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

Trina Kellar[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]

September 2025

Affiliations:
[1] University of Queensland school of health, Brisbane Australia
[2] Royal Brisbane and Women's Hospital department of gastroenterology, Brisbane Australia
[3] University of Melbourne department of medicine, Melbourne Australia
[4] St Vincent's Hospital Melbourne department of gastroenterology, Melbourne Australia
[5] University of Melbourne school of agriculture, food and ecosystem sciences, Melbourne Australia
[6] Monash University department of gastroenterology and nutrition, Melbourne Australia
[7] Alfred Hospital department of gastroenterology, Melbourne Australia
[8] Royal Melbourne Hospital department of gastroenterology, Melbourne Australia
[9] Western Sydney University school of medicine, Sydney Australia
[10] Parkville Youth Mental Health and Wellbeing Service, Melbourne Australia
[11] Swinburne University department of psychological sciences, Melbourne Australia
[12] University of Adelaide faculty of health and medical sciences, Adelaide Australia
[13] Royal North Shore Hospital department of gastroenterology, Sydney Australia
[14] University of Newcastle, Hunter Medical Research Institute, Newcastle Australia
[15] John Hunter Hospital department of gastroenterology

Abstract

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

Table of Contents

1.1 Scope and Purpose

Gastroparesis has historically been defined as typical upper gastrointestinal (GI) symptoms of post-prandial nausea, vomiting, early satiety and bloating, with delayed gastric emptying, in the absence of mechanical obstruction. This position statement refers specifically to the idiopathic sub-type, where no cause can be identified with traditional diagnostic techniques. Global epidemiological data for idiopathic gastroparesis (IGP) are lacking, and the population prevalence of asymptomatic delayed gastric emptying is unknown (1). Whilst considered a rare disease, IGP appears to be increasing in studies from Western populations (2, 3). When severe, the 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 primarily as a motor disorder, with symptoms attributed to the delayed gastric emptying. This preconception has carved a deep bias in study design, interpretation, and therapeutic pursuits(6).

There is now increasing acceptance that idiopathic gastroparesis 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), and there is substantial overlap with other functional gastroduodenal disorders and eating disorders, in particular chronic nausea vomiting syndrome, and rumination syndrome. Whilst 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 post-prandial satiety, fullness and pain 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 most importantly, and economic cost to healthcare 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 current challenges surrounding idiopathic gastroparesis, 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 recent Rome consensus maintained the historic focus on IGP as a motor disorder and was unable to establish consensus on the majority of recommendations. The above guidelines provide an extensive summary of the literature to date (see: (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.

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 interest within the GESA Luminal Faculty Committee meeting in October 2024 to provide a national standard of care to improve consistency of practice and treatment outcomes and minimise harm across public and private health institutions in Australia. A core working group of 8 members and chair (TK) 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. All positive responses were accepted, with 12 final working group members (reflected in the list of authors) representing neurogastroenterology, nutrition, psychology, and psychiatry. Sections were allocated to authors in their field of expertise, who all contributed meaningfully. The concept was presented to the GESA luminal faculty patient advocacy group via online meeting in August 2025, and one member with lived experience was elected by that group to represent the patient experience. External review of the initial draft was sought from abroad range of experts locally and internationally in nuclear medicine, surgery, psychiatry and dietetics including clinicians with eating disorder expertise, general gastroenterology, neurogastroenterology, intestinal failure, and paediatric gastroenterology (see Acknowledgements). Feedback was incorporated by the working group through multiple revisions before drafting final statements, and consensus achieved as outlined in section 2.2. The document was then presented at World Congress of Gastroenterology@Australian Gastroenterology Week 2025 in Melbourne, September 2025 for public comment, before finalisation.

1.3 Declaration of funding

GESA provided pre-approved financial support for project coordination, graphic design, and medical 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 idiopathic gastroparesis in the adult sector within 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 healthcare support for many clinicians practising in regional Australia, and 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 on the GESA online resources website to reflect current practice.

1.6 Endorsements

Endorsement was sought from the GESA Luminal Faculty Committee for expert review prior to presentation to the GESA Board.

2 Methodology

2.1 Grading of evidence and strength of recommendation

Subsection authors undertook a formal review of the literature using Medline, Embase, Pubmed, CINAHL, and PsychINFO, and hand-search of references. University research librarian support was utilised for the development of PICO questions and search strategies at the discretion of each co-author. Limits applied were peer-reviewed articles, in human adults, published in English, between January 1985 and January 2025. Following the drafting of the statements outlined in section 1.2, 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 as detailed in the supporting references (21-27), and strength of recommendation as Strong or Conditional as per Guyatt *et al* (28). It should be noted that where the quality of supporting evidence was Low or Very Low, this highlights a lack of available high-quality research rather than refuting the statement. A strong recommendation may still be appropriate in poorly researched questions, *en balance*, with expert consensus. The references included in this document to support discussion points represent only a selection of key articles from individual literature reviews.

2.2 Modified Delphi approach

A modified Delphi approach was applied to the 20 drafted statements in September 2025, via anonymous online voting administered by the GESA project support officer. A 4-point Likert scale was used to indicate agreement as follows: Strongly agree, Agree, Disagree, Strongly Disagree, and provided the opportunity for comments. Consensus was deemed to be reached with ≥ 85% strong agreement or agreement with one round of voting, while 80-84% strong agreement or agreement was deemed borderline endorsement (see section 3). 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 AGREE reporting checklist (29).

