxine	• Pre-synaptic SRI and NRI. Equally strong for duloxetine. • NRI for venlafaxine in higher doses. • Milnacipran stronger NRI than SRI effects.	Motility: inhibitory effect on gastric and colonic tone, but not to the degree of TCAs; more studies are needed	Sensitivity: few studies available; area requiring further research	• Treatment of associated pain (based on efficacy in fibromyalgia, back pain, and headache) in FGIDs. • Potential use for painful FGIDs; however, formal evidence in treatment of specific FGID-related pain is lacking.	• Nausea, • Agitation, • Dizziness, • Sleep disturbance, • Fatigue, and • Liver dysfunction
NA and specific serotonergic antidepressants					
• Mirtazapine, • Mianserin, • Trazodone	• Indirect effects resulting in increased NA and serotonergic activity through α2 antagonism on NA and 5-HT neurons. • Also 5-HT2, 5-HT3, H1, muscarinic-1 antagonism	Motility: lack of detailed studies	Sensitivity: lack of detailed studies	• Potential use for treatment of early satiation, weight loss, and chronic nausea/vomiting. • Side effect profile can be useful to improve sleep.	• Sedation, • Headache, • Dry mouth, and • Weight gain

(Continues)

TABLE 3 | (Continued)

Drug class, drug	Mode of action	Actions on GI sensorimotor function		Relevance to symptom control	Side effects
Azapirones					
• Buspirone, • Tandospirone	• Partial pre- and post-synaptic 5-HT1 agonists	Motility: enhanced esophageal contractions and increased gastric accommodation in health and FD	Sensitivity: limited data suggest no effect	• Treatment of associated anxiety. • Potential use for treatment of early satiety, fullness, and nausea, but consistent evidence in FGIDs is lacking.	• Sedation, • Headache, and • Vertigo
Atypical antipsychotics					
• Aripiprazole, • Levosulpiride, • Olanzapine, • Quetiapine, • Sulpiride	• D2 receptor antagonism as main mechanism. • Partial D2 agonism for the sulpirides. • Various profiles of 5-HT2A antagonism (olanzapine, quetiapine), 5-HT1A agonism (quetiapine), H1, α1, α2, muscarinic-1 receptor antagonism.	Motility: lack of data	Sensitivity: limited data suggest decreased gastric sensitivity in functional dyspepsia	• Potential use in augmentation for pain reduction; however, formal evidence in treatment of specific FGID pain currently lacking. • Low evidence in FGIDs. • Potential use of sulpirides for nausea and dyspepsia, but formal evidence is lacking. • Improved sleep.	• Sedation, • Dizziness, • Weight gain, • Hyperlipidemia, and • Diabetes
Delta ligand agents					
• Gabapentin, • Pregabalin	• α2δ subunit blockage of (mostly presynaptic) voltage-sensitive calcium channels	Motility: no data	Sensitivity: decreased sensitivity to rectal distension in IBS	• Treatment of associated general anxiety disorder or fibromyalgia/abdominal wall pain. • Potential use for treatment of neuropathic pain in FGIDs. However, formal evidence in FGIDs is lacking.	• Sedation, • Headache, • Vertigo, • Weight gain, and • Peripheral edema.

Abbreviations: EPS = epigastric pain syndrome; FD = functional dyspepsia; FGID = functional gastrointestinal disorder; GI = gastrointestinal; IBS = irritable bowel syndrome; NA = noradrenaline; NRI = noradrenaline reuptake inhibition; OCD = obsessive-compulsive disorder; SNRI = serotonin–noradrenaline reuptake inhibitor; SRI = serotonin inhibition; SSRI = selective serotonin reuptake inhibitor; TCA = tricyclic antidepressant.

[a]Reprinted from Gastroenterology, Volume 154 (4), Drossman DA, Tack J, Ford AC, et al., Neuromodulators for functional gastrointestinal disorders (disorders of gut–brain interaction): a Rome Foundation Working Team report, pages 1146–1147, Copyright 2018, with permission from Elsevier.

psychosocial burden and low quality of life [51]. Anxiety and depression have a pooled prevalence of 49% and 39%, respectively [52], higher than the 27% for each in DGBI [53]. Disorders of mood, sleep, personality, trauma, eating, and persistent pain frequently co-occur in this cohort. As such, cognitive behavioral therapy, hypnosis, mindfulness-based stress reduction, and acceptance and commitment-based therapy benefit many, and for all patients living with severe chronic illness, psychological intervention provides support and neuromodulation to improve functionality and quality of life. For all IGP patients with disordered eating behavior, early and ongoing co-management by mental health clinicians with eating disorder expertise is recommended. The vast majority of studies have shown improvement in gastric emptying and functional GI symptoms post-eating disorder treatment, noting variability in outcomes due to small sample sizes and non-standardized testing protocols [54].

