GESA
Gastroenterological Society of Australia

Gastroenterological Society of Australia position statement on the assessment and management of idiopathic gastroparesis

ANGMA

anzaed
AUSTRALIA & NEW ZEALAND ACADEMY FOR EATING DISORDERS

AuSPEN

New Zealand Society of Gastroenterology Inc

ISBN: 978-1-7640409-2-1

First published online November 2025.

Disclaimer

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. The compilers of this position statement shall not be liable for any loss, direct, indirect or consequential, on whatsoever account for any omission or negligent misstatement contained herein, or by reason of, arising from or in relation to any such user, by any other person, company or body relying or acting upon or purporting to rely or act upon any matter contained therein or arising thereout.

Suggested citation

Kellar T, Basnayake C, Biesiekierski J, et al. Gastroenterological Society of Australia position statement on the assessment and management of idiopathic gastroparesis. Melbourne: Gastroenterological Society of Australia, 2025.

Contact

Gastroenterological Society of Australia
Level 3, 517 Flinders Lane
Melbourne VIC 3000 Australia
Phone: 1300 766 176
Website: www.gesa.org.au
Email: gesa@gesa.org.au

Working Group

Trina Kellar (Chair),[1,2] Chamara Basnayake,[3,4] Jessica Biesiekierski,[5] Rebecca Burgell,[6,7] Jessica Fitzpatrick,[6,7] Geoffrey Hebbard,[8,3] Vincent Ho,[9] Hannah Kim,[8,10] Simon R Knowles,[11] Christopher K Rayner,[12] May Wong,[13] Nicholas J Talley[14,15]

[1] School of Health, University of Queensland, Brisbane, Australia
[2] Department of Gastroenterology, Royal Brisbane and Women's Hospital, Brisbane, Australia
[3] Department of Medicine, University of Melbourne, Melbourne, Australia
[4] Department of Gastroenterology, St Vincent's Hospital Melbourne, Melbourne, Australia
[5] School of Agriculture, Food and Ecosystem Sciences, University of Melbourne, Melbourne, Australia
[6] Department of Gastroenterology and Nutrition, Monash University, Melbourne, Australia
[7] Department of Gastroenterology, Alfred Hospital, Melbourne, Australia
[8] Department of Gastroenterology, Royal Melbourne Hospital, Melbourne, Australia
[9] School of Medicine, Western Sydney University, Sydney, Australia
[10] Parkville Youth Mental Health and Wellbeing Service, Melbourne, Australia
[11] Department of Psychological Sciences, Swinburne University, Melbourne, Australia
[12] Faculty of Health and Medical Sciences, University of Adelaide, Adelaide, Australia
[13] Department of Gastroenterology, Royal North Shore Hospital, Sydney, Australia
[14] Hunter Medical Research Institute, University of Newcastle, Newcastle, Australia
[15] Department of Gastroenterology, John Hunter Hospital, Newcastle, Australia

Abstract

Idiopathic gastroparesis (IGP) treatment guidelines have to date focused on delayed gastric emptying as the cause of this disorder's associated symptoms of postprandial nausea, vomiting, early satiety, fullness and pain. The efficacy of treatments targeting gastric emptying is low, and treatment outcomes are poor, resulting in substantial impacts on personal and socioeconomic health. Recent advances in understanding the pathophysiology underlying symptom genesis in IGP have shown this disorder to be much more complex than delayed gastric emptying, with abnormalities in gastric accommodation, contractility, arrhythmias, pyloric dysfunction, downstream dysmotility and, notably, visceral hypersensitivity. Gastric emptying time on scintigraphy, which is the current gold-standard test for defining IGP, correlates poorly with symptoms of gastroparesis and varies in an individual over time. This, along with a diagnostic overlap with functional gastroduodenal disorders, has challenged the currently accepted fundamental diagnostic and treatment principles for IGP. Here, we provide the first Australian clinical guidance document for idiopathic gastroparesis, with a call to redefine it as a sensorimotor disorder. Twenty consensus statements are provided, based on available evidence and multidisciplinary expert consensus. This position statement aims to assist clinicians across Australia to improve consistency of care, minimise harm and improve quality of life for all patients living with this challenging disorder.

Table of Contents

1 Introduction

1.1 Scope and purpose

Gastroparesis has historically been defined as a condition presenting with the cardinal upper gastrointestinal symptoms of postprandial nausea, vomiting, early satiety, fullness and bloating, with delayed gastric emptying, in the absence of mechanical obstruction. The common subtypes are idiopathic, diabetic and postsurgical gastroparesis. This position statement refers specifically to the idiopathic subtype, where no cause can be identified using traditional diagnostic techniques. Global epidemiological data for idiopathic gastroparesis (IGP) are lacking, and the population prevalence of asymptomatic delayed gastric emptying is unknown.[1] Although considered a rare disease, IGP appears to be increasing in Western populations.[2,3] When IGP is severe, its individual and socioeconomic impacts are high due to loss of quality of life and productivity.[4,5] There have been few recent therapeutic developments, and available treatments targeting gastric emptying are often ineffective. This in part reflects the historic classification of IGP as primarily a motor disorder, with symptoms attributed to delayed gastric emptying. This preconception has carved a deep bias in study design, interpretation and therapeutic pursuits.[6]

There is now increasing acceptance that IGP is a sensorimotor disease on a spectrum with functional gastroduodenal disorders, a concept advocated by leaders in the field since the 1990s.[7-13] Functional dyspepsia and gastroparesis have been shown to be clinically indistinguishable.[14] There is also substantial overlap between gastroparesis and other functional gastroduodenal disorders 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[15,16] — this distinction may lead to ongoing limitations in research and suboptimal clinical care.

This shift in concept is timely. In Western societies, presentations with gastroparesis-like disorders 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 artificial nutrition support and invasive treatment modalities for IGP, carrying high iatrogenic risk to the individual, as well as economic cost to health care systems. 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 behaviour is substantial.[17,18]

International guidelines from European[16] and North American societies[19] and the Rome Foundation[20] acknowledge the challenges surrounding IGP, including our limited understanding of the pathophysiology underlying symptoms, poor correlation of symptoms with gastric emptying, presence of overlapping clinical phenotypes and lack of effective therapies. Despite this recognition, the international consensus group recently convened by the Rome Foundation maintained the historic focus on IGP as a motor disorder and was unable to establish consensus on most consensus statements.

The existing international guidelines provide an extensive summary of the literature to date,[16,19,20] which we will not reiterate here. Rather, following review of the literature, our working group aimed to provide guidance that is highly clinically applicable, with clear consensus on testing and treatment recommendations.

Accordingly, here, we present the first Australian position statement on the assessment and management of idiopathic gastroparesis. As a sensorimotor disorder, the recommendations incorporate multidisciplinary treatment approaches for both gastroparesis and overlapping functional gastroduodenal disorders, where appropriate, using locally available therapies. This national position statement aims to support all clinicians to improve the lives of patients living with this disorder.

1.2 Working group and external review

The decision to develop this position statement arose from the Gastroenterological Society of Australia (GESA) Luminal Faculty Committee meeting in October 2024. Interest in providing a national standard of care was expressed, with the aim of improving consistency of practice and treatment outcomes and minimising harm across public and private health care institutions in Australia. A core working group of eight members and a chair were elected from the Luminal Faculty in December 2024. Invitations to join the working group were then sent to clinicians from multiple disciplines with expertise in IGP nationwide, aiming for differing viewpoints and representation from each Australian state and territory. All positive responses were accepted, with 12 final working group members representing the fields of neurogastroenterology, nutrition, psychology and psychiatry.

Sections were allocated to authors in their field of expertise. The concept was presented to the current GESA Luminal Faculty patient advocacy group at an online meeting in August 2025, and the group elected one member with lived experience to represent the patient experience in the development of this document.

External review of the initial draft of this position statement was sought from a broad range of experts, both locally and internationally, in nuclear medicine, surgery, psychiatry and dietetics, including clinicians with expertise in eating disorders, general gastroenterology, neurogastroenterology, intestinal failure and paediatric gastroenterology (see Acknowledgements). The working group incorporated feedback through multiple revisions before drafting final statements for the consensus development process (see section 2.2). The document was presented at the World Congress of Gastroenterology@Australian Gastroenterology Week 2025 in Melbourne in September 2025 for public comment, before finalisation.

1.3 Declaration of funding

GESA provided pre-approved financial support for project coordination, graphic design and editorial support. The funding body did not influence the content of the position statement.

1.4 Competing interests

The working group members declare no potential conflicts of interest relevant to the preparation or content of this document.

1.5 Disclaimer

This document was written with the intention of providing clinical guidance to clinicians managing adult patients with IGP in Australia. Clinical decision making must be determined by the individual circumstances of each patient and is the responsibility of the treating clinician/s. We acknowledge the limitations in accessing tertiary health care support for many clinicians practising in regional Australia, and we hope that this document may provide a framework for recommended practice even where resources are limited. As scientific advances occur in the assessment and management of IGP, this document will be updated to reflect current practice.

1.6 Endorsements

Expert review and endorsement of the document were obtained from the following groups:

- GESA Luminal Faculty Committee
- Australasian Neurogastroenterology and Motility Association (ANGMA)
- Australasian Society of Parenteral and Enteral Nutrition (AuSPEN)
- Australia & New Zealand Academy for Eating Disorders (ANZAED)
- New Zealand Society of Gastroenterology (NZSG)

2 Methods

2.1 Grading of evidence and strength of recommendations

Section authors undertook a formal review of the literature using MEDLINE, EMBASE, PubMed, CINAHL and PsycINFO databases, along with hand-searching of references. University research librarian support was used for the development of PICO (patient, intervention, comparison, outcome) questions and search strategies at the discretion of each author. Inclusion criteria were peer-reviewed articles reporting studies in adults, published in English between January 1985 and January 2025.

The Grading of Recommendations, Assessment, Development and Evaluation (GRADE) process was applied to the final 20 statements using a standard template, to assess the quality of evidence as high, moderate, low or very low,[21-27] and the strength of recommendation as strong or conditional.[28] The quality of evidence was deemed not applicable for statements considered good practice points by consensus, where a literature search was not relevant. Where the quality of supporting evidence was found to be low or very low, this highlights a lack of available high-quality research rather than necessarily a refutation of the statement. On balance, a strong recommendation may still be appropriate when there is expert consensus for poorly researched questions. The references included in this document to support discussion points represent a selection of key articles from the literature reviews.

2.2 Modified Delphi approach

A modified Delphi approach was applied to the 20 draft statements in September 2025, using anonymous online voting among the working group, administered by the GESA project support officer. A four-point Likert scale (strongly agree, agree, disagree, strongly disagree) was used to indicate agreement, and respondents were given the opportunity to provide comments. Consensus was deemed to be reached when there was ≥85% agreement or strong agreement in one round of voting, whereas 80%–84% agreement or strong agreement was deemed borderline endorsement. No major revisions to statements or grading were required, and no statements were removed. Comments were incorporated into points of disagreement, with reference to the Appraisal of Guidelines for Research and Evaluation (AGREE) reporting checklist.[29] The final 20 statements are presented in Table 1.