3 Summary of statements

	Endorsed	Level of Evidence	Strength of Recommendation	Agreement
Statement 1: Idiopathic gastroparesis is a sensorimotor disorder. There is substantial overlap with functional gastroduodenal disorders and eating disorders.	Yes	Low	Strong	**100%** SA – 100% A – 0 % D – 0 % SD – 0 %
Statement 2: A comprehensive medical, surgical, and psychosocial history is needed, including psychological comorbidity and nutritional assessment.	Yes	N/A	Consensus	**100%** SA – 100% A – 0 % D – 0 % SD – 0 %
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.	Yes	Low	Strong	**100%** SA – 75 % A – 25 % D – 0 % SD – 0 %
Statement 4: Initial work-up should include all tests indicated in the clinical context to identify structural GI and systemic diseases.	Yes	N/A	Consensus	**92 %** SA – 67 % A – 25% D – 8 % SD – 0 %
Statement 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 % D – 0 % SD – 0 %
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.	Yes	Low	Strong	**100%** SA – 42 % A – 58 % D – 0 % SD – 0 %
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.	Borderline	Very low	Conditional	**84%** SA – 17 % A – 67 % D – 17 % SD – 0 %
Statement 8: Routine assessment for vascular compression syndromes, hypermobility spectrum disorders, mast cell disorders, autonomic dysfunction, and microbial dysbiosis is not recommended. If suspected, sub-specialist input is recommended to guide appropriate testing and interpretation of test results in the clinical context.	Yes	Low	Strong	**100%** SA – 58 % A – 42 % D – 0 % SD – 0 %
Statement 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 % D – 0 % SD – 0 %
Statement 10: Dietary therapy should prioritise oral nutritional rehabilitation, with the aim of improving symptoms where possible whilst not compromising nutritional status.	Yes	Low	Strong	**100%** SA – 83 % A – 17 % D – 0 % SD – 0 %

Statement 11: Temporary nasogastric tube feeding should only be considered where there is malnutrition, with ongoing weight loss, and medical instability, despite intensive oral nutrition support.	Yes	Low	Strong	**100%** SA – 58 % A – 42 % D – 0 % SD – 0 %
Statement 12: The decision to initiate long-term enteral tube feeding should be made only with formal multidisciplinary team consultation.	Yes	N/A	Consensus	**100%** SA – 75 % A – 25 % D – 0 % SD – 0 %
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.	Yes	Low	Strong	**100%** SA – 75 % A – 25 % D – 0 % SD – 0 %
Statement 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 % SD – 0 %
Statement 15: Limited evidence supports a trial of prokinetic therapy in idiopathic gastroparesis, while the use of antiemetics is largely empirical. Metoclopramide or domperidone are recommended first line treatment.	Yes	Low	Conditional	**100%** SA – 50 % A – 50 % D – 0 % SD – 0 %
Statement 16: Neuromodulators are under-researched in idiopathic gastroparesis, though evidence-based in DGBI. Given the overlap in functional gastroduodenal symptoms, neuromodulators are recommended adjunctive treatment, with choice of agent targeting the predominant GI symptoms.	Yes	Low	Conditional	**100%** SA – 75 % A – 25 % D – 0 % SD – 0 %
Statement 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 % D – 0 % SD – 0 %
Statement 18: Mental health clinicians are recommended core members of the multidisciplinary care team in idiopathic gastroparesis in all individuals with significant psychosocial or psychiatric comorbidity.	Yes	Low	Strong	**100%** SA – 75 % A – 25 % D – 0 % SD – 0 %
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.	Yes	Low	Strong	**100%** SA – 83 % A – 17 % D – 0 % SD – 0 %
Statement 20: There is insufficient evidence to recommend intrapyloric Botox, surgical pyloroplasty, gastric electrical stimulation or G-POEM 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 % SD – 0 %

Table 1. Summary of Statements. Level of evidence and strength of recommendation were rated according to Grading of Recommendations Assessment, Development and Evaluation (GRADE). Agreement was rated as per a modified Delphi consensus approach; see section 2.1 and 2.2. N/A = not applicable. Statements were endorsed when ≥85% strongly agreed or agreed, and deemed borderline when 80-84% strongly agreed or agreed.

4 Pathophysiology

While international guidelines describe gastroparesis as a motor disorder characterised by delayed gastric emptying, it is clear that the 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 IGP 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 associated, feature. Gastric electrical stimulation, which emerged as a treatment for gastroparesis, ameliorates vomiting symptoms unrelated to gastric emptying, implying that symptom improvement results from modulation of neural sensory pathways not gastric emptying(31). Successful medical therapy for idiopathic gastroparesis is associated with normalisation of electrogastrography (EGG), indicating the relevance of gastric dysrhythmias (32)(33, 34). The latter appear linked to reduced numbers of interstitial cells of Cajal (ICC) (35) and fibrosis on full thickness gastric biopsies (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 is often present in patients with IGP, 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 (37). A CT-scan gastric volumetry study indicated that gastroparesis was associated with reduced gastric volume after gaseous distension compared to patients with gastro-oesophageal reflux disease (GORD), while they rated abdominal pain more intensely, suggesting that reduced fundal accommodation could be linked to visceral hypersensitivity in gastroparesis (38). A substantial proportion of gastroparesis patients exhibit abnormal fundic accommodation in barostat studies (39). Gastric scintigraphy and nutrient drink test are more applicable 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 idiopathic gastroparesis, and abdominal pain is a feature in 30% of refractory cases(39). Our understanding of the neuroimmune mechanisms contributing to symptom pathogenesis in DGBI including functional dyspepsia is rapidly evolving, and likely to be relevant to IGP also. Complex immunological pathways underlie peripheral pain sensitization, a hallmark of chronic visceral pain(42-44).