From the clinician's perspective, patient experiences of trauma, personality vulnerabilities and abnormal illness behavior can heavily impact healthcare interactions and therapeutic outcomes. Countertransference (put simply, the clinician's unconscious feelings and reactions to the patient's feelings and behaviors toward them) and splitting (a psychological defense mechanism where all-or-nothing thinking is felt, for example, by the patient toward the treating team) may complicate the therapeutic relationship. These factors, particularly when combined with the feelings of helplessness that health practitioners may experience in the face of chronic illness, can increase the risk of iatrogenic harm through inappropriate rejection, fragmentation or escalation of care. Mental health clinicians are the core members of the MDT with the formal expertise needed to assess and manage this facet of care.

Statement 18. Mental health clinicians are recommended core members of the multidisciplinary care team for all individuals with IGP and significant psychosocial or psychiatric comorbidity. (Low quality of evidence; Strong recommendation).

Statement 19. Evidence-based psychological interventions for overlapping disorders, such as DGBI and persistent pain disorders, should be provided early in the treatment of IGP. (Low quality of evidence; Strong recommendation).

4.5 | Interventional Therapies

A randomized sham-controlled crossover trial of intrapyloric botulinum toxin (botox) injection in patients with gastroparesis found no improvement in either gastric emptying or symptoms [55]. A pilot study reported improved symptomatic response when patients were selected based on endoscopic functional luminal imaging probe measurement of pyloric distensibility [56]. Currently there is insufficient evidence to support the use of intrapyloric botox as treatment, or as a screening test for further pyloric interventions.

Gastric peroral endoscopic myotomy (G-POEM) has emerged as a minimally invasive therapeutic option to reduce pyloric resistance to gastric emptying. Initial studies suggested G-POEM significantly improves symptoms and gastric emptying in patients with refractory gastroparesis. Three non-randomized trials showed success rates of 58%–60% at 6 months [57], maintained variably from 75% at 3 years [58] to 87% at 5 years [59]. Obesity, duration of gastroparesis, psychiatric comorbidity, and opioid use have been associated with poor outcomes [60]. However only one sham-controlled RCT has been published, including 11 patients with IGP. In the G-POEM group ($n = 21$), 71% achieved a 50% reduction in symptoms and improved gastric emptying at 6 months versus 22% in the sham group; subgroup analysis was inconclusive for IGP [61]. An RCT comparing G-POEM with intrapyloric botox found no difference in clinical response or gastric emptying times [62], whereas a meta-analysis of G-POEM versus surgical pyloroplasty found them to have comparable clinical outcomes, but greater cost-effectiveness with G-POEM [63]. The mechanism behind the potential benefit of G-POEM is yet to be elucidated, given previous interventions targeting the pylorus have failed. Further longitudinal sham-controlled studies are needed to confirm the flagship study and guide patient selection.

In an RCT, gastric electrical stimulation was non-superior to placebo in IGP, with no difference in vomiting when stimulation was turned on or off in a blinded fashion [64]. A 4-month double-blind sham-controlled RCT in refractory vomiting—78% with all-cause gastroparesis, the IGP fraction unknown—found a reduction in vomiting despite no improvement in gastric emptying [65], raising the possibility of a neuromodulatory effect, which is under investigation.

Statement 20. There is insufficient evidence to recommend intrapyloric botulinum toxin injection, surgical pyloroplasty, gastric electrical stimulation, or G-POEM in medically refractory IGP. These therapies should only be trialed following multidisciplinary team consensus. (Low quality of evidence; Conditional recommendation).

5 | Conclusion

In this first Australian position statement on the assessment and management of IGP, 20 statements have been refined by consensus and given a grade of evidence and strength of recommendation based on available evidence and expert opinion. A novel treatment algorithm provides clear and relevant guidance to clinicians as a framework to support multidisciplinary care within the biopsychosocial model of disease.

Beyond traditional recommendations targeting gastric emptying alone, this working group recommends the adjunctive application of treatments established in functional gastroduodenal disorders to IGP. A key focus is to minimize iatrogenic harm. Mental health clinicians form an essential part of the treatment team. Specialist tertiary MDT input is recommended as standard of care if first-line treatment fails. When disordered eating behavior is present, a shared model of care with eating disorder clinicians is advocated. Restrictive diets, long-term tube feeding and parenteral nutrition should be avoided whenever possible. Interventional endoscopic and surgical treatment options should only be considered with formal tertiary MDT consensus.