3 Summary of statements

Table 1. Summary of consensus statements

No.	Statement	Endorsed	Quality of evidence*	Strength of recommendation*	Agreement†
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 specialising in eating disorders is recommended for all patients with disordered eating behaviour, 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 standardised low-fat egg-based meal or a validated variant, with greater than 10% gastric retention at 4 hours 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 prioritise oral nutritional rehabilitation, with the aim of improving symptoms where possible, while not compromising nutritional status.	Yes	Low	Strong	**100%** SA: 83% A: 17%
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%

No.	Statement	Endorsed	Quality of evidence*	Strength of recommendation*	Agreement†
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, while 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%
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 trialled following multidisciplinary team consensus.	Yes	Low	Conditional	**92%** SA: 50% A: 42% D: 8%

A = agree; D = disagree; NA = not applicable; SA = strongly agree.
* 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.
† 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.

4 Pathophysiology

Although international guidelines describe gastroparesis as a motor disorder characterised by delayed gastric emptying, it is clear that its pathophysiology is much more complex. Abnormal gastric accommodation and contractility, gastric arrhythmias, pyloric dysfunction, small bowel dysmotility and visceral hypersensitivity have all been documented in IGP,[30] highlighting that it is better understood as a sensorimotor disorder.

Consistent with this, the correlation between delayed gastric emptying and symptom severity is poor, and treatment strategies targeting motility provide inconsistent clinical benefits. Moreover, functional dyspepsia and gastroparesis have been shown to be indistinguishable on clinical grounds and histopathology,[14] raising the question of whether delayed gastric emptying is a defining, or merely an associated, feature. Gastric electrical stimulation, which emerged as a treatment for gastroparesis, ameliorates vomiting symptoms unrelated to gastric emptying, implying that symptomatic improvement results from modulation of neural sensory pathways, not gastric emptying.[31] Successful medical therapy for IGP is associated with normalisation of electrogastrography, indicating the relevance of gastric dysrhythmias.[32-34] The latter appear linked to reduced numbers of interstitial cells of Cajal[35] and fibrosis on full-thickness gastric biopsy specimens,[36] suggesting a distinct underlying pathological abnormality in a subset of patients, although sample sizes were small and population-wide reference ranges are lacking.

Impaired gastric fundic accommodation — in which the ability of the proximal stomach to act as a reservoir for ingested food is impaired, leading to abnormal redistribution of food from the proximal to distal stomach — is often present in patients with IGP.[37] A computed tomography gastric volumetry study comparing patients with gastroparesis and patients with gastro-oesophageal reflux disease indicated that gastroparesis was associated with reduced gastric volume after gaseous distension, while patients with gastroparesis rated abdominal pain more intensely, suggesting that reduced fundal accommodation could be linked to visceral hypersensitivity in gastroparesis.[38] A substantial proportion of patients with gastroparesis exhibit abnormal fundic accommodation in barostat studies.[39] Gastric scintigraphy and the nutrient drink test are more applicable investigations in clinical practice, but their ability to identify individuals with impaired fundic accommodation is limited.[40,41]

Visceral hypersensitivity, including sensitivity to nutrients, is increasingly recognised in the pathogenesis of symptoms of IGP, and abdominal pain is a feature in 30% of refractory cases.[39] Our understanding of the neuroimmune mechanisms contributing to symptom pathogenesis in disorders of gut–brain interaction (DGBI), including functional dyspepsia, is rapidly evolving and likely to be relevant to IGP. Complex immunological pathways underlie peripheral pain sensitisation, a hallmark of chronic visceral pain.[42-44]

The recognition of gastroparesis as a sensorimotor disorder has important clinical implications (see section 6). Patients with IGP may benefit from a combination of pharmacological, dietary and psychological treatments that target both motor and sensory aspects of symptom genesis.

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

5 Assessment

5.1 Overview

Suspected IGP requires a comprehensive biopsychosocial assessment, given the limited pathophysiological information provided by available testing modalities and the potential for disease overlap.

5.2 Clinical assessment

A comprehensive medical, nutritional and psychosocial history is essential. Screening questionnaires are not intended to be used as diagnostic tools but may be helpful to monitor progress.[45] Potential adverse effects of all recent prescription and non-prescription medications should be reviewed. This is particularly so for opioid, anticholinergic, antimuscarinic, antispasmodic, antipsychotic and centrally acting agents, weight loss agents, cannabinoids and illicit substances, as these may alter gastric emptying or exacerbate symptoms. The limited utility of motility testing should be discussed and documented if a patient is unable to cease any of these agents, and persistent pain or addiction specialist support should be considered if appropriate.

Time should be allowed to explore past and present psychological and neurodevelopmental comorbidities and perpetuating factors in the biopsychosocial model, including persistent pain, functional disorders and adverse life events. This may be performed by the clinician or a mental health team member. Trauma-informed, neurodiversity-affirming care and patient–doctor confidentiality are essential and provide an opportunity to build trust, dispel stigma and correct misinformation. Training is available in advanced communication skills, and formal supervision from a mental health clinician is available for clinicians.

Eating disorders are characterised by a disturbance of eating or related behaviour that results in significant impairment in physical health or psychosocial functioning. Disordered eating, as defined by the National Eating Disorders Collaboration, includes symptoms and behaviour of eating disorders, but at a lesser frequency or lower severity. Here, we use the term “disordered eating behaviour” to encompass symptoms that may be related to either disordered eating or an eating disorder. All patients require formal assessment for nutritional adequacy and disordered eating behaviour (see section 6.3). Eating disorders, DGBI and delayed gastric emptying are not independent diagnoses and frequently coexist. For example, 20%–80% of patients with an eating disorder have delayed gastric emptying,[46-49] whereas 95%–98% experience functional gastrointestinal symptoms.[50,51] Our understanding of the overlap between avoidant restrictive food intake disorder, other restrictive eating disorders and DGBI with restricted oral intake is evolving.[52] A recent meta-analysis of studies that used screening tools found evidence of disordered eating in a third of patients with IGP but emphasised that the tools are prone to overestimating eating disorders in people with gastrointestinal disorders.[53] Given this complexity, co-assessment by a clinician with expertise in eating disorders is strongly recommended for all patients with disordered eating behaviour.

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 specialising in eating disorders is recommended for all patients with disordered eating behaviour, due to the high comorbid prevalence of disordered eating and eating disorders. (Low quality of evidence; Strong recommendation)

5.3 Initial investigations

Initial work-up should exclude structural gastrointestinal abnormalities, including mechanical gastric outlet obstruction, and systemic diseases that are relevant in the clinical context, including the patient's personal and family history. Alarm features warrant urgent consideration of upper gastrointestinal endoscopy and cross-sectional imaging. Biopsies for gastroduodenal eosinophils and mast cells are not currently recommended, as clinically relevant reference intervals have not been established.[54]

Basic blood tests should include measurement of haemoglobin, electrolyte and blood glucose levels, coeliac serology, thyroid function tests, fasting haematinic tests and a macro- and micronutrient screen.

The "test and treat" strategy for *Helicobacter pylori* eradication is recommended in functional dyspepsia guidelines, depending on local epidemiology. There are no studies of this approach in patients with IGP but, given its overlap with functional dyspepsia, we support an individualised test and treat approach to *H. pylori* infection, following discussion of the limited treatment utility.

Radiological investigations may include small bowel and biliary tract imaging for pain-predominant presentations, and central nervous system imaging for persistent unexplained nausea or focal neurological features.

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)

5.4 Measurement of gastric emptying

By definition, the diagnosis of IGP requires measurement of gastric emptying. However, even in the highest-quality studies, the correlation between symptoms and delayed gastric emptying is weak,[55] with high individual variability over time irrespective of symptoms.[14] This reflects the complexity of the sensorimotor abnormalities responsible for symptoms in IGP (see section 4). Indeed, the role of measuring gastric emptying in patients with typical symptoms has been questioned.[11]

Despite these limitations, international guidelines recommend 4-hour gastric emptying scintigraphy using a standardised egg white-based low-fat meal, with greater than 10% retention at 4 hours deemed abnormal.[19,20] The percentage retention cannot be used to phenotype patients or predict treatment response.[8,39] Breath tests using ^{13}C stable isotopes are an alternative method.

In Australia, comparability of gastric emptying measurements is hampered by the heterogeneity of meals and measurement protocols in use, with specified reference ranges relying on published values for validated meals or local normative values.[56] The egg white-based meal is most widely used, but variants may be offered based on patient factors, such as allergy or cultural preferences, if validated reference ranges are available. Higher-calorie mixed-composition solid meals have been recommended as more physiological[20] but are not commercially available in Australia. We acknowledge the importance of advocating for one standardised test meal and protocol in Australia but agree that this is not possible in the absence of a commercially available test meal.

Gastric emptying time is highly variable within individuals over time and is affected by many factors (see section 5.2). A large prospective study showed that 42% of people diagnosed with

gastroparesis had normal gastric emptying when retested at 48 weeks, without a change in symptoms.[14] Where modifiable factors are present, we suggest (with borderline consensus) considering repeating a gastric emptying study within 3–12 months of an abnormal result, after all confounding factors have been optimised, to improve validity. In the case of discordant results, the better result should be accepted as representative of the stomach's capacity to empty normally. Disagreement among the working group on this point arose from the fundamental limitations of gastric emptying as a measure; given it reflects only one aspect of IGP pathophysiology and correlates poorly with symptoms, it was felt that the validity could not be justifiably improved by repeating a poor test. Ultimately, this statement highlights the importance of addressing all potentially confounding factors before performing a gastric emptying study, with any residual confounding factors being documented in the radiology report or clinician correspondence for future reference, to ensure interpretation of the test result in the clinical context.

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.

Given our understanding of IGP as a sensorimotor disorder, tests of gastroduodenal sensorimotor function may be key to routine standard assessment in the future, but they remain research tools 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.[57] A combination of these modalities is likely to provide more accurate assessment, given the breadth of the underlying pathophysiology. This is a key area for future development.

Statement 5
The rate of gastric emptying correlates poorly with symptoms and measures 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. (Moderate quality of evidence; Strong recommendation)

Statement 6
The recommended nuclear scintigraphy test should include a standardised low-fat egg-based meal or a validated variant, with greater than 10% gastric retention at 4 hours 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 optimisation of all reversible factors, to improve validity. (Very low quality of evidence; Conditional recommendation; borderline endorsement)

5.5 Further investigations

There is increasing public concern regarding vascular compression syndromes as a potential cause of gastroparesis-like symptoms. Rarely, acquired superior mesenteric artery syndrome can occur secondary to severe weight loss from any cause, manifesting as duodenal obstruction by acute angulation between the superior mesenteric artery and aorta, seen on dedicated imaging.[58] It is important to note that an asymptomatic reduced vascular angle is prevalent in population-wide radiological studies.[59] Non-invasive weight restoration is the recommended first-line treatment, with follow-up imaging if there is ongoing concern. Median arcuate ligament syndrome, involving coeliac artery compression causing chronic foregut ischaemia, has not been studied specifically in relation to gastroparesis but also has a high

asymptomatic radiological prevalence.[60,61] Routine assessment for vascular compression syndromes is not recommended for people with symptoms of IGP.