The recognition of gastroparesis as a sensorimotor disorder has important clinical implications, as detailed in the Management section below. IGP patients may benefit from a combination of pharmacologic, dietary and psychological treatments which 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 evidence; Strong recommendation)

5 Assessment

5.1 Overview

Suspected idiopathic gastroparesis (IGP) requires a comprehensive biopsychosocial assessment, given the limited pathophysiological information provided by currently 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 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, particularly opioid, anticholinergic, antimuscarinic, antispasmodic, antipsychotic and centrally acting agents, weight loss agents, cannabinoids, and illicit substances; these may alter gastric emptying and/or exacerbate symptoms. The limited utility of motility testing should be discussed and documented if unable to cease these agents, and persistent pain or addiction specialist support considered if appropriate.

Time should be allowed to explore past and present psychological and neurodevelopmental comorbidity and perpetuating factors within 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 with a mental health clinician is available for clinicians.

Disordered eating as defined by the National Eating Disorders Collaboration includes symptoms and behaviours of eating disorders but at a lesser frequency or lower severity. Eating disorders are characterised by a disturbance of eating and/or related behaviours that results in significant impairment in physical health or psychosocial functioning. In this article 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 as detailed in the Nutrition section below. Eating disorders, DGBI, and delayed gastric emptying are not independent diagnoses and frequently co-exist. For example, 20-80% of eating disorder patients have delayed gastric emptying (46-49), while 95-98% suffer functional GI symptoms(50, 51). Our understanding of the overlap between avoidant restrictive food intake disorder (ARFID), other restrictive eating disorders and DGBI with restricted oral intake is evolving (see (52). A recent meta-analysis found evidence of disordered eating in one third of IGP patients using screening tools (53), but emphasised the tools are prone to overestimating eating disorders in people with gastrointestinal disorders. Given this complexity, co-assessment by a clinician with eating disorder expertise is strongly recommended in all cases where disordered eating behaviours are present.

Statement 2:
A comprehensive medical, surgical, and psychosocial history is needed, including psychological comorbidity and nutritional assessment. (Level 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 evidence; Strong recommendation)

5.3 Initial Investigations:
Initial work-up should exclude structural GI pathology including mechanical gastric outlet obstruction, and relevant systemic diseases in the clinical context, including personal and family history. Alarm features warrant urgent consideration of upper GI 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 haemoglobin, electrolytes, coeliac serology, thyroid function, fasted haematinics, blood glucose, macro- and micronutrient screen.

Helicobacter pylori 'test and treat' eradication is recommended in functional dyspepsia (FD) guidelines dependent on local epidemiology. There are no studies in IGP, but given the overlap with FD 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 GI and systemic diseases. (Level of evidence Not Applicable; Consensus recommendation)

5.4 Measurement of Gastric Emptying:
By definition, the diagnosis of IGP requires the measurement of gastric emptying. Even in the highest quality studies however, the correlation between symptoms and delayed gastric emptying is weak (55), with high individual variability over time irrespective of symptoms (56). This reflects the complexity of sensorimotor abnormalities responsible for symptoms in IGP (see the Pathophysiology section above). 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 4h 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 normal ranges reliant on published values for validated meals and/or local normative values(57). The egg white 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. The working group acknowledged the importance of advocating for one standardised test meal and protocol across Australia, though agreed that this is not possible at present 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 as outlined in the Clinical Assessment section. A large prospective study showed that 42% of those diagnosed with gastroparesis had normal gastric emptying when re-tested at 48 weeks, without a change in symptoms(14). Where modifiable factors are present, the working group suggests consideration be given to repeating an abnormal gastric emptying study within 3-12 months after all confounding factors have been optimised, to improve validity, and

suggests that in the case of discordant results, the better result should be accepted as representative of the capacity of the stomach to empty normally. This statement was not endorsed, achieving a borderline consensus of 84% where 85% agreement was required for endorsement. Disagreement arose from the fundamental limitations of gastric emptying as a measure; given it reflects only one aspect of IGP pathology, 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, and any residual confounding factors should be 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 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(58). A combination of these modalities is likely to provide more accurate assessment given the breadth of the underlying pathology. The working group identifies this as 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 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 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 evidence; Conditional recommendation; Borderline endorsement)

5.5 Further Investigations:
There is increasing public concern surrounding vascular compression syndromes as a potential cause of gastroparesis-like symptoms. Acquired superior mesenteric artery (SMA) syndrome can occur rarely secondary to severe weight loss in this cohort, manifesting as duodenal obstruction by acute angulation between SMA and aorta on specialist imaging (59). It is important to note that an asymptomatic reduced vascular angle is prevalent in population-wide radiological studies (60). Non-invasive weight restoration is the recommended first line treatment, with follow-up imaging if there is ongoing concern. Median arcuate ligament syndrome (MALS), involving coeliac artery compression causing chronic foregut ischaemia, has not been studied specifically in relation to gastroparesis, but again has a high asymptomatic radiological prevalence (61, 62). Routine assessment for vascular compression syndromes is not recommended for symptoms of idiopathic gastroparesis.

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 GI dysmotility. Routine screening is not recommended.

There is insufficient evidence at present to support routine testing for mast cell activation syndrome, autonomic dysfunction, small intestinal bacterial overgrowth, or microbial dysbiosis. Sub-speciality input is required if the above disorders are suspected.