Key areas for development have been identified as follows: (1) interdisciplinary research targeting the many mechanisms underlying symptom pathogenesis; (2) defining a combination of testing modalities to better phenotype the sensory and motor pathologies; (3) publicly available educational resources to mitigate misinformation; (4) a shift in Australian public hospital funding to support mental health clinicians as core members of the MDT.

Overall, it remains clear that IGP is under-researched and poorly understood. We call on the international community of Neurogastroenterology Societies to work together to redefine IGP by incorporating the many pathophysiological mechanisms now established, and to recognize IGP as a sensorimotor disorder. Employing this understanding will enable us to refocus research toward the development of novel targeted therapies, and ultimately improve the lives of individuals living with this challenging disorder.

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The data that support the findings of this study are available from the corresponding author upon reasonable request.

References

1. Y. Ye, Y. Yin, S. Y. Huh, C. Almansa, D. Bennett, and M. Camilleri, "Epidemiology, Etiology, and Treatment of Gastroparesis: Real-World Evidence From a Large US National Claims Database," *Gastroenterology* 162, no. 1 (2022): 109–121.e5.

2. B. E. Lacy, J. L. Wise, and D. J. Cangemi, "Gastroparesis: Time for a Paradigm Change," *Current Opinion in Gastroenterology* 39, no. 6 (2023): 503–511.

3. P. J. Pasricha, M. Grover, K. P. Yates, et al., "Functional Dyspepsia and Gastroparesis in Tertiary Care Are Interchangeable Syndromes With Common Clinical and Pathologic Features," *Gastroenterology* 160, no. 6 (2021): 2006–2017.

4. L. Wauters, R. Dickman, V. Drug, et al., "United European Gastroenterology (UEG) and European Society for Neurogastroenterology and Motility (ESNM) Consensus on Functional Dyspepsia," *United European Gastroenterology Journal* 9, no. 3 (2021): 307–331.

5. J. Schol, L. Wauters, R. Dickman, et al., "United European Gastroenterology (UEG) and European Society for Neurogastroenterology and Motility (ESNM) Consensus on Gastroparesis," *United European Gastroenterology Journal* 9, no. 3 (2021): 287–306.

6. A. Giedinghagen, "The Tic in TikTok and (Where) All Systems Go: Mass Social Media Induced Illness and Munchausen's by Internet as Explanatory Models for Social Media Associated Abnormal Illness Behavior," *Clinical Child Psychology and Psychiatry* 28, no. 1 (2023): 270–278.

7. M. Camilleri, B. Kuo, L. Nguyen, et al., "ACG Clinical Guideline: Gastroparesis," *American Journal of Gastroenterology* 117, no. 8 (2022): 1197–1220.

8. J. Schol, I. H. Huang, F. Carbone, et al., "Rome Foundation and International Neurogastroenterology and Motility Societies' Consensus on Idiopathic Gastroparesis," *Lancet Gastroenterology & Hepatology* 10, no. 1 (2025): 68–81.

9. G. Guyatt, T. Agoritsas, R. Brignardello-Petersen, et al., "Core GRADE 1: Overview of the Core GRADE Approach," *BMJ* 389 (2025): e081903.

10. P. J. Pasricha, M. Grover, K. P. Yates, et al., "Progress in Gastroparesis—A Narrative Review of the Work of the Gastroparesis Clinical Research Consortium," *Clinical Gastroenterology and Hepatology* 20, no. 12 (2022): 2684–2695.e3.

11. M. A. Schalla and A. Stengel, "Gastrointestinal Alterations in Anorexia Nervosa—A Systematic Review," *European Eating Disorders Review* 27, no. 5 (2019): 447–461.

12. V. Hanel, M. A. Schalla, and A. Stengel, "Irritable Bowel Syndrome and Functional Dyspepsia in Patients With Eating Disorders—A Systematic Review," *European Eating Disorders Review* 29, no. 5 (2021): 692–719.

13. K. Staller, S. R. Abber, and H. Burton Murray, "The Intersection Between Eating Disorders and Gastrointestinal Disorders: A Narrative Review and Practical Guide," *Lancet Gastroenterology & Hepatology* 8, no. 6 (2023): 565–578.

14. H. Mikhael-Moussa, V. Bertrand, E. Lejeune, et al., "The Association of Avoidant/Restrictive Food Intake Disorder (ARFID) and Neurogastroenterology Disorders (Including Disorders of Gut-Brain Interaction [DGBI]): A Scoping Review," *Neurogastroenterology and Motility* 37, no. 9 (2025): e70039.

15. S. Shivji, J. R. Conner, and R. Kirsch, "Mast Cell Evaluation in Gastrointestinal Biopsies: Should We Be Counting? A Critical Review and Practical Guide for the Surgical Pathologist," *Histopathology* 82, no. 7 (2023): 960–973.