Despite a sharp increase in diagnoses of hypermobility spectrum disorders in Western populations, there is to date no evidence of a causal link between hypermobility syndromes, such as Ehlers–Danlos syndrome variants, and gastrointestinal dysmotility. Routine screening for these disorders is not recommended.

There is insufficient evidence to support routine testing for mast cell activation syndrome, autonomic dysfunction, small intestinal bacterial overgrowth or microbial dysbiosis. Subspeciality input is required if any of these disorders is 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)

6 Management

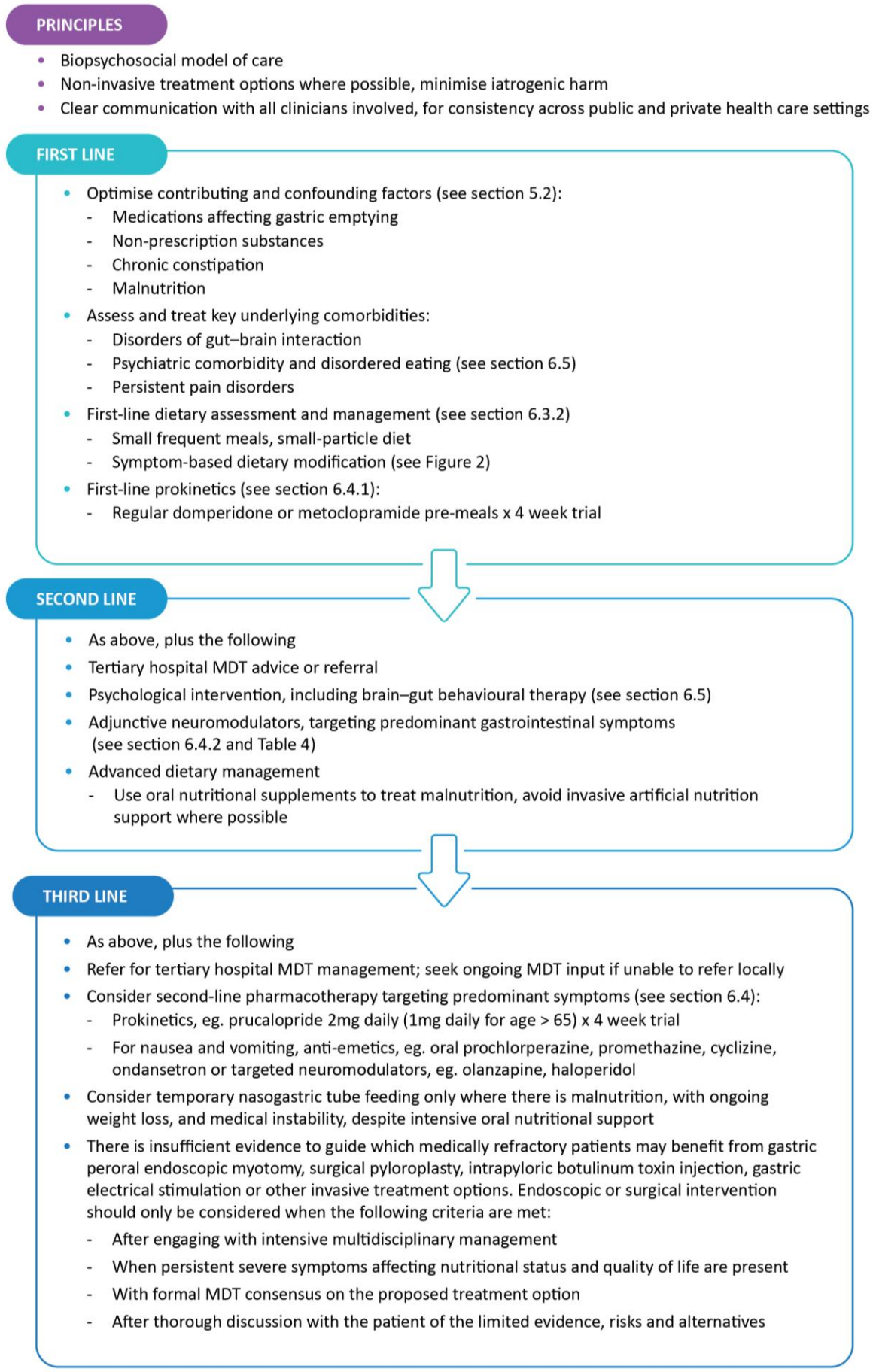
6.1 Overview

Consistent with the poor correlation between delayed gastric emptying and symptoms of gastroparesis, all available treatments that accelerate gastric emptying have shown low efficacy. Acknowledging IGP as being a sensorimotor disorder on a spectrum with functional gastroduodenal disorders endorses the adjunctive use of DGBI treatments to target its key symptoms, such as nausea or pain. The historic divide of gastroparesis from functional gastroduodenal disorders based on gastric emptying has meant that very few treatments targeting visceral hypersensitivity have been studied specifically in the context of IGP, making the available evidence for these treatments unavoidably low quality. Therefore, while awaiting advances in the understanding of symptom genesis in IGP, many of the following recommendations are extrapolated from the key overlapping disorders, where appropriate.

6.2 Biopsychosocial model of care

IGP should be managed within the biopsychosocial model of care, as outlined in the treatment algorithm in **Figure 1**. If first-line treatment is unsuccessful, advice or referral to a tertiary multidisciplinary team (MDT) is recommended. The core members of the MDT should include representatives from gastroenterology, dietetics, psychology and psychiatry, with expertise in neurogastroenterology. Additional input from eating disorder, pain, surgical and other medical subspecialties should be available to the MDT as needed. In regions or health care settings where a tertiary MDT is not available, primary clinicians should seek formal advice from the nearest expert centre with an MDT. In particular, formal MDT input should be sought before initiating long-term enteral tube feeding (ETF) or interventional therapies. Core treatment principles include minimising iatrogenic harm by using the least invasive investigation and management possible and engaging with key providers outside the MDT for consistency of care across public and private health care settings.

Figure 1. Treatment algorithm for idiopathic gastroparesis



MDT = multidisciplinary team.

6.3 Nutritional management

6.3.1 Nutritional assessment and monitoring

Gastroparesis presents unique nutritional challenges that require a dedicated MDT approach. All patients with gastroparesis should undergo a comprehensive nutritional assessment by a gastrointestinal dietitian at diagnosis and at regular intervals as clinically indicated thereafter. The nutritional assessment should include evaluation of current intake of macro- and micronutrients, eating behaviour and patterns, body image, food beliefs, previous dietary interventions and related quality of life.[62] 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%).[62] When disordered eating behaviour is identified during assessment, co-management with an eating disorder service is recommended. Disordered eating behaviour may pre-date IGP or may develop as an attempt to minimise the symptoms of IGP, or it may co-develop in a bidirectional manner. Efficacy of interventions should be assessed using validated tools, including body composition measurements, micronutrient assessments, symptom scores, eating disorder screening tools and food-related quality of life measures.

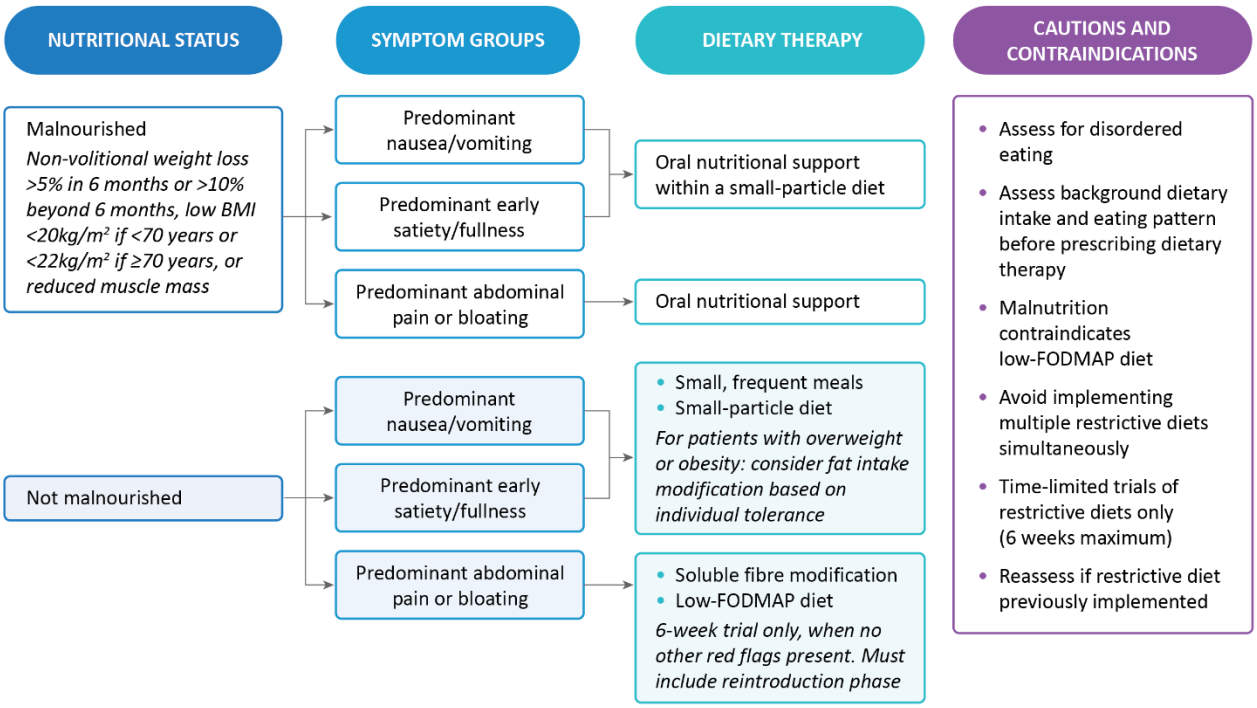
Statement 9
All patients with gastroparesis should undergo a comprehensive nutritional assessment by a gastrointestinal dietitian at diagnosis and as clinically needed thereafter. (Low quality of evidence; Strong recommendation)

6.3.2 Dietary interventions

The majority of patients with IGP can and should be managed with oral nutritional rehabilitation. About 58% of patients respond symptomatically to dietary therapy combined with prokinetic medication.[63] Dietary therapy should be prescribed by a gastrointestinal dietitian and may be tailored to the patient's symptoms (**Figure 2**), although meeting nutritional requirements remains the priority of nutritional support.[19]

Treatment planning must consider the overlap between functional dyspepsia and gastroparesis symptoms, background dietary patterns and disordered eating behaviour. **Figure 2** provides a suggested decision-making framework for selecting appropriate dietary interventions based on nutritional status and predominant symptoms. Various dietary approaches have been studied in patients with gastroparesis, with varying levels of evidence (**Table 2**). Detailed sample meal plans for each dietary approach are provided in **Appendix 1** to guide clinicians in practical implementation.