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

6 Management

6.1 Overview

Consistent with the poor correlation between delayed gastric emptying and symptoms, all available treatments that accelerate gastric emptying have demonstrated low efficacy. Acknowledging IGP as a sensorimotor disorder on a spectrum with functional gastroduodenal disorders endorses the adjunctive use of DGBI treatments to target the key symptoms such as nausea or pain. Due to the historic divide of gastroparesis from functional gastroduodenal disorders based on gastric emptying, very few treatments targeting visceral hypersensitivity have been studied specifically in IGP, making the available evidence for these treatments unavoidably low quality. Therefore, whilst 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

Idiopathic gastroparesis should be managed within the biopsychosocial model of care, as outlined in the treatment algorithm below (see: **Figure 1**). If first line treatment is unsuccessful, advice and/or on-referral to a tertiary multidisciplinary team (MDT) is recommended. The core members of the MDT should include representation from gastroenterology, dietetics, psychology and psychiatry, with expertise in neurogastroenterology. Additional representation should be available to the MDT as needed from eating disorder, pain, surgical, and other medical sub-specialities. In regions or healthcare 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 prior to initiating long-term enteral tube feeding, 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 settings.

6.3 Nutritional Management

6.3.1 Nutritional Assessment and Monitoring

Gastroparesis presents unique nutritional challenges requiring a dedicated MDT approach. All patients with gastroparesis should undergo a comprehensive nutritional assessment by a gastrointestinal dietitian at diagnosis and regularly thereafter. The nutritional assessment should include evaluation of current intake of macro- and micronutrients, eating behaviours and patterns, body image, food beliefs, previous dietary interventions, and related quality of life (63). Patients with gastroparesis have high rates of micronutrient deficiencies, including vitamin D (61%), E (80%), folate (68%), calcium (70%), iron (69%), magnesium (72%) and potassium

(86%) (63). 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 develop as an attempt to minimise the symptoms of the IGP, and may change over time in a bidirectional manner. Efficacy of interventions should be assessed using validated tools including body composition, 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 evidence; Strong recommendation)

6.3.2 Dietary interventions

The majority of patients with IGP can and should be managed with oral nutritional rehabilitation. Approximately 58% of patients respond symptomatically to dietary therapy combined with prokinetic medication (64). Dietary therapy should be prescribed by a gastrointestinal dietitian, and may be tailored to symptoms (**Figure 1**) though 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 1** 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 gastroparesis with varying levels of evidence, as summarised in **Table 1**. Detailed sample meal plans for each dietary approach are provided in **Appendix 2** to guide clinicians in practical implementation.

Generally, patients should consume smaller, more frequent meals (6-10 daily) (64-66), ensure food is well-chewed or blended (66), and remain upright for at least 1-2 hours after eating (64, 67). These practical approaches complement individualised dietary modifications, and are part of a strategy termed “effortful eating” (68). Whilst low fat diets have been recommended in some guidelines due to the physiology of fat delaying gastric emptying, there is limited evidence in IGP and the clinical benefit of fat restriction alone is unproven (69)(70). In individuals with malnutrition, caloric restriction is contraindicated.

Although oral nutritional supplements have not been specifically studied in idiopathic gastroparesis, they represent 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, while 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 in patients with anorexia nervosa significantly improved delayed gastric emptying and symptoms (71).

Given the symptom overlap between gastroparesis and functional dyspepsia, dietary strategies used in functional dyspepsia may also be considered. A low-FODMAP diet has limited but emerging evidence for improving epigastric symptoms, early satiety, bloating, and abdominal pain in functional dyspepsia (72, 73). Such a diet may be trialled for a limited period of 6 weeks, with subsequent reintroduction of food groups(74). However, restrictive diets should be avoided in patients with established malnutrition and those at risk, and malnutrition is a contraindication to the low FODMAP diet.

Constipation should be aggressively managed in IGP using standard treatments as reviewed elsewhere (see: (75)), to minimise confounding symptoms and optimise gastrointestinal

motility(76, 77). In addition, slowly fermentable or viscous fibres such as partially hydrolysed guar gum (PHGG) or low dose psyllium may be beneficial. PHGG has shown particular benefit for global pain and functional GI symptoms in IBS (78).

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

6.3.3 Artificial nutritional support considerations

Initiation of enteral tube feeding (ETF) should be approached with caution in patients with idiopathic gastroparesis. The decision to initiate ETF should be made only after referral to a tertiary referral expert centre and consultation with a MDT, as tube feeding does not consistently improve global symptoms and carries risks of iatrogenic harm (79). For patients with medical instability due to severe malnutrition requiring 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 corelate well with symptom severity(8). Principles to guide MDT decision-making regarding ETF are provided in **Table 3**.

Long term ETF should be undertaken only after careful deliberation and consensus within an experienced MDT. In one study of gastroparesis patients, 53% (19/36) of patients who proceeded to nasoduodenal feeding showed no symptom improvement yet were still advanced to PEJ placement (64). Other observational studies published in abstract form indicate that most patients (13/15) with a DGBI undergoing long-term ETF have no improvement in GI symptoms (79), and complication rates are relatively high (64, 79).