16. P. Vijayvargiya, S. Jameie-Oskooei, M. Camilleri, V. Chedid, P. J. Erwin, and M. H. Murad, "Association Between Delayed Gastric Emptying and Upper Gastrointestinal Symptoms: A Systematic Review and Meta-Analysis," *Gut* 68, no. 5 (2019): 804–813.

17. G. Karamanolis, P. Caenepeel, J. Arts, and J. Tack, "Determinants of Symptom Pattern in Idiopathic Severely Delayed Gastric Emptying: Gastric Emptying Rate or Proximal Stomach Dysfunction?," *Gut* 56, no. 1 (2007): 29–36.

18. NSW Agency for Clinical Innovation, "Gastric Emptying Scintigraphy: Clinical Practice Guide," Sydney ACI 2023.

19. D. A. Carson, S. Bhat, T. C. L. Hayes, et al., "Abnormalities on Electrogastrography in Nausea and Vomiting Syndromes: A Systematic Review, Meta-Analysis, and Comparison to Other Gastric Disorders," *Digestive Diseases and Sciences* 67, no. 3 (2022): 773–785.

20. H. P. Parkman, K. P. Yates, W. L. Hasler, et al., "Dietary Intake and Nutritional Deficiencies in Patients With Diabetic or Idiopathic Gastroparesis," *Gastroenterology* 141, no. 2 (2011): 486–498.e7.

21. D. Strijbos, D. Keszthelyi, F. G. Smeets, et al., "Therapeutic Strategies in Gastroparesis: Results of Stepwise Approach With Diet and Prokinetics, Gastric Rest, and PEG-J: A Retrospective Analysis," *Neurogastroenterology and Motility* 31, no. 6 (2019): e13588.

22. B. N. Limketkai, W. LeBrett, L. Lin, and N. D. Shah, "Nutritional Approaches for Gastroparesis," *Lancet Gastroenterology & Hepatology* 5, no. 11 (2020): 1017–1026.

23. C. J. Homko, F. Duffy, F. K. Friedenberg, G. Boden, and H. P. Parkman, "Effect of Dietary Fat and Food Consistency on Gastroparesis Symptoms in Patients With Gastroparesis," *Neurogastroenterology and Motility* 27, no. 4 (2015): 501–508.

24. H. M. Staudacher, A. N. Nevin, C. Duff, B. J. Kendall, and G. J. Holtmann, "Epigastric Symptom Response to Low FODMAP Dietary Advice Compared With Standard Dietetic Advice in Individuals With Functional Dyspepsia," *Neurogastroenterology and Motility* 33, no. 11 (2021): e14148.

25. J. Serra, D. Pohl, F. Azpiroz, et al., "European Society of Neurogastroenterology and Motility Guidelines on Functional Constipation in Adults," *Neurogastroenterology and Motility* 32, no. 2 (2020): e13762.

26. H. C. Tjeerdsma, A. J. Smout, and L. M. Akkermans, "Voluntary Suppression of Defecation Delays Gastric Emptying," *Digestive Diseases and Sciences* 38, no. 5 (1993): 832–836.

27. H. P. Parkman, E. Sharkey, R. W. McCallum, et al., "Constipation in Patients With Symptoms of Gastroparesis: Analysis of Symptoms and Gastrointestinal Transit," *Clinical Gastroenterology and Hepatology* 20, no. 3 (2022): 546–558.e5.

28. P. J. Pasricha, R. Colvin, K. Yates, et al., "Characteristics of Patients With Chronic Unexplained Nausea and Vomiting and Normal Gastric Emptying," *Clinical Gastroenterology and Hepatology* 9, no. 7 (2011): 567–576.e1–4.

29. P. H. Comerlato, J. Stefani, and L. V. Viana, "Mortality and Overall and Specific Infection Complication Rates in Patients Who Receive Parenteral Nutrition: Systematic Review and Meta-Analysis With Trial Sequential Analysis," *American Journal of Clinical Nutrition* 114, no. 4 (2021): 1535–1545.

30. M. R. Ingrosso, M. Camilleri, J. Tack, G. Ianiro, C. J. Black, and A. C. Ford, "Efficacy and Safety of Drugs for Gastroparesis: Systematic Review and Network Meta-Analysis," *Gastroenterology* 164, no. 4 (2023): 642–654.

31. R. Pittayanon, Y. Yuan, N. P. Bollegala, et al., "Prokinetics for Functional Dyspepsia: A Systematic Review and Meta-Analysis of Randomized Control Trials," *American Journal of Gastroenterology* 114, no. 2 (2019): 233–243.