Figure 2. Decision framework for nutritional recommendations for patients with gastroparesis



This framework guides individualised nutritional management based on a patient's 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.

Table 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[64]; 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)[65]
Low-FODMAP diet	Restricts fermentable carbohydrates	- Evidence from functional dyspepsia but not specifically gastroparesis[66,67]; however, high symptom overlap - No evidence for improving gastric emptying - Contraindicated in malnourished patients
Fibre modification	Selective use of fibres (PHGG, psyllium)	- Paradoxical effects: may slow gastric emptying but reduce symptoms[68] - Baseline fibre intake usually already low in patients with gastroparesis[69] - PHGG shown to improve irritable bowel syndrome symptoms, specifically bloating and pain[70]

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

Generally, patients should consume smaller and more frequent meals (6–10 meals daily),[63,65,71] ensuring food is well-chewed or blended,[71] and they should remain upright for at least 1–2 hours after eating.[63,72] These practical approaches complement individualised dietary modifications and are part of a strategy termed "effortful eating".[73] Although low-fat diets have been

recommended in some guidelines, due to the physiology of fat delaying gastric emptying, there is limited evidence for this approach in patients with IGP, and the clinical benefit of fat restriction alone is unproven.[74,75] In individuals with malnutrition, caloric restriction is contraindicated.

Although oral nutritional supplements have not been specifically studied in IGP, they are a reasonable and practical strategy to address inadequate oral intake and established malnutrition. Once malnutrition is present, the primary objective of nutritional therapy should be its reversal, whereas symptom management requires a multifaceted approach. Notably, evidence from other conditions suggests that improving nutritional status can enhance gastric emptying; for example, completion of a re-nutrition program significantly reduced delayed gastric emptying and symptoms in patients with anorexia nervosa.[76]

Given the symptom overlap between gastroparesis and functional dyspepsia, dietary strategies used for patients with functional dyspepsia may also be considered. A low-FODMAP (fermentable oligosaccharides, disaccharides, monosaccharides and polyols) diet has limited but emerging evidence for reducing epigastric symptoms, early satiety, bloating and abdominal pain in patients with functional dyspepsia.[66,67] Such a diet may be trialled for a limited period of 6 weeks, with subsequent reintroduction of food groups.[77] However, restrictive diets should be avoided in patients with established or risk of malnutrition, and malnutrition is a contraindication to the low-FODMAP diet.

Constipation should be aggressively managed in patients with IGP using standard treatments,[78] to minimise confounding symptoms and optimise gastrointestinal motility.[79,80] In addition, slowly fermentable or viscous fibres, such as partially hydrolysed guar gum or low-dose psyllium, may be beneficial. Partially hydrolysed guar gum has shown particular benefit for global pain and functional gastrointestinal symptoms in patients with irritable bowel syndrome.[70]

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

6.3.3 Artificial nutritional support considerations

Initiation of ETF should be approached with caution in patients with IGP. The decision to initiate ETF should be made only after referral to a tertiary referral expert centre and consultation with an MDT, as tube feeding does not consistently relieve global symptoms and carries risk of iatrogenic harm.[81] For patients with medical instability from severe malnutrition who require immediate intervention, temporary nasogastric tube feeding may be considered as a bridging intervention until MDT assessment is available. Short-term nasogastric feeding is recommended over post-pyloric feeding, as gastric emptying time does not correlate well with symptom severity.[8] Principles to guide MDT decision making about ETF are provided in **Table 3**.

Table 3. Multidisciplinary team decision-making principles for temporary 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 behaviour.
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 individualised 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.

ETF = enteral tube feeding; MDT = multidisciplinary team.

Long-term ETF should be undertaken only after careful deliberation and consensus within an experienced MDT. In one study of patients with gastroparesis, 19 of 36 patients (53%) who proceeded to nasoduodenal feeding showed no symptomatic improvement but were nevertheless advanced to percutaneous endoscopic jejunostomy.[63] Other observational studies indicate that most patients with DGBI undergoing long-term ETF have no abatement of gastrointestinal symptoms (13/15 patients in one study),[81] and complication rates are relatively high (Appendix 2).[63,81]

Management of pain, visceral hypersensitivity, psychosocial aspects and disordered eating behaviour should be addressed in the MDT setting before making a decision to initiate long-term ETF.[73] It should be noted that even tube feeding may result in inconsistent nutritional improvement.[63,81,82] Long-term ETF should be reserved only for those at medical risk due to severe malnutrition and instituted only after all other reasonable steps have been attempted, with expert input. The goal of ETF should be primarily to reduce medical risk from malnutrition, rather than to treat symptoms.

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 enteral tube feeding should be made only with formal multidisciplinary team consultation. (Quality of evidence not applicable; Consensus recommendation)

Statement 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. (Low quality of evidence; Strong recommendation)

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.[83] If ETF is not tolerated because of symptoms, intensive multidisciplinary management of the associated DGBI is recommended, rather than escalation to parenteral nutrition.

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)

6.4 Pharmacotherapy

6.4.1 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 generalised to IGP.[84,85] A network meta-analysis of 29 trials in patients with gastroparesis of any aetiology indicated symptom benefit over placebo for dopamine antagonists.[86] A separate meta-analysis of 29 trials of prokinetics in patients with functional dyspepsia indicated global symptom benefit.[87]

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.[88] Adverse effects may include acute dystonia, prolonged QT interval and tardive dyskinesia. Domperidone does not cross the blood–brain barrier, reducing neurological side effects, although it may induce QT prolongation. Despite favourable evidence in diabetic gastroparesis,[89] only one of six placebo-controlled trials of domperidone included patients with IGP, but it did show symptom benefit.[90]

Use of prucalopride and erythromycin for patients with IGP is off-label. Erythromycin accelerated gastric emptying in the short term and reduced symptoms in patients with IGP in an uncontrolled study, but the prokinetic effect was diminished after 4 weeks, limiting its long-term utility.[91] Prucalopride, which is approved for use for 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.[92] Cisapride, a 5-HT_4 receptor agonist, was withdrawn due to a risk of prolonged QT arrhythmias.

Antiemetics such as phenothiazines (e.g. prochlorperazine) and antihistamines (e.g. promethazine, cyclizine) are used empirically in IGP.[89] Intravenous administration of cyclizine can induce euphoria and dependence.[93] Haloperidol was found to be superior to placebo for treating nausea in emergency presentations of patients with gastroparesis.[94] The 5-HT_3 receptor antagonist granisetron, administered via transdermal patches, reduced nausea and vomiting in open-label studies of patients with IGP.[95,96] Although a study found that the NK-1 receptor antagonist aprepitant reduced “gastroparesis-like” nausea and vomiting, compared with placebo, this was insufficient to satisfy the study’s prespecified primary outcome.[97]

The safety profile of all medications must be confirmed specifically for each patient, as standard medical care.

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

6.4.2 Pharmacological neuromodulation

Though widely used, few studies have assessed neuromodulator medications in patients with IGP. The only placebo-controlled randomised controlled trial (RCT) — the Nortriptyline for Idiopathic Gastroparesis (NORIG) trial in 2013 — allocated 130 patients with IGP to receive escalating doses of nortriptyline (from 10 mg to 75 mg) versus placebo over 15 weeks. It found no difference between the groups in the proportion of patients experiencing a 50% reduction in global Gastroparesis Cardinal Symptom Index (GCSI) scores (23% benefit with nortriptyline, 21% with placebo). Dose escalation failed in nearly half the participants due to medication intolerance, while 29% from the treatment group and 9% from the placebo group stopped treatment, despite equal numbers of reported adverse events between the groups.[98]

Although amitriptyline has not been formally tested in patients with IGP, placebo-controlled trials have shown global symptom benefit in those with functional dyspepsia.[99] In one RCT of 292 patients, 21% had delayed gastric emptying. Amitriptyline 50 mg daily over 12 weeks reduced functional dyspepsia symptoms, whereas escitalopram 10 mg daily did not, and patients with delayed gastric emptying were less likely to have improved global scores. Neither nortriptyline nor amitriptyline induced any further delay in gastric emptying.[100,101]

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, although 20% of participants stopped treatment because of adverse effects.[102] An 8-week RCT showed improvement in postprandial symptoms of functional dyspepsia with mirtazapine 15 mg daily, although gastric emptying was not measured.[103]

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, regardless of whether gastric emptying was delayed.[104]

The atypical antipsychotic medicines olanzapine and quetiapine are used as adjunctive therapy for functional nausea, and serotonin–noradrenaline reuptake inhibitors are used for unexplained pain, but these have not been studied in patients with IGP.

We recommend 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. The Rome Foundation 2018 report details the pharmacology, symptom targets and required precautions when prescribing neuromodulators for patients with DGBI (**Table 4**).[105]

Statement 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. (Low quality of evidence; Conditional recommendation)

Table 4. Summary of gut–brain neuromodulatory medications*

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	Motility: slow GI transit, largely related to their anticholinergic and noradrenergic properties Sensitivity: limited and inconsistent evidence that TCAs	• 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	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	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	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

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	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	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 date Sensitivity: decreased sensitivity to rectal distension in IBS	Motility: no date 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.

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.

6.4.3 Cannabinoids

Patients with gastroparesis often use cannabinoids (46% of patients in one study), with most perceiving symptom relief.[106,107] However, use of cannabinoids remains controversial, as they retard gastric emptying,[108] and large epidemiological studies (*n* = 41,374) indicate they are associated with higher health care utilisation.[109,110] A single placebo-controlled RCT of cannabidiol in 44 patients with gastroparesis found a reduction in global GCSI score and vomiting episodes. The cannabidiol group tolerated higher-volume satiation tests despite slower gastric emptying.[111] A second uncontrolled prospective study of 24 patients found similar symptomatic improvements, although no physiological endpoints were examined.[112]

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)

6.5 Psychological interventions

There is a marked absence of research into psychological interventions in gastroparesis, due to the historic focus on IGP as a motor disorder. Only one study has been published, in patients with postsurgical gastroparesis, which found that psychosocial support, music and massage therapy and family psychoeducation improved mood and residual gastric volume compared with standard medical care.[113] Acceptance of the established overlap with functional dyspepsia enables evidence from DGBI to be applied to patients with IGP.[114-120] However, psychological therapies are under-researched and underused even in DGBI, despite the acceptance of brain–gut behavioural therapy.[118] Research involving multidisciplinary approaches that include psychologists and psychiatrists for the management of DGBI report improved patient-reported outcomes (e.g. anxiety, depression, quality of life) and increased cost-effectiveness.[121]

Regardless, input from psychologists and psychiatrists is often indicated due to the high co-occurrence of mental health disorders in patients with IGP. Gastroparesis is associated with a significant psychosocial burden and low quality of life.[122-125] Anxiety and depression have reported pooled prevalences in patients with gastroparesis of 49% and 39%, respectively,[126] which are notably higher than the respective prevalences of 27.8% and 27.0% in patients with DGBI.[127] There is a strong evidence base for psychological interventions in disorders of mood, sleep, personality, trauma, eating and persistent pain, which frequently co-occur in this cohort. As such, cognitive behaviour therapy, hypnosis, mindfulness-based stress reduction, and acceptance and commitment-based therapy are likely to be of benefit. In the absence of targeted therapy, psychotherapy provides support and neuromodulation, which are beneficial to all patients living with chronic gastroduodenal symptoms, with the aim of reducing symptoms and improving tolerance and quality of life.