Management of pain, visceral hypersensitivity, psychosocial components, and disordered eating behaviour should be addressed in the MDT setting prior to a decision for ETF (68). It should be noted that even tube feeding may result in inconsistent nutritional improvement (64, 79, 80). Long term ETF should be reserved for those only 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 for the reduction of medical risk from malnutrition, rather than as a treatment for 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 nutrition support. (Low evidence; Strong recommendation)

Statement 12:
The decision to initiate long-term enteral tube feeding should be made only with formal multidisciplinary team consultation. (Level 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 evidence; Strong recommendation)

There is currently no evidence supporting parenteral nutrition in 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(81). If ETF is not tolerated due to 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 evidence; Strong recommendation)

Table 2. Dietary approaches for gastroparesis

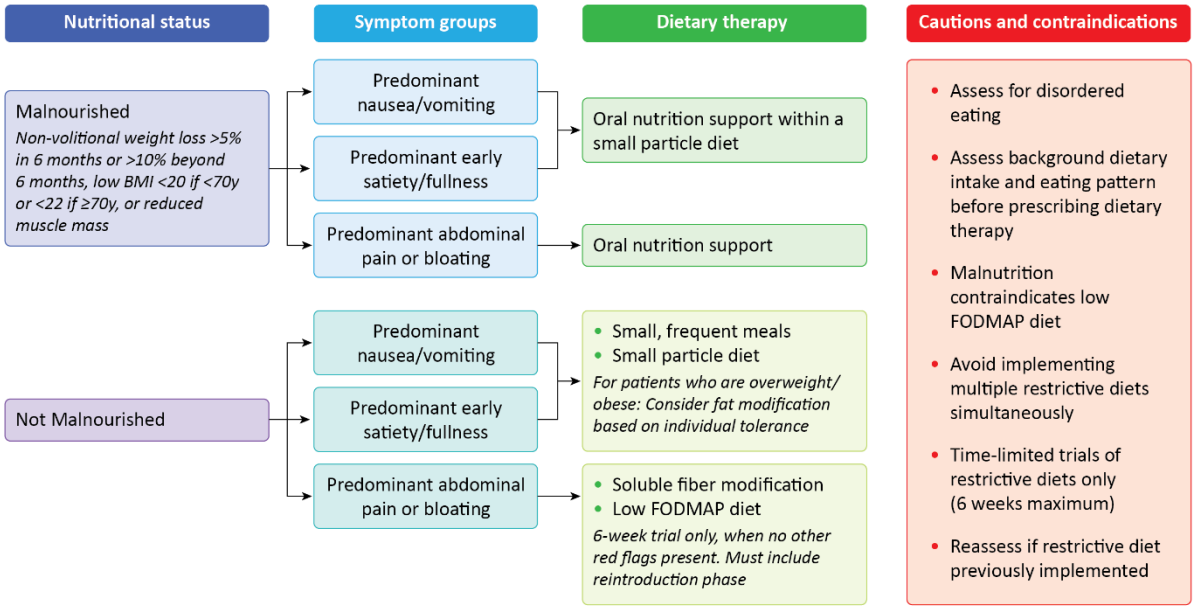
Dietary therapy	Key features	Limitations
Small food particle size	Food mechanically altered to reduce particle size	- Evidence primarily from diabetic gastroparesis - Not a crossover design study, limiting strength of findings - Glycaemic control improvements were not monitored - Definition of "low particle size" inconsistent between studies e.g., rice excluded despite being low particle size
Low FODMAP	Restricts fermentable carbohydrates	- Evidence from functional dyspepsia but not specifically gastroparesis, however significant symptom overlap - No evidence for improving gastric emptying - Contraindicated in malnourished patients
Fibre modification	Selective use of fibres (PHGG, psyllium) [So et al 2021]	- Paradoxical effects: may slow gastric emptying but improve symptoms - Baseline fibre intake usually already low in gastroparesis - PHGG shown to improve IBS symptoms, specifically bloating and pain [(78)]

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 co-existing structural, psychosocial and psychiatric contributors including disordered eating behaviour
Indication	Enteral feeding should be considered only for patients who are severely malnourished with ongoing objective weight loss despite MDT oral nutritional rehabilitation efforts.
Symptom management	ETF is indicated for nutritional support in severe malnutrition, not primarily for symptom relief.
Risk-benefit assessment	The risks of tube feeding (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 prior to initiating tube feeding.
Weight considerations	For patients with high body weight who have experienced significant recent weight loss (>10% within 6-month timeframe), the risk of malnutrition complications versus risks of invasive intervention must be carefully balanced.

MDT: multidisciplinary team; ETF: enteral tube feeding

Figure 1. Decision framework for nutritional recommendations for gastroparesis. This framework guides individualised nutritional management based on nutritional status and predominant symptoms. *See Appendix 1* for small particle and texture modified meal plans.



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 generalized to IGP (82, 83). A network meta-analysis of 29 trials in gastroparesis of any aetiology indicated symptom benefit over placebo for dopamine antagonists(84), while a meta-analysis of 29 trials of prokinetics in functional dyspepsia indicated global symptom benefit(85).

Metoclopramide and domperidone are the only prokinetics approved for gastroparesis in Australia. Only one of 4 placebo-controlled trials of metoclopramide in gastroparesis included idiopathic patients, and showed symptomatic improvement after 3 weeks(86). Adverse effects may include acute dystonia, prolonged QT interval, and tardive dyskinesia. Domperidone does not cross the blood brain barrier, reducing neurological side effects, though may induce QT prolongation. Despite favourable evidence in diabetic gastroparesis(87), only one of 6 placebo-controlled trials of domperidone included IGP patients, but did show symptom benefit(88).

Use of prucalopride and erythromycin is off-label. Erythromycin accelerated gastric emptying acutely and improved symptoms in IGP in an uncontrolled study, but the prokinetic effect was diminished after 4 weeks, limiting its long-term utility(89). Prucalopride, approved for use in constipation and devoid of cardiac effects, improved symptoms and gastric emptying over placebo in one 4-week double-blind crossover study involving predominantly IGP patients(90). Cisapride, a 5HT4 agonist, was withdrawn due to a risk of prolonged QT arrhythmias.