32. M. S. Perkel, C. Moore, T. Hersh, and E. D. Davidson, "Metoclopramide Therapy in Patients With Delayed Gastric Emptying: A Randomized, Double-Blind Study," *Digestive Diseases and Sciences* 24, no. 9 (1979): 662–666.

33. R. H. Davis, M. H. Clench, and J. R. Mathias, "Effects of Domperidone in Patients With Chronic Unexplained Upper Gastrointestinal Symptoms: A Double-Blind, Placebo-Controlled Study," *Digestive Diseases and Sciences* 33, no. 12 (1988): 1505–1511.

34. R. D. Richards, K. Davenport, and R. W. McCallum, "The Treatment of Idiopathic and Diabetic Gastroparesis With Acute Intravenous and Chronic Oral Erythromycin," *American Journal of Gastroenterology* 88, no. 2 (1993): 203–207.

35. F. Carbone, K. Van den Houte, E. Clevers, et al., "Prucalopride in Gastroparesis: A Randomized Placebo-Controlled Crossover Study," *American Journal of Gastroenterology* 114, no. 8 (2019): 1265–1274.

36. F. Bailey and A. Davies, "The Misuse/Abuse of Antihistamine Antiemetic Medication (Cyclizine) by Cancer Patients," *Palliative Medicine* 22, no. 7 (2008): 869–871.

37. C. J. Roldan, K. A. Chambers, L. Paniagua, S. Patel, M. Cardenas-Turanzas, and Y. Chathampally, "Randomized Controlled Double-Blind Trial Comparing Haloperidol Combined With Conventional Therapy to Conventional Therapy Alone in Patients With Symptomatic Gastroparesis," *Academic Emergency Medicine* 24, no. 11 (2017): 1307–1314.

38. D. Midani and H. P. Parkman, "Granisetron Transdermal System for Treatment of Symptoms of Gastroparesis: A Prescription Registry Study," *Journal of Neurogastroenterology and Motility* 22, no. 4 (2016): 650–655.

39. P. J. Pasricha, K. P. Yates, I. Sarosiek, et al., "Aprepitant Has Mixed Effects on Nausea and Reduces Other Symptoms in Patients With Gastroparesis and Related Disorders," *Gastroenterology* 154, no. 1 (2018): 65–76.e11.

40. H. P. Parkman, M. L. Van Natta, T. L. Abell, et al., "Effect of Nortriptyline on Symptoms of Idiopathic Gastroparesis: The NORIG Randomized Clinical Trial," *JAMA* 310, no. 24 (2013): 2640–2649.

41. B. E. Lacy, Y. A. Saito, M. Camilleri, et al., "Effects of Antidepressants on Gastric Function in Patients With Functional Dyspepsia," *American Journal of Gastroenterology* 113, no. 2 (2018): 216–224.

42. N. J. Talley, G. R. Locke, Y. A. Saito, et al., "Effect of Amitriptyline and Escitalopram on Functional Dyspepsia: A Multicenter, Randomized Controlled Study," *Gastroenterology* 149, no. 2 (2015): 340–349.e2.

43. M. Malamood, A. Roberts, R. Kataria, H. P. Parkman, and R. Schey, "Mirtazapine for Symptom Control in Refractory Gastroparesis," *Drug Design, Development and Therapy* 11 (2017): 1035–1041.

44. L. Cao, G. Li, J. Cao, F. Li, and W. Han, "Randomized Clinical Trial: The Effects of Mirtazapine in Functional Dyspepsia Patients," *Therapeutic Advances in Gastroenterology* 18 (2025): 17562848241311129.

45. H. P. Parkman, K. P. Yates, I. Sarosiek, et al., "Buspirone for Early Satiety and Symptoms of Gastroparesis: A Multi-Centre, Randomised, Placebo-Controlled, Double-Masked Trial (BESST)," *Alimentary Pharmacology & Therapeutics* 57, no. 11 (2023): 1272–1289.

46. D. A. Drossman, J. Tack, A. C. Ford, E. Szigethy, H. Törnblom, and L. Van Oudenhove, "Neuromodulators for Functional Gastrointestinal Disorders (Disorders of Gut-Brain Interaction): A Rome

Foundation Working Team Report," *Gastroenterology* 154, no. 4 (2018): 1140–1171.e1.

47. T. Zheng, J. BouSaba, A. Taylor, et al., "A Randomized, Controlled Trial of Efficacy and Safety of Cannabidiol in Idiopathic and Diabetic Gastroparesis," *Clinical Gastroenterology and Hepatology* 21, no. 13 (2023): 3405–3414.e4.