Experience of trauma, personality vulnerabilities and abnormal illness beliefs can also significantly affect therapeutic outcomes.[123,128] These factors can increase the risk of splitting, countertransference and iatrogenic harm through inappropriate rejection, fragmentation or escalation of care, particularly when combined with the helplessness that health practitioners may experience in the face of chronic illness.

Comorbid eating disorders are common in patients with gastrointestinal disorders,[48,52] but there is a lack of high-quality research to guide management. IGP guidelines have traditionally recommended exclusion of eating disorders, omitting guidance for those with coexisting disorders.[19,20] When disordered eating or an eating disorder is present in a patient with IGP, it is important for the gastroenterologist to work closely with eating disorder clinicians to co-assess and co-manage these patients, to optimise their outcomes.[129]

Mental health clinicians provide pivotal assessment and formulation of how these mental health issues intersect with IGP, in addition to offering psychoeducation and psychotherapy. Additionally, psychiatrists have psychotropic medication expertise.[130] There are few mental health clinicians with expertise in gastrointestinal conditions in Australia. In their absence, close collaboration with an experienced general mental health clinician is recommended.

Statement 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. (Low quality of evidence; Strong recommendation)

Statement 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. (Low quality of evidence; Strong recommendation)

6.6 Interventional therapies

The European Society of Gastrointestinal Endoscopy 2020 guideline recommends against the use of pyloric botulinum toxin (botox) injection in unselected patients or as a screening test for further pyloric interventions.[131] A randomised sham-controlled crossover trial of intrapyloric botox injection in patients with gastroparesis found no improvement in either gastric emptying or symptoms.[132] One pilot study reported that pyloric distensibility measured by an endoscopic functional luminal imaging probe predicted symptomatic response to intrapyloric botox injection, but further data are needed.[133]

Gastric peroral endoscopic myotomy (G-POEM) has emerged as a promising, minimally invasive therapeutic option to reduce pyloric resistance to gastric emptying.[134] Initial studies suggest G-POEM provides significant symptomatic relief and improved gastric emptying in patients with refractory gastroparesis. Three non-randomised trials showed success rates of 58%–60% at 6 months,[135-137] with long-term success varying from 75% at 3 years[138] to 87% at 5 years.[139] High body mass index, longer duration of gastroparesis, psychiatric comorbidity and narcotic medication use have been associated with poor outcomes.[140] Only one sham-controlled RCT has been published, including 41 patients (17 with diabetic gastroparesis, 13 with postsurgical gastroparesis and 11 with IGP). Of the 21 patients randomly assigned to receive G-POEM, 71% benefited, with a 50% reduction in GCSI score and improved gastric emptying 6 months after the procedure, compared with 22% with the sham procedure.[141] Subgroup analysis was inconclusive in the patients with IGP. Moreover, an RCT comparing botox injection with G-POEM found no difference in clinical success rate or gastric emptying times,[142] while a meta-analysis of G-POEM versus surgical pyloroplasty suggested similar clinical outcomes, but greater cost-effectiveness with G-POEM.[143] It is unclear what mechanism would favour G-POEM over the previous unsuccessful interventions targeting the pylorus. Further longitudinal sham-controlled studies are needed to confirm early findings and guide patient selection.

In an RCT, gastric electrical stimulation was not found to be superior to placebo in patients with IGP, with no difference in vomiting between those having stimulation turned on or off in a blinded fashion.[144] However, a 4-month double-blind sham-controlled RCT of 133 patients with refractory vomiting — 78% of whom had gastroparesis, although the percentage with IGP was not defined — found a reduction in vomiting frequency during periods of stimulation, unrelated to baseline gastric emptying. Adverse events predominantly related to the implantation site.[31]

Given there is no consistent effect on gastric emptying, an underlying neuromodulator effect is proposed, with further studies underway.

Statement 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 trialled following multidisciplinary team consensus. (Low quality of evidence; Conditional recommendation)

7 Conclusion

This is the first Australian position statement on the assessment and management of IGP. In contrast to prior guidelines, we propose that gastroparesis should be considered a chronic sensorimotor disorder rather than an isolated motility disorder. Twenty statements have been developed and refined by consensus and given a grade of evidence and strength of recommendation based on available evidence and expert opinion.

The literature indicates that, in many cases, distinguishing gastroparesis from functional gastroduodenal disorders — particularly functional dyspepsia but also chronic nausea and vomiting syndrome — is not possible based on symptoms or gastric emptying time. Gastric emptying is highly variable over time and correlates poorly with symptoms. The evidence gathered in this position statement suggests that current terminology and a reliance on gastric emptying as the defining feature of IGP are problematic, may translate to suboptimal management and have constrained new therapeutic developments.

A novel recommendation from this position statement is the application of treatments established for functional dyspepsia and DGBI to IGP, in addition to the current recommended treatments targeting gastric emptying. A core focus of treatment is to minimise iatrogenic harm. Given the biopsychosocial comorbidity associated with IGP, multidisciplinary care is advised. Specialist tertiary MDT input is recommended as standard of care if first-line treatment fails. When disordered eating behaviour 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.

A trial of prokinetic or antiemetic medication is recommended in practice, combined with formal dietary assessment and management, although evidence for specific agents is limited. This position statement recommends a symptom-based approach to the adjunctive use of treatments established for DGBI, persistent pain and psychiatric disorders, which commonly overlap with IGP. Early psychological support is recommended, with mental health clinicians forming a core part of the treatment team for patients with IGP. Interventional endoscopic and surgical treatment options should only be considered if engagement with intensive multidisciplinary treatment is unsuccessful, and with formal tertiary MDT consensus.

We have identified several key areas of need for future development: interdisciplinary research extending beyond gastric emptying to the many pathophysiological mechanisms underlying symptom genesis; a combination of testing modalities which more accurately phenotypes both the sensory and motor aspects of IGP; the development of medically endorsed educational resources to combat online misinformation; and finally, a basic shift in Australian public hospital funding recognising mental health clinicians as core members of the MDT.

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

Acknowledgements
The working group acknowledges, with thanks, the following experts for providing external review and input into the development of this document:

- Jane Andrews, Gastroenterologist
- Jamee Barugh, Dietitian
- Andrew Buckle, Gastroenterologist
- Chris Cederwall, Gastroenterologist (New Zealand)
- Charlotte Daker, Gastroenterologist (New Zealand)
- Asma Fikree, Gastroenterologist (United Kingdom)
- Karen Jones, Nuclear Medicine Physician
- Jim Kantidakis, Psychologist
- Geoffrey Kohn, Surgeon
- Kate Lane, Dietitian
- Lee Martin, Dietitian (United Kingdom)
- Kate Murphy, Psychiatrist
- Greg O'Grady, Surgeon (New Zealand)
- Zoe Raos, Gastroenterologist (New Zealand)
- Nikhil Thapar, Paediatric Gastroenterologist

GESA Luminal Faculty Committee members:
- Charles Cock, Gastroenterologist
- Magnus Halland, Gastroenterologist
- Allison Malcolm, Gastroenterologist
- Heidi Staudacher, Dietitian
- Abhinav Vasudevan, Gastroenterologist

Special thanks are extended to the members of the GESA Luminal Faculty patient advocacy group for their vital feedback, which has been incorporated into this document to ensure representation of the patient experience.

Appendices

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 fibre
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 fibre
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 fibre
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 fibre
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 fibre
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 fibre

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); fibre: 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 fibre
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 fibre
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 fibre
Afternoon tea	Sustagen (250 mL)	0.9 kJ energy 13 g protein 30 g carbohydrates 5 g fat 0 g fibre
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 fibre
Supper	Puréed fruit (3/4 cup)	0.7 MJ energy 6 g protein 35 g carbohydrates 1 g fat 6 g fibre

Nutritional analysis: energy: 7.2 MJ; protein: 92 g (22% of energy); carbohydrates: 160 g (44% of energy); fat: 74 g (37% of energy); fibre: 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

Table A3. Nutritional outcomes of enteral feeding in gastroparesis: evidence summary

Author, year, location	Population and study design	Outcomes
Gallo, 2023, Australia (abstract only)[81]	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)[82]	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.
Strijbos, 2019, Netherlands[63]	Gastroparesis (*n* = 86) Diabetes 26%, postsurgical 27%, idiopathic 38%, generalised 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.