Antiemetics such as phenothiazines (eg. prochlorperazine) and antihistamines (eg. promethazine, cyclizine) are used empirically in IGP(87). Intravenous administration of cyclizine can induce euphoria and dependence(91). Haloperidol was superior to placebo for nausea in emergency presentations in gastroparesis patients(92). The 5HT3 antagonist granisetron, via transdermal patches, improved nausea and vomiting in open label studies in IGP(93, 94). While the NK-1 receptor antagonist aprepitant reduced ‘gastroparesis-like’ nausea and vomiting over

placebo, this was insufficient to satisfy the pre-specified primary outcome(95). The safety profile of all medications must be confirmed specific to 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 and domperidone are recommended first line. (Low evidence; Conditional recommendation)

6.4.2 Pharmacological neuromodulation

Though used widely, few studies have assessed neuromodulator medications in IGP. The only placebo-controlled randomised controlled trial (RCT) – the NORIG trial (2013) – allocated 130 patients with IGP to escalating nortriptyline (10 to 75mg) versus placebo over 15 weeks, and found no difference in the proportion experiencing 50% reduction in global gastroparesis cardinal symptom index (GCSI) scores (23% benefit with nortriptyline, 21% with placebo). Nearly half the participants failed dose escalation due to medication intolerance, while 29% from the treatment group and 9% from the placebo group stopped treatment, despite equal reported adverse events (96).

While amitriptyline has not been formally tested in IGP, placebo-controlled trials have demonstrated global symptom benefit in functional dyspepsia (97). One RCT of 292 patients included 21% with delayed gastric emptying. Amitriptyline 50mg daily over 12 weeks improved FD symptoms, whilst escitalopram 10mg daily did not, and those with delayed emptying were less likely to improve global scores (69). Neither nortriptyline nor amitriptyline induced any further delay in gastric emptying (98) (99).

An open-label trial of mirtazapine 15mg daily in 30 patients with IGP found improvements in nausea, vomiting and appetite at 2 and 4 weeks, although 20% terminated treatment due to adverse effects(100). Another 8 week RCT showed improvement in post-prandial symptoms of functional dyspepsia with mirtazapine 15mg daily, although gastric emptying was not measured (101).

The BESST trial (2023) compared buspirone to placebo over 4 weeks 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 (102).

The atypical antipsychotic medications olanzapine and quetiapine are used as adjunctive therapy for functional nausea, and serotonin noradrenaline reuptake inhibitors for unexplained pain, though have not been studied in IGP.

We recommend the use of neuromodulators in IGP as second line therapy (see **Figure 2**). 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 GI symptoms. The Rome Foundation 2018 report details the pharmacology, symptom targets, and precautions when prescribing neuromodulators in DGBI (103).

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

6.4.3 Cannabinoids

Patients with gastroparesis frequently use cannabinoids (46% in one study), with the majority perceiving symptom relief (104, 105). However, cannabinoid use remains controversial as they retard gastric emptying (106), and large epidemiological studies (n = 41,374) indicate they are is associated with higher healthcare utilisation(107) (108). A single placebo-controlled RCT of cannabidiol in patients with gastroparesis (n = 44 during) found a reduction in global GCSI and vomiting episodes. The cannabidiol group tolerated higher volumes satiation tests despite slower gastric emptying (109). A second uncontrolled prospective study (n = 24) found similar symptomatic improvements, although no physiologic endpoints were examined (110).

Statement 17:
Cannabinoids slow gastric emptying but paradoxically may improve symptoms of gastroparesis including satiation. There is insufficient evidence to recommend their use. (Low 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 post-surgical gastroparesis, which found that psychosocial support, music and massage therapy, and family psychoeducation improved mood and improved residual gastric volume compared to standard medical care(111). Our acceptance of the established overlap with functional dyspepsia enables evidence from DGBI to be applied to patients with IGP (refer to (112-118), noting that psychological therapies are under-researched and under-utilised even in DGBI, despite the acceptance of brain-gut behavioural therapy (116). 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 (119).

Regardless, input from psychologists and psychiatrists is often indicated due to the high co-occurrence of mental health disorders. Gastroparesis is associated with significant psychosocial burden and low quality of life (120-123). Anxiety and depression have a reported pooled prevalence of 49% and 39%, respectively(124), which is notably higher than the prevalence of 27.8% and 27.0% in DGBI (see: (125). There is a strong evidence base for psychological interventions in the disorders of mood, sleep, personality, trauma, eating, and persistent pain which frequently co-occur in this cohort. As such, cognitive behaviour therapy (CBT), hypnosis, mindfulness-based stress reduction (MBSR), and acceptance and commitment-based therapy (ACT) are likely to benefit. In the absence of targeted therapy, psychotherapy provides support and neuromodulation which is beneficial to all patients living with chronic gastroduodenal symptoms, with the aim of improving symptoms, tolerance and quality of life.

Experience of trauma, personality vulnerabilities and abnormal illness beliefs, can also impact significantly on therapeutic outcomes(121, 126). 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 health practitioners may experience in the face of chronic illness.

Comorbid eating disorders are common in GI disorders (refer to (48, 52)), but there is a lack of high-quality research to guide management. Historic IGP guidelines have recommend exclusion of eating disorders, omitting guidance for those with co-existing disorders (19, 20) . When disordered eating or an eating disorder is present in patients with IGP, we highlight the

importance of gastroenterologists working closely with eating disorder clinicians to co-assess and co-manage these patients in order to optimise outcomes(127).