48. Y. Kilani, D. A. Gonzalez Mosquera, P. C. Puelo, et al., "The Impact of Cannabis Use in Gastroparesis: A Propensity-Matched Analysis of 41,374 Gastroparesis Patients," *American Journal of Gastroenterology* 121, no. 1 (2026): 248–257, https://doi.org/10.14309/ajg.0000000000003479.

49. L. Keefer, S. K. Ballou, D. A. Drossman, G. Ringstrom, S. Elsenbruch, and B. Ljótsson, "A Rome Working Team Report on Brain–Gut Behavior Therapies for Disorders of Gut–Brain Interaction," *Gastroenterology* 162, no. 1 (2022): 300–315.

50. E. Dendrinos, H. Kim, D. Hubik, et al., "Clinical Outcomes of Patients Seen by Psychiatrists in a Multidisciplinary Clinic for Disorders of Gut-Brain Interaction," *Internal Medicine Journal* 55, no. 10 (2025): 1687–1694.

51. S. Woodhouse, G. Hebbard, and S. R. Knowles, "Psychological Controversies in Gastroparesis: A Systematic Review," *World Journal of Gastroenterology* 23, no. 7 (2017): 1298–1309.

52. J. Overs, S. Morgan, P. Apputhurai, C. Tuck, and S. R. Knowles, "Comparing the Prevalence and Association Between Anxiety, Depression and Gastrointestinal Symptoms in Gastroparesis Versus Functional Dyspepsia: A Systematic Review and Meta-Analysis," *Journal of Psychosomatic Research* 183 (2024): 111834.

53. P. Apputhurai, O. S. Palsson, S. I. Bangdiwala, A. D. Sperber, A. Mikocka-Walus, and S. R. Knowles, "Confirmatory Validation of the Patient Health Questionnaire - 4 (PHQ-4) for Gastrointestinal Disorders: A Large-Scale Cross-Sectional Survey," *Journal of Psychosomatic Research* 180 (2024): 111654.

54. C. Riedlinger, G. Schmidt, A. Weiland, et al., "Which Symptoms, Complaints and Complications of the Gastrointestinal Tract Occur in Patients With Eating Disorders? A Systematic Review and Quantitative Analysis," *Frontiers in Psychiatry* 11 (2020): 195.

55. J. Arts, L. Holvoet, P. Caenepeel, et al., "Clinical Trial: A Randomized-Controlled Crossover Study of Intrapyloric Injection of Botulinum Toxin in Gastroparesis," *Alimentary Pharmacology & Therapeutics* 26, no. 9 (2007): 1251–1258.

56. C. Desprez, C. Melchior, F. Wuestenberghs, et al., "Pyloric Distensibility Measurement Predicts Symptomatic Response to Intrapyloric Botulinum Toxin Injection," *Gastrointestinal Endoscopy* 90, no. 5 (2019): 754–760.e1.

57. K. Vosoughi, Y. Ichkhanian, P. Benias, et al., "Gastric Per-Oral Endoscopic Myotomy (G-POEM) for Refractory Gastroparesis: Results From an International Prospective Trial," *Gut* 71, no. 1 (2022): 25–33.

58. A. Canakis, S. S. Deliwala, M. Frohlinger, J. P. Canakis, and D. G. Adler, "Long-Term Outcomes (≥3 Years) After Gastric Peroral Endoscopic Myotomy for Refractory Gastroparesis: A Systematic Review and Meta-Analysis," *iGIE* 2, no. 3 (2023): 344–349.e3.

59. J. Tan, H. Wu, X. Yang, et al., "A Long-Term Follow-Up Study of Gastric Peroral Endoscopic Myotomy (G-POEM) in a Large Cohort of Patients With Postsurgical Gastroparesis," *Surgical Endoscopy* 38, no. 12 (2024): 7416–7425.

60. M. M. Abdelfatah, A. Noll, N. Kapil, et al., "Long-Term Outcome of Gastric Per-Oral Endoscopic Pyloromyotomy in Treatment of Gastroparesis," *Clinical Gastroenterology and Hepatology* 19, no. 4 (2021): 816–824.

61. J. Martinek, R. Hustak, J. Mares, et al., "Endoscopic Pyloromyotomy for the Treatment of Severe and Refractory Gastroparesis: A Pilot, Randomised, Sham-Controlled Trial," *Gut* 71, no. 11 (2022): 2170–2178.

62. J. M. Gonzalez, F. Mion, M. Pioche, et al., "Gastric Peroral Endoscopic Myotomy Versus Botulinum Toxin Injection for the Treatment of Refractory Gastroparesis: Results of a Double-Blind Randomized Controlled Study," *Endoscopy* 56, no. 5 (2024): 345–352.