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

References

1. Moshiree B, Potter M, Talley NJ. Epidemiology and Pathophysiology of Gastroparesis. *Gastrointest Endosc Clin N Am* 2019; **29**(1): 1-14.
2. Ye Y, Jiang B, Manne S, et al. Epidemiology and outcomes of gastroparesis, as documented in general practice records, in the United Kingdom. *Gut* 2021; **70**(4): 644-53.
3. Ye Y, Yin Y, Huh SY, Almansa C, Bennett D, Camilleri M. Epidemiology, Etiology, and Treatment of Gastroparesis: Real-World Evidence From a Large US National Claims Database. *Gastroenterology* 2022; **162**(1): 109-21.e5.
4. Lacy BE, Crowell MD, Mathis C, Bauer D, Heinberg LJ. Gastroparesis: Quality of Life and Health Care Utilization. *J Clin Gastroenterol* 2018; **52**(1): 20-4.
5. Wadhwa V, Mehta D, Jobanputra Y, Lopez R, Thota PN, Sanaka MR. Healthcare utilization and costs associated with gastroparesis. *World J Gastroenterol* 2017; **23**(24): 4428-36.
6. Lacy BE, Wise JL, Cangemi DJ. Gastroparesis: time for a paradigm change. *Curr Opin Gastroenterol* 2023; **39**(6): 503-11.
7. Stanghellini V, Tosetti C, Paternico A, et al. Risk indicators of delayed gastric emptying of solids in patients with functional dyspepsia. *Gastroenterology* 1996; **110**(4): 1036-42.
8. Pasricha PJ, Colvin R, Yates K, et al. Characteristics of patients with chronic unexplained nausea and vomiting and normal gastric emptying. *Clin Gastroenterol Hepatol* 2011; **9**(7): 567-76.e1-4.
9. Lacy BE. Functional dyspepsia and gastroparesis: one disease or two? *Am J Gastroenterol* 2012; **107**(11): 1615-20.
10. Stanghellini V, Tack J. Gastroparesis: separate entity or just a part of dyspepsia? *Gut* 2014; **63**(12): 1972.
11. Chokshi RV, Chang L. Is it time to abandon gastric emptying in patients with symptoms of gastroparesis and functional dyspepsia? *Clin Gastroenterol Hepatol* 2022; **20**(3): e359-e60.
12. Cangemi DJ, Lacy BE. Gastroparesis: Myths, Misconceptions, and Management. *Clin Exp Gastroenterol* 2023; **16**: 65-78.
13. Egboh SM, Duncanson K, Potter M, Keely S, Talley NJ. Functional dyspepsia and gastroparesis: are they distinct disorders, a spectrum of diseases or one disease? *eGastroenterology* 2025; **3**(1): e100119.
14. Pasricha PJ, Grover M, Yates KP, et al. Functional Dyspepsia and Gastroparesis in Tertiary Care are Interchangeable Syndromes With Common Clinical and Pathologic Features. *Gastroenterology* 2021; **160**(6): 2006-17.
15. Wauters L, Dickman R, Drug V, et al. United European Gastroenterology (UEG) and European Society for Neurogastroenterology and Motility (ESNM) consensus on functional dyspepsia. *United European Gastroenterol J* 2021; **9**(3): 307-31.
16. Schol J, Wauters L, Dickman R, et al. United European Gastroenterology (UEG) and European Society for Neurogastroenterology and Motility (ESNM) consensus on gastroparesis. *United European Gastroenterol J* 2021; **9**(3): 287-306.
17. Salzberg MR, Kim H, Basnayake C, Holt D, Kamm MA. Role of social media in the presentation of disorders of gut-brain interaction: Review and recommendations. *J Gastroenterol Hepatol* 2024; **39**(11): 2281-92.
18. Giedinghagen A. 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. *Clin Child Psychol Psychiatry* 2023; **28**(1): 270-8.
19. Camilleri M, Kuo B, Nguyen L, et al. ACG Clinical Guideline: Gastroparesis. *Am J Gastroenterol* 2022; **117**(8): 1197-220.
20. Schol J, Huang IH, Carbone F, et al. Rome Foundation and international neurogastroenterology and motility societies' consensus on idiopathic gastroparesis. *Lancet Gastroenterol Hepatol* 2025; **10**(1): 68-81.
21. Balshem H, Helfand M, Schünemann HJ, et al. GRADE guidelines: 3. Rating the quality of evidence. *J Clin Epidemiol* 2011; **64**(4): 401-6.

22. Guyatt G, Agoritsas T, Brignardello-Petersen R, et al. Core GRADE 1: overview of the Core GRADE approach. *BMJ* 2025; **389**: e081903.
23. Guyatt G, Zeng L, Brignardello-Petersen R, et al. Core GRADE 2: choosing the target of certainty rating and assessing imprecision. *BMJ* 2025; **389**: e081904.
24. Guyatt G, Schandelmaier S, Brignardello-Petersen R, et al. Core GRADE 3: rating certainty of evidence-assessing inconsistency. *BMJ* 2025; **389**: e081905.
25. Guyatt G, Wang Y, Eachempati P, et al. Core GRADE 4: rating certainty of evidence-risk of bias, publication bias, and reasons for rating up certainty. *BMJ* 2025; **389**: e083864.
26. Guyatt G, Iorio A, De Beer H, et al. Core GRADE 5: rating certainty of evidence-assessing indirectness. *BMJ* 2025; **389**: e083865.
27. Guyatt G, Yao L, Murad MH, et al. Core GRADE 6: presenting the evidence in summary of findings tables. *BMJ* 2025; **389**: e083866.
28. Guyatt G, Vandvik PO, Iorio A, et al. Core GRADE 7: principles for moving from evidence to recommendations and decisions. *BMJ* 2025; **389**: e083867.
29. Brouwers MC, Kerkvliet K, Spithoff K. The AGREE Reporting Checklist: a tool to improve reporting of clinical practice guidelines. *BMJ* 2016; **352**: i1152.
30. Pasricha PJ, Grover M, Yates KP, et al. Progress in gastroparesis - a narrative review of the work of the Gastroparesis Clinical Research Consortium. *Clin Gastroenterol Hepatol* 2022; **20**(12): 2684-95.e3.
31. Ducrotte P, Coffin B, Bonaz B, et al. Gastric Electrical Stimulation Reduces Refractory Vomiting in a Randomized Crossover Trial. *Gastroenterology* 2020; **158**(3): 506-14.e2.
32. Rothstein RD, Alavi A, Reynolds JC. Electrogastrography in patients with gastroparesis and effect of long-term cisapride. *Dig Dis Sci* 1993; **38**(8): 1518-24.
33. O'Grady G, Angeli TR, Du P, et al. Abnormal initiation and conduction of slow-wave activity in gastroparesis, defined by high-resolution electrical mapping. *Gastroenterology* 2012; **143**(3): 589-98.e3.
34. Gharibans AA, Calder S, Varghese C, et al. Gastric dysfunction in patients with chronic nausea and vomiting syndromes defined by a noninvasive gastric mapping device. *Sci Transl Med* 2022; **14**(663): eabq3544.
35. Lin Z, Sarosiek I, Forster J, Damjanov I, Hou Q, McCallum RW. Association of the status of interstitial cells of Cajal and electrogastrogram parameters, gastric emptying and symptoms in patients with gastroparesis. *Neurogastroenterol Motil* 2010; **22**(1): 56-61, e10.
36. Bekkelund M, Sangnes DA, Gunnar Hatlebakk J, Aabakken L. Pathophysiology of idiopathic gastroparesis and implications for therapy. *Scand J Gastroenterol* 2019; **54**(1): 8-17.
37. Troncon LE, Bennett RJ, Ahluwalia NK, Thompson DG. Abnormal intragastric distribution of food during gastric emptying in functional dyspepsia patients. *Gut* 1994; **35**(3): 327-32.
38. Debourdeau A, Gonzalez JM, Mathias F, Prost C, Barthet M, Vitton V. Gastric volumetry for the assessment of fundic compliance and visceral hypersensitivity in patients with gastroparesis: a retrospective comparative study. *Scand J Gastroenterol* 2024; **59**(3): 254-9.
39. Karamanolis G, Caenepeel P, Arts J, Tack J. Determinants of symptom pattern in idiopathic severely delayed gastric emptying: gastric emptying rate or proximal stomach dysfunction? *Gut* 2007; **56**(1): 29-36.
40. Orthey P, Yu D, Van Natta ML, et al. Intragastric Meal Distribution During Gastric Emptying Scintigraphy for Assessment of Fundic Accommodation: Correlation with Symptoms of Gastroparesis. *J Nucl Med* 2018; **59**(4): 691-7.
41. Kamal AN, Harris KB, Sarvapalli S, Sayuk GS, Gyawali CP, Gabbard S. Nutrient Drink Test to Assess Gastric Accommodation in Cyclic Vomiting Syndrome: Single-blinded Parallel Grouped Prospective Study. *J Neurogastroenterol Motil* 2023; **29**(1): 65-71.
42. Vanuytsel T, Bercik P, Boeckxstaens G. Understanding neuroimmune interactions in disorders of gut-brain interaction: from functional to immune-mediated disorders. *Gut* 2023; **72**(4): 787-98.

43. Wei L, Singh R, Ghoshal UC. Enterochromaffin cells-gut microbiota crosstalk: underpinning the symptoms, pathogenesis, and pharmacotherapy in disorders of gut-brain interaction. *J Neurogastroenterol Motil* 2022; **28**(3): 357-75.
44. Aguilera-Lizarraga J, Hussein H, Boeckxstaens GE. Immune activation in irritable bowel syndrome: what is the evidence? *Nat Rev Immunol* 2022; **22**(11): 674-86.
45. Tack J, Carbone F, Chang L, Lacy BE. Patient-Reported Outcomes in Disorders of Gut-Brain Interaction. *Gastroenterology* 2024; **166**(4): 572-87.e1.
46. Riedlinger C, Schmidt G, Weiland A, et al. Which symptoms, complaints and complications of the gastrointestinal tract occur in patients with eating disorders? A systematic review and quantitative analysis. *Front Psychiatry* 2020; **11**: 195.
47. Schalla MA, Stengel A. Gastrointestinal alterations in anorexia nervosa - A systematic review. *Eur Eat Disord Rev* 2019; **27**(5): 447-61.
48. Staller K, Abber SR, Burton Murray H. The intersection between eating disorders and gastrointestinal disorders: a narrative review and practical guide. *Lancet Gastroenterol Hepatol* 2023; **8**(6): 565-78.
49. Szmukler GI, Young GP, Lichtenstein M, Andrews JT. A serial study of gastric emptying in anorexia nervosa and bulimia. *Aust N Z J Med* 1990; **20**(3): 220-5.
50. Hanel V, Schalla MA, Stengel A. Irritable bowel syndrome and functional dyspepsia in patients with eating disorders - a systematic review. *Eur Eat Disord Rev* 2021; **29**(5): 692-719.
51. Wiklund CA, Rania M, Kuja-Halkola R, Thornton LM, Bulik CM. Evaluating disorders of gut-brain interaction in eating disorders. *Int J Eat Disord* 2021; **54**(6): 925-35.
52. Mikhael-Moussa H, Bertrand V, Lejeune E, et al. The association of avoidant/restrictive food intake disorder (ARFID) and neurogastroenterology disorders (including disorders of gut-brain interaction [DGBI]): a scoping review. *Neurogastroenterol Motil* 2025; **37**(9): e70039.
53. Soliman OM, Mikocka-Walus A, Warner MM, Skvarc D, Olive L, Knowles SR. Systematic Review and Meta-Analysis: Examining the Psychometric Evaluations of Disordered Eating Scales in Adults Living With Gastrointestinal Conditions. *Neurogastroenterol Motil* 2025; **37**(5): e15018.
54. Shivji S, Conner JR, Kirsch R. Mast cell evaluation in gastrointestinal biopsies: should we be counting? A critical review and practical guide for the surgical pathologist. *Histopathology* 2023; **82**(7): 960-73.
55. Vijayvargiya P, Jameie-Oskooei S, Camilleri M, Chedid V, Erwin PJ, Murad MH. Association between delayed gastric emptying and upper gastrointestinal symptoms: a systematic review and meta-analysis. *Gut* 2019; **68**(5): 804-13.
56. NSW Agency for Clinical Innovation. Gastric emptying scintigraphy: clinical practice guide. Sydney: ACI; 2023.
57. Carson DA, Bhat S, Hayes TCL, et al. Abnormalities on electrogastrography in nausea and vomiting syndromes: a systematic review, meta-analysis, and comparison to other gastric disorders. *Dig Dis Sci* 2022; **67**(3): 773-85.
58. Oka A, Awoniyi M, Hasegawa N, et al. Superior mesenteric artery syndrome: Diagnosis and management. *World J Clin Cases* 2023; **11**(15): 3369-84.
59. Rosa-Jiménez F, Rodríguez González FJ, Puente Gutiérrez JJ, Muñoz Sánchez R, Adarraga Cansino MD, Zambrana García JL. Duodenal compression caused by superior mesenteric artery: study of 10 patients [English, Spanish]. *Rev Esp Enferm Dig* 2003; **95**(7): 485-9, 0-4.
60. Baskan O, Kaya E, Gungoren FZ, Erol C. Compression of the celiac artery by the median arcuate ligament: multidetector computed tomography findings and characteristics. *Can Assoc Radiol J* 2015; **66**(3): 272-6.
61. Terlouw LG, Moelker A, Abrahamsen J, et al. European guidelines on chronic mesenteric ischaemia - joint United European Gastroenterology, European Association for Gastroenterology, Endoscopy and Nutrition, European Society of Gastrointestinal and Abdominal Radiology, Netherlands Association of Hepatogastroenterologists, Hellenic