Mental health clinicians provide pivotal assessment and formulation of how these mental health issues intersect with IGP, in addition to psychoeducation and psychotherapy. Additionally, psychiatrists provide psychotropic medication expertise(128). Mental health clinicians with expertise in GI conditions are few 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 in idiopathic gastroparesis in all individuals with significant psychosocial or psychiatric comorbidity. (Low 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 evidence; Strong recommendation)

6.6 Interventional Therapies

The European Society for Gastrointestinal Endoscopy 2020 consensus recommends against the use of pyloric botulinum toxin (Botox) injection in unselected patients, and also against its use as a screening test for further pyloric interventions(129). A randomised sham-controlled cross over trial found no improvement in either gastric emptying or symptoms (130). One pilot study reported that pyloric distensibility measured by an endoscopic functional luminal imaging probe (EndoFLIP) predicted symptomatic response to intrapyloric Botox, but further data are needed(131).

Gastric peroral endoscopic myotomy (G-POEM) has emerged as a promising, minimally invasive therapeutic option to reduce pyloric resistance to gastric emptying(132). Initial studies suggest significant symptom relief and improved gastric emptying in refractory gastroparesis. Three non-randomised trials showed success rates of 58–60% at 6 months(133-135), with long term success varying from 75% at 3 years(136) to 87% at 5 years(137). High BMI, longer duration of gastroparesis, psychiatric comorbidity, and narcotic medication use have been associated with poor outcomes(138). Only one sham-controlled RCT has been published to date, including 41 patients (17 diabetic, 13 post-surgical, and 11 idiopathic). Of the 21 patients randomised to G-POEM, 71% benefited, with 50% global GCSI symptom reduction and improved gastric emptying 6 months post-procedure, versus 22% with the sham procedure(139). Subgroup analysis was inconclusive in those with IGP. Moreover, a RCT comparing Botox to G-POEM found no difference in clinical success rate or gastric emptying times (140), while a meta-analysis of G-POEM versus surgical pyloroplasty suggested similar clinical outcomes, but greater cost effectiveness with G-POEM (141). 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.

Gastric electrical stimulation was not superior to placebo in IGP, with no difference in vomiting when randomised to stimulation on or off in a blinded fashion(142). However, a 4 month double-blind sham-controlled RCT of 133 patients with refractory vomiting – 78% of whom had gastroparesis, though percentage with IGP 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 Botox, surgical pyloroplasty, gastric electrical stimulation or G-POEM in medically refractory idiopathic gastroparesis. These therapies should only be trialled following multidisciplinary team consensus. (Low Evidence; Conditional recommendation)