63. M. Aziz, M. K. Gangwani, H. Haghbin, et al., "Gastric Peroral Endoscopic Myotomy Versus Surgical Pyloromyotomy/Pyloroplasty for Refractory Gastroparesis: Systematic Review and Meta-Analysis," *Endoscopy International Open* 11, no. 4 (2023): E322–E329.

64. R. W. McCallum, I. Sarosiek, H. P. Parkman, et al., "Gastric Electrical Stimulation With Enterra Therapy Improves Symptoms of Idiopathic Gastroparesis," *Neurogastroenterology and Motility* 25, no. 10 (2013): 815–e636.

65. P. Ducrotte, B. Coffin, B. Bonaz, et al., "Gastric Electrical Stimulation Reduces Refractory Vomiting in a Randomized Crossover Trial," *Gastroenterology* 158, no. 3 (2020): 506–514.e2.

66. E. A. Olausson, S. Störsrud, H. Grundin, M. Isaksson, S. Attvall, and M. Simrén, "A Small Particle Size Diet Reduces Upper Gastrointestinal Symptoms in Patients With Diabetic Gastroparesis: A Randomized Controlled Trial," *American Journal of Gastroenterology* 109, no. 3 (2014): 375–385.

67. O. Goyal, S. Nohria, S. Batta, A. Dhaliwal, P. Goyal, and A. Sood, "Low Fermentable Oligosaccharides, Disaccharides, Monosaccharides, and Polyols Diet Versus Traditional Dietary Advice for Functional Dyspepsia: A Randomized Controlled Trial," *Journal of Gastroenterology and Hepatology* 37, no. 2 (2022): 301–309.

68. D. So, P. R. Gibson, J. G. Muir, and C. K. Yao, "Dietary Fibres and IBS: Translating Functional Characteristics to Clinical Value in the Era of Personalized Medicine," *Gut* 70, no. 12 (2021): 2383–2394.

69. H. Suresh, J. Zhou, and V. Ho, "The Short-Term Effects and Tolerability of Low-Viscosity Soluble Fibre on Gastroparesis Patients: A Pilot Clinical Intervention Study," *Nutrients* 13, no. 12 (2021): 4298.

70. R. Gallo, E. Armstrong, H. Griffin, D. Russell, and G. Hebbard, "Impact of Enteral Tube Feeding for Individuals With Disorders of the Gut Brain Interaction," *Clinical Nutrition ESPEN* 58 (2023): P604.

71. L. D. Martin, X. Wang, C. Day, et al., "Enteral Tube Feeding in Functional Gastrointestinal Disorders: A Game of Chance?," *Clinical Nutrition ESPEN* 57 (2023): P849.

Appendix 1

TABLE A1 | Meal plan summary: soft, small-particle diet.

Meal	Food	Nutrients
Breakfast	Quick oats (1/2 cup, dry) Low-fat milk (3/4 cup) Mashed banana (1/2 banana) Nut spread (2 tsp)	1.5 MJ energy 15 g protein 45 g carbohydrates 12 g fat 6 g fiber
Morning tea	Greek yoghurt (1/2 cup, regular fat) Stewed apples (1/2 cup)	1.1 MJ energy 9 g protein 39 g carbohydrates 7 g fat 2 g fiber
Lunch	Lean minced beef (3/4 cup, cooked) Mashed potatoes (1/2 cup) Mashed pumpkin (1/4 cup) Mashed carrots (1/4 cup) Gravy (1 tbsp)	2.0 MJ energy 35 g protein 20 g carbohydrates 28 g fat 5 g fiber
Afternoon tea	Wholemeal bread (1 slice) Hummus (2 tbsp)	0.4 MJ energy 4.5 g protein 14 g carbohydrates 2 g fat 3 g fiber
Dinner	Baked white fish (85 g) Mashed sweet potato (3/4 cup) Mashed broccoli (1/2 cup) Olive oil (1 tsp)	1.44 MJ energy 25 g protein 37 g carbohydrates 6 g fat 7 g fiber
Supper	Greek yoghurt (1/2 cup, plain, 2%) Peaches in juice (1/2 cup)	0.9 MJ energy 7 g protein 38 g carbohydrates 6 g fat 1 g fiber

Note: **Nutritional analysis:** energy: 7.3 MJ; protein: 97 g (22.6% of energy); carbohydrates: 188 g (42.71% of energy); fat: 60 g (30% of energy); fiber: 24 g; iron: 11 mg; vitamin B12: 7 mg; zinc: 14.0 mg; folate: 399 μg; vitamin C: 139 mg; calcium: 1013 mg.

TABLE A2 | Meal plan summary: texture-modified diet that includes liquids.