Society of Gastroenterology, Cardiovascular and Interventional Radiological Society of Europe, and Dutch Mesenteric Ischemia Study group clinical guidelines on the diagnosis and treatment of patients with chronic mesenteric ischaemia. *United European Gastroenterol J* 2020; **8**(4): 371-95.
62. Parkman HP, Yates KP, Hasler WL, et al. Dietary intake and nutritional deficiencies in patients with diabetic or idiopathic gastroparesis. *Gastroenterology* 2011; **141**(2): 486-98, 98.e1-7.
63. Strijbos D, Keszthelyi D, Smeets FG, et al. Therapeutic strategies in gastroparesis: results of stepwise approach with diet and prokinetics, gastric rest, and PEG-J: a retrospective analysis. *Neurogastroenterol Motil* 2019; **31**(6): e13588.
64. Olausson EA, Störsrud S, Grundin H, Isaksson M, Attvall S, Simrén M. A small particle size diet reduces upper gastrointestinal symptoms in patients with diabetic gastroparesis: a randomized controlled trial. *Am J Gastroenterol* 2014; **109**(3): 375-85.
65. Limketkai BN, LeBrett W, Lin L, Shah ND. Nutritional approaches for gastroparesis. *Lancet Gastroenterol Hepatol* 2020; **5**(11): 1017-26.
66. Goyal O, Nohria S, Batta S, Dhaliwal A, Goyal P, Sood A. Low fermentable oligosaccharides, disaccharides, monosaccharides, and polyols diet versus traditional dietary advice for functional dyspepsia: a randomized controlled trial. *J Gastroenterol Hepatol* 2022; **37**(2): 301-9.
67. Staudacher HM, Nevin AN, Duff C, Kendall BJ, Holtmann GJ. Epigastric symptom response to low FODMAP dietary advice compared with standard dietetic advice in individuals with functional dyspepsia. *Neurogastroenterol Motil* 2021; **33**(11): e14148.
68. So D, Gibson PR, Muir JG, Yao CK. Dietary fibres and IBS: translating functional characteristics to clinical value in the era of personalised medicine. *Gut* 2021; **70**(12): 2383-94.
69. Parkman HP, Yates KP, Hasler WL, et al. Dietary intake and nutritional deficiencies in patients with diabetic or idiopathic gastroparesis. *Gastroenterology* 2011; **141**(2): 486-98, 98.e1-7.
70. Suresh H, Zhou J, Ho V. The Short-Term Effects and Tolerability of Low-Viscosity Soluble Fibre on Gastroparesis Patients: A Pilot Clinical Intervention Study. *Nutrients* 2021; **13**(12): 4298.
71. Olausson EA, Störsrud S, Grundin H, Isaksson M, Attvall S, Simrén M. A small particle size diet reduces upper gastrointestinal symptoms in patients with diabetic gastroparesis: a randomized controlled trial. *Am J Gastroenterol* 2014; **109**(3): 375-85.
72. Moore JG, Datz FL, Christian PE, Greenberg E, Alazraki N. Effect of body posture on radionuclide measurements of gastric emptying. *Dig Dis Sci* 1988; **33**(12): 1592-5.
73. Paine P, McMahon M, Farrer K, Overshott R, Lal S. Jejunal feeding: when is it the right thing to do? *Frontline Gastroenterol* 2019; **11**(5): 397-403.
74. Homko CJ, Duffy F, Friedenberg FK, Boden G, Parkman HP. Effect of dietary fat and food consistency on gastroparesis symptoms in patients with gastroparesis. *Neurogastroenterol Motil* 2015; **27**(4): 501-8.
75. Mogilevski T, Rosella S, Nguyen A, et al. Characterisation of biomarkers of intestinal barrier function in response to a high fat/high carbohydrate meal and corticotropin releasing hormone. *PLoS One* 2024; **19**(2): e0294918.
76. Rigaud D, Bedig G, Merrouche M, Vulpillat M, Bonfils S, Apfelbaum M. Delayed gastric emptying in anorexia nervosa is improved by completion of a renutrition program. *Dig Dis Sci* 1988; **33**(8): 919-25.
77. Marsh A, Eslick EM, Eslick GD. Does a diet low in FODMAPs reduce symptoms associated with functional gastrointestinal disorders? A comprehensive systematic review and meta-analysis. *Eur J Nutr* 2016; **55**(3): 897-906.
78. Serra J, Pohl D, Azpiroz F, et al. European society of neurogastroenterology and motility guidelines on functional constipation in adults. *Neurogastroenterol Motil* 2020; **32**(2): e13762.

79. Boccia G, Buonavolontà R, Coccorullo P, Manguso F, Fuiano L, Staiano A. Dyspeptic symptoms in children: the result of a constipation-induced cologastric brake? *Clin Gastroenterol Hepatol* 2008; **6**(5): 556-60.
80. Tjeerdsma HC, Smout AJ, Akkermans LM. Voluntary suppression of defecation delays gastric emptying. *Dig Dis Sci* 1993; **38**(5): 832-6.
81. Gallo R, Armstrong E, Griffin H, Russell D, Hebbard G. Impact of enteral tube feeding for individuals with disorders of the gut brain interaction [abstract]. *Clin Nutr ESPEN* 2023; **58**: 604.
82. Martin LD, Wang X, Day C, et al. Enteral tube feeding in functional gastrointestinal disorders: A game of chance? [abstract]. *Clini Nutr ESPEN* 2023; **57**: 849.
83. Comerlato PH, Stefani J, Viana LV. Mortality and overall and specific infection complication rates in patients who receive parenteral nutrition: systematic review and meta-analysis with trial sequential analysis. *Am J Clin Nutr* 2021; **114**(4): 1535-45.
84. Janssen P, Harris MS, Jones M, et al. The relation between symptom improvement and gastric emptying in the treatment of diabetic and idiopathic gastroparesis. *Am J Gastroenterol* 2013; **108**(9): 1382-91.
85. Vijayvargiya P, Camilleri M, Chedid V, Mandawat A, Erwin PJ, Murad MH. Effects of Promotility Agents on Gastric Emptying and Symptoms: A Systematic Review and Meta-analysis. *Gastroenterology* 2019; **156**(6): 1650-60.
86. Ingrosso MR, Camilleri M, Tack J, Ianiro G, Black CJ, Ford AC. Efficacy and Safety of Drugs for Gastroparesis: Systematic Review and Network Meta-analysis. *Gastroenterology* 2023; **164**(4): 642-54.
87. Pittayanon R, Yuan Y, Bollegala NP, et al. Prokinetics for Functional Dyspepsia: A Systematic Review and Meta-Analysis of Randomized Control Trials. *Am J Gastroenterol* 2019; **114**(2): 233-43.
88. Perkel MS, Moore C, Hersh T, Davidson ED. Metoclopramide therapy in patients with delayed gastric emptying: a randomized, double-blind study. *Dig Dis Sci* 1979; **24**(9): 662-6.
89. Camilleri M, Parkman HP, Shafi MA, Abell TL, Gerson L. Clinical guideline: management of gastroparesis. *Am J Gastroenterol* 2013; **108**(1): 18-37.
90. Davis RH, Clench MH, Mathias JR. Effects of domperidone in patients with chronic unexplained upper gastrointestinal symptoms: a double-blind, placebo-controlled study. *Dig Dis Sci* 1988; **33**(12): 1505-11.
91. Richards RD, Davenport K, McCallum RW. The treatment of idiopathic and diabetic gastroparesis with acute intravenous and chronic oral erythromycin. *Am J Gastroenterol* 1993; **88**(2): 203-7.
92. Carbone F, Van den Houte K, Clevers E, et al. Prucalopride in Gastroparesis: A Randomized Placebo-Controlled Crossover Study. *Am J Gastroenterol* 2019; **114**(8): 1265-74.
93. Bailey F, Davies A. The misuse/abuse of antihistamine antiemetic medication (cyclizine) by cancer patients. *Palliat Med* 2008; **22**(7): 869-71.
94. Roldan CJ, Chambers KA, Paniagua L, Patel S, Cardenas-Turanzas M, Chathampally Y. Randomized Controlled Double-blind Trial Comparing Haloperidol Combined With Conventional Therapy to Conventional Therapy Alone in Patients With Symptomatic Gastroparesis. *Acad Emerg Med* 2017; **24**(11): 1307-14.
95. Midani D, Parkman HP. Granisetron Transdermal System for Treatment of Symptoms of Gastroparesis: A Prescription Registry Study. *J Neurogastroenterol Motil* 2016; **22**(4): 650-5.
96. Simmons K, Parkman HP. Granisetron transdermal system improves refractory nausea and vomiting in gastroparesis. *Dig Dis Sci* 2014; **59**(6): 1231-4.
97. Pasricha PJ, Yates KP, Sarosiek I, et al. Aprepitant Has Mixed Effects on Nausea and Reduces Other Symptoms in Patients With Gastroparesis and Related Disorders. *Gastroenterology* 2018; **154**(1): 65-76.e11.