Meal	Foods	Nutrients
Breakfast	Scrambled eggs (2 eggs) Wholemeal bread (1 slice)	1.3 MJ energy 18 g protein 15 g carbohydrates 19 g fat 2 g fiber
Morning tea	Smoothie: Skim milk powder (10 g) Mashed banana (1/2 banana) Greek yoghurt (1/2 cup, plain, 2%) Low-fat milk (1/2 cup)	1.1 MJ energy 20 g protein 34 g carbohydrates 5 g fat 4 g fiber
Lunch	Minced meat (1/3 cup, cooked) Mashed potatoes (1/2 cup) Mashed pumpkin (1/4 cup) Mashed carrots (1/4 cup)	1.5 MJ energy 17 g protein 20 g carbohydrates 22 g fat 4 g fiber

Meal	Foods	Nutrients
Afternoon tea	Sustagen (250 mL)	0.9 kJ energy 13 g protein 30 g carbohydrates 5 g fat 0 g fiber
Dinner	Puree soup of: Mashed sweet potato (3/4 cup) Mashed broccoli (1/2 cup) Olive oil (1 tsp) Puréed chicken (1/4 cup) Puréed spinach (1/4 cup)	1.7 MJ energy 25 g protein 27 g carbohydrates 21 g fat 8 g fiber
Supper	Puréed fruit (3/4 cup)	0.7 MJ energy 6 g protein 35 g carbohydrates 1 g fat 6 g fiber

Note: **Nutritional analysis:** energy: 7.2 MJ; protein: 92 g (22% of energy); carbohydrates: 160 g (44% of energy); fat: 74 g (37% of energy); fiber: 24 g; iron: 15 mg; vitamin B12: 6.7 μg; zinc: 10 mg; folate: 221 μg; vitamin C: 99 mg; calcium: 1151 mg.

Appendix 2

Summary of Dietary Approaches for Gastroparesis

Dietary therapy	Key features	Limitations
Small food particle size	Food mechanically altered to reduce particle size	• Evidence primarily from patients with diabetic gastroparesis [66]; not a crossover design study, limiting strength of findings; glycaemic control improvements were not monitored • Definition of "small particle size" inconsistent between studies (e.g., rice excluded despite having small particle size) [22]
Low-FODMAP diet	Restricts fermentable carbohydrates	• Evidence from functional dyspepsia but not specifically gastroparesis [24, 67]; however, high symptom overlap • No evidence for improving gastric emptying • Contraindicated in malnourished patients
Fiber modification	Selective use of fibers (PHGG, psyllium)	• Paradoxical effects: may slow gastric emptying but reduce symptoms [68] • Baseline fiber intake usually already low in patients with gastroparesis [20] • PHGG shown to improve irritable bowel syndrome symptoms, specifically bloating and pain [69]

Abbreviations: FODMAP = fermentable oligosaccharides, disaccharides, monosaccharides and polyols; PHGG = partially hydrolysed guar gum.

Appendix 3

Nutritional Outcomes of Enteral Feeding in Gastroparesis: Evidence Summary

Author, year, location	Population and study design	Outcomes
Gallo, 2023, Australia (abstract only) [70]	Disorders of gut–brain interaction ($n = 15$) Retrospective	Six patients (40%) experienced weight gain after tube insertion, six (40%) had no weight change and three (20%) experienced weight loss.
Martin, 2023, United Kingdom (abstract only) [71]	Disorders of gut–brain interaction ($n = 15$) Retrospective	Eight of 15 patients continued long-term enteral feeding (median, 4.3 years), although three (of six at admission) remained underweight (BMI < 18.5 kg/m^2). Seven of 15 patients discontinued enteral feeding after a median of 0.3 years (IQR, 0–1.5 years) and one patient (of three at admission) remained underweight.

Author, year, location	Population and study design	Outcomes
Strijbos, 2019, Netherlands [21]	Gastroparesis ($n = 86$) Diabetes 26%, postsurgical 27%, idiopathic 38%, generalized motility disorder 8% Retrospective	Of 86 patients, 36 commenced 3 months of nasoduodenal enteral feeding after not responding to diet and prokinetic therapy. Weight gain occurred regardless of symptomatic improvement (17/36 were symptomatic responders, gaining a mean of 2.5 kg [$p = 0.018$] from baseline, compared with 19/36 whose symptoms did not respond and who gained 2.1 kg [$p = 0.027$]) For the 19 patients who did not achieve symptomatic improvement with nasoduodenal enteral feeds, PEG-J was instituted. After 6 months of PEG-J feeding, a mean weight gain of 5.1 kg (range, −5 to +21 kg, $p = 0.002$) was observed; this did not differ between those whose symptoms responded to PEG-J and those who did not.

Abbreviations: BMI = body mass index; IQR = interquartile range; PEG-J = percutaneous endoscopic gastrostomy with jejunal extension.