98. Parkman HP, Van Natta ML, Abell TL, et al. Effect of nortriptyline on symptoms of idiopathic gastroparesis: the NORIG randomized clinical trial. *JAMA* 2013; **310**(24): 2640-9.
99. Braak B, Klooker TK, Wouters MM, Lei A, van den Wijngaard RM, Boeckxstaens GE. Randomised clinical trial: the effects of amitriptyline on drinking capacity and symptoms in patients with functional dyspepsia, a double-blind placebo-controlled study. *Aliment Pharmacol Ther* 2011; **34**(6): 638-48.
100. Lacy BE, Saito YA, Camilleri M, et al. Effects of Antidepressants on Gastric Function in Patients with Functional Dyspepsia. *Am J Gastroenterol* 2018; **113**(2): 216-24.
101. Talley NJ, Locke GR, Saito YA, et al. Effect of amitriptyline and escitalopram on functional dyspepsia: a multicenter, randomized controlled study. *Gastroenterology* 2015; **149**(2): 340-9.e2.
102. Malamood M, Roberts A, Kataria R, Parkman HP, Schey R. Mirtazapine for symptom control in refractory gastroparesis. *Drug Des Devel Ther* 2017; **11**: 1035-41.
103. Cao L, Li G, Cao J, Li F, Han W. Randomized clinical trial: the effects of mirtazapine in functional dyspepsia patients. *Therap Adv Gastroenterol* 2025; **18**: 17562848241311129.
104. Parkman HP, Yates KP, Sarosiek I, et al. Buspirone for early satiety and symptoms of gastroparesis: a multi-centre, randomised, placebo-controlled, double-masked trial (BESST). *Aliment Pharmacol Ther* 2023; **57**(11): 1272-89.
105. Drossman DA, Tack J, Ford AC, Szigethy E, Törnblom H, Van Oudenhove L. Neuromodulators for Functional Gastrointestinal Disorders (Disorders of Gut-Brain Interaction): A Rome Foundation Working Team Report. *Gastroenterology* 2018; **154**(4): 1140-71.e1.
106. Jehangir A, Parkman HP. Cannabinoid Use in Patients With Gastroparesis and Related Disorders: Prevalence and Benefit. *Am J Gastroenterol* 2019; **114**(6): 945-53.
107. Parkman HP, Sharkey EP, Nguyen LA, et al. Marijuana Use in Patients with Symptoms of Gastroparesis: Prevalence, Patient Characteristics, and Perceived Benefit. *Dig Dis Sci* 2020; **65**(8): 2311-20.
108. Camilleri M, Zheng T. Cannabinoids and the Gastrointestinal Tract. *Clin Gastroenterol Hepatol* 2023; **21**(13): 3217-29.
109. Kilani Y, Gonzalez Mosquera DA, Puelo PC, et al. The impact of cannabis use in gastroparesis: a propensity-matched analysis of 41,374 gastroparesis patients. *Am J Gastroenterol*, 8 Apr, 2025. https://www.ncbi.nlm.nih.gov/pubmed/40197432 (accessed Apr 8).
110. Parkman HP, Xin Y, Wilson LA, et al. Characterization of patients with symptoms of gastroparesis having frequent emergency department visits and hospitalizations. *Clin Gastroenterol Hepatol* 2025; **23**(11): 2023-38.
111. Zheng T, BouSaba J, Taylor A, et al. A Randomized, Controlled Trial of Efficacy and Safety of Cannabidiol in Idiopathic and Diabetic Gastroparesis. *Clin Gastroenterol Hepatol* 2023; **21**(13): 3405-14.e4.
112. Barbash B, Mehta D, Siddiqui MT, Chawla L, Dworkin B. Impact of Cannabinoids on Symptoms of Refractory Gastroparesis: A Single-center Experience. *Cureus* 2019; **11**(12): e6430.
113. Liu Y, Song X, Zhang Y, Zhou L, Ni R. The effects of comprehensive mental intervention on the recovery time of patients with postsurgical gastroparesis syndrome. *J Clin Nurs* 2014; **23**(21-22): 3138-47.
114. Slouha E, Patel B, Mohamed A, Razeq Z, Clunes LA, Kollias TF. Psychotherapy for Irritable Bowel Syndrome: A Systematic Review. *Cureus* 2023; **15**(12): e51003.
115. Black CJ, Thakur ER, Houghton LA, Quigley EMM, Moayyedi P, Ford AC. Efficacy of psychological therapies for irritable bowel syndrome: systematic review and network meta-analysis. *Gut* 2020; **69**(8): 1441-51.
116. Mikocka-Walus A, Evans S, Linardon J, Wilding H, Knowles SR. Psychotherapy appears to improve symptoms of functional dyspepsia and anxiety: systematic review with meta-analysis. *Psychol Health Med* 2023; **28**(5): 1309-35.

117. Ballou S, Vasant DH, Guadagnoli L, et al. A primer for the gastroenterology provider on psychosocial assessment of patients with disorders of gut-brain interaction. *Neurogastroenterol Motil* 2024; **36**(12): e14894.
118. Keefer L, Ballou SK, Drossman DA, Ringstrom G, Elsenbruch S, Ljótsson B. A Rome Working Team Report on brain–gut behavior therapies for disorders of gut–brain interaction. *Gastroenterology* 2022; **162**(1): 300-15.
119. Knowles SR, Keefer L, Mikocka-Walus AA, editors. Psychogastroenterology for adults: a handbook for mental health professionals. London: Routledge; 2019.
120. Goodoory VC, Khasawneh M, Thakur ER, et al. Effect of Brain-Gut Behavioral Treatments on Abdominal Pain in Irritable Bowel Syndrome: Systematic Review and Network Meta-Analysis. *Gastroenterology* 2024; **167**(5): 934-43.e5.
121. Dendrinos E, Kim H, Hubik D, et al. Clinical outcomes of patients seen by psychiatrists in a multidisciplinary clinic for disorders of gut-brain interaction. *Intern Med J* 2025; **55**(10): 1687-94.
122. Simons M, Cline M. Scoping review: the social and emotional impacts of gastroparesis. *Transl Gastroenterol Hepatol* 2024; **9**: 42.
123. Woodhouse S, Hebbard G, Knowles SR. Exploration of the psychosocial issues associated with gastroparesis: a qualitative investigation. *J Clin Nurs* 2017; **26**(21-22): 3553-63.
124. Bennell J, Taylor C. A loss of social eating: the experience of individuals living with gastroparesis. *J Clin Nurs* 2013; **22**(19-20): 2812-21.
125. Woodhouse S, Hebbard G, Knowles SR. Psychological controversies in gastroparesis: A systematic review. *World J Gastroenterol* 2017; **23**(7): 1298-309.
126. Overs J, Morgan S, Apputhurai P, Tuck C, Knowles SR. Comparing the prevalence and association between anxiety, depression and gastrointestinal symptoms in gastroparesis versus functional dyspepsia: a systematic review and meta-analysis. *J Psychosom Res* 2024; **183**: 111834.
127. Apputhurai P, Palsson OS, Bangdiwala SI, Sperber AD, Mikocka-Walus A, Knowles SR. Confirmatory validation of the patient health questionnaire - 4 (PHQ-4) for gastrointestinal disorders: a large-scale cross-sectional survey. *J Psychosom Res* 2024; **180**: 111654.
128. Treisman GJ, King JE. Chapter 27 - Psychiatric aspects of gastroparesis. In: McCallum RW, Parkman HP, eds. Gastroparesis: pathophysiology, clinical presentation, diagnosis and treatment. London: Academic Press; 2021: 377-88.
129. Kellar T, Basnayake C, Burgell RE, et al. Disorders of gut–brain interaction, eating disorders and gastroparesis: a call for coordinated care and guidelines on nutrition support. *Med J Aust* 2025; **222**(1): 10-2.
130. Keightley P, Knowles SR. Psychological assessment, formulation, and intervention planning in gastrointestinal cohorts with psychological issues. In: Knowles SR, Keefer L, Mikocka-Walus AA, eds. Psychogastroenterology for Adults: A Handbook for Mental Health Professionals: Taylor & Francis Group; 2019: 172-90.
131. Weusten BLAM, Barret M, Bredenoord AJ, et al. Endoscopic management of gastrointestinal motility disorders - Part 1: European Society of Gastrointestinal Endoscopy (ESGE) Guideline. *Endoscopy* 2020; **52**(6): 498-515.
132. Arts J, Holvoet L, Caenepeel P, et al. Clinical trial: a randomized-controlled crossover study of intrapyloric injection of botulinum toxin in gastroparesis. *Aliment Pharmacol Ther* 2007; **26**(9): 1251-8.
133. Desprez C, Melchior C, Wuestenberghs F, et al. Pyloric distensibility measurement predicts symptomatic response to intrapyloric botulinum toxin injection. *Gastrointest Endosc* 2019; **90**(5): 754-60.e1.
134. Khashab MA, Stein E, Clarke JO, et al. Gastric peroral endoscopic myotomy for refractory gastroparesis: first human endoscopic pyloromyotomy (with video). *Gastrointest Endosc* 2013; **78**(5): 764-8.

135. Vosoughi K, Ichkhanian Y, Benias P, et al. Gastric per-oral endoscopic myotomy (G-POEM) for refractory gastroparesis: results from an international prospective trial. *Gut* 2022; **71**(1): 25.
136. Gregor L, Wo J, DeWitt J, et al. Gastric peroral endoscopic myotomy for the treatment of refractory gastroparesis: a prospective single-center experience with mid-term follow-up (with video). *Gastrointest Endosc* 2021; **94**(1): 35-44.
137. Conchillo JM, Straathof JWA, Mujagic Z, et al. Gastric peroral endoscopic pyloromyotomy for decompensated gastroparesis: comprehensive motility analysis in relation to treatment outcomes. *Endosc Int Open* 2021; **9**(2): E137-E44.
138. Canakis A, Deliwala SS, Frohlinger M, Canakis JP, Adler DG. Long-term outcomes (≥3 years) after gastric peroral endoscopic myotomy for refractory gastroparesis: a systematic review and meta-analysis. *iGIE* 2023; **2**(3): 344-9.e3.
139. Tan J, Wu H, Yang X, et al. A long-term follow-up study of gastric peroral endoscopic myotomy (G-POEM) in a large cohort of patients with postsurgical gastroparesis. *Surg Endosc* 2024; **38**(12): 7416-25.
140. Abdelfatah MM, Noll A, Kapil N, et al. Long-term Outcome of Gastric Per-Oral Endoscopic Pyloromyotomy in Treatment of Gastroparesis. *Clin Gastroenterol Hepatol* 2021; **19**(4): 816-24.
141. Martinek J, Hustak R, Mares J, et al. Endoscopic pyloromyotomy for the treatment of severe and refractory gastroparesis: a pilot, randomised, sham-controlled trial. *Gut* 2022; **71**(11): 2170-8.
142. Gonzalez JM, Mion F, Pioche M, 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* 2024; **56**(5): 345-52.
143. Aziz M, Gangwani MK, Haghbin H, et al. Gastric peroral endoscopic myotomy versus surgical pyloromyotomy/pyloroplasty for refractory gastroparesis: systematic review and meta-analysis. *Endosc Int Open* 2023; **11**(4): E322-E9.
144. McCallum RW, Sarosiek I, Parkman HP, et al. Gastric electrical stimulation with Enterra therapy improves symptoms of idiopathic gastroparesis. *Neurogastroenterol Motil* 2013; **25**(10): 815-e636.