Figure 2. Idiopathic Gastroparesis Treatment Algorithm

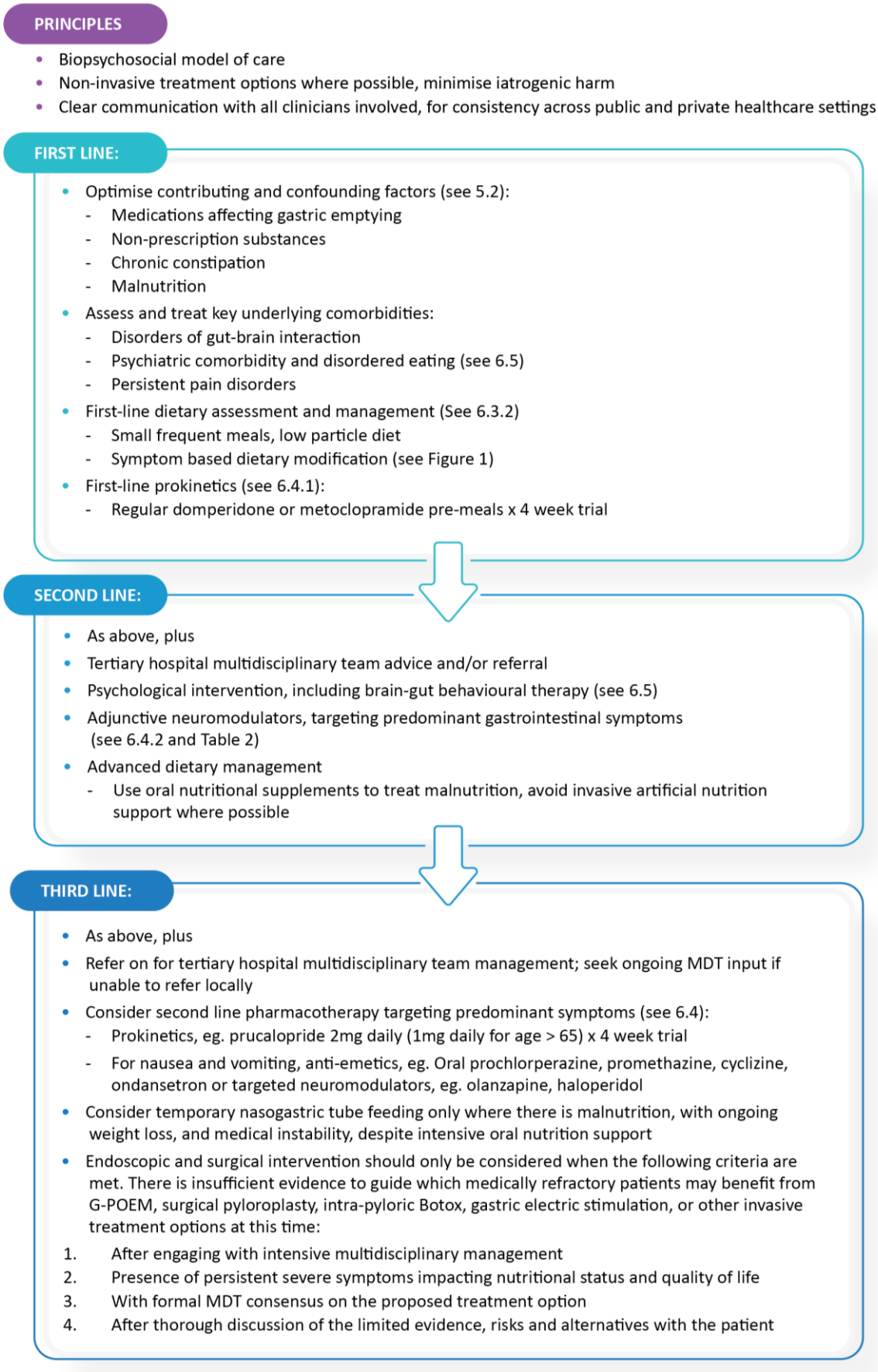


Table 4. Summary of Gut-Brain Neuromodulators by Class, Mode of Action, Actions on Gastrointestinal Sensorimotor Function, Relevance to Gastrointestinal Symptom, and Side Effects. Adapted from Drossman *et al*, Neuromodulators for functional gastrointestinal disorders (Disorders of gut-brain interaction): a Rome Foundation working team report published 2018 (103).

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-HT_2, 5-HT_3, 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	Pain reduction. Best documented for IBS, but also FD (EPS). Potential usefulness in all FGIDs where pain is a prominent feature. Side effect profile can be useful in order to reduce diarrhea and improve sleep.	Drowsiness, dry mouth, constipation, sexual dysfunction, arrhythmias, and weight gain
SSRI Citalopram, escitalopram, fluoxetine, paroxetine, sertraline	Presynaptic SRI.	Motility: enhancement of gastric and small bowel propulsive motility Sensitivity: no major impact on visceral sensitivity in healthy subjects or patients with FGIDs	Treatment of associated anxiety, phobic features, and OCD in FGIDs.	Agitation, diarrhea, insomnia, night sweats, headache, weight loss, and sexual dysfunction.
SNRI Duloxetine, milnacipran, venlafaxine	Pre-synaptic SRI and NRI. Equally strong for duloxetine. NRI for venlafaxine in higher doses. Milnacipran stronger NRI than SRI effects.	Motility: inhibitory effect on gastric and colonic tone, but not to the degree of TCAs; more studies are needed Sensitivity: few studies available; area requiring further research	Treatment of associated pain (based on efficacy in fibromyalgia, back pain, and headache) in FGIDs. Potential use for painful FGIDs; however, formal evidence in treatment of specific FGID-related pain is lacking.	Nausea, agitation, dizziness, sleep disturbance, fatigue, and liver dysfunction
NA and specific serotonergic antidepressants Mirtazapine, mianserin, trazodone	Indirect effects resulting in increased NA and serotonergic activity through α2 antagonism on NA and 5-HT neurons. Also 5-HT_2, 5-HT_3, H1, muscarinic-1 antagonism	Motility: lack of detailed studies Sensitivity: lack of detailed studies	Potential use for treatment of early satiation, weight loss, and chronic nausea/vomiting. Side effect profile can be useful to improve sleep.	Sedation, headache, dry mouth, and weight gain
Azapirones Buspirone, tandospirone	Partial pre- and post-synaptic 5-HT_1 agonists	Motility: enhanced esophageal contractions and increased gastric accommodation in health and FD Sensitivity: limited data suggest no effect	Treatment of associated anxiety. Potential use for treatment of early satiety, fullness. and nausea, but consistent evidence in FGIDs is lacking.	Sedation, headache, and vertigo
Atypical antipsychotics Aripiprazole, levosulpiride, olanzapine, quetiapine, sulpiride	D_2 receptor antagonism as main mechanism. Partial D_2 agonism for the sulpirides. Various profiles of 5-HT_{2A} antagonism (olanzapine, quetiapine), 5-HT_{1A} agonism (quetiapine), H1, α1, α2, muscarinic-1 receptor antagonism.	Motility: lack of data Sensitivity: limited data suggest decreased gastric sensitivity in functional dyspepsia	Potential use in augmentation for pain reduction; however, formal evidence in treatment of specific FGID pain currently lacking. Low evidence in FGIDs. Potential use of sulpirides for nausea and dyspepsia, but formal evidence is lacking. Improved sleep.	Sedation, dizziness, weight gain, hyperlipidemia, and diabetes
Delta ligand agents Gabapentin, pregabalin,	α2δ subunit blockage of (mostly presynaptic) voltage-sensitive calcium channels	Motility: no 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.

NRI, noradrenaline reuptake inhibitor; SRI, serotonin inhibitor.

7 Conclusion

Here we present the first Australian position statement on the assessment and management of idiopathic gastroparesis. In contrast to prior guidelines, the expert panel proposes that gastroparesis should be considered a chronic sensorimotor disorder rather than an isolated motility disorder. Twenty statements were developed and refined by consensus and given a GRADE of evidence and strength of recommendation based on current 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 reliance on gastric emptying as the defining feature of idiopathic gastroparesis is problematic. It may translate to suboptimal management, and has constrained new therapeutic developments.

A novel recommendation from this consensus is the application of treatments established in functional dyspepsia and disorders of gut-brain interaction 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 idiopathic gastroparesis, multidisciplinary care is advised. Specialist tertiary multidisciplinary team input is recommended as standard of care where 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 and/or antiemetic medication is recommended in practice, combined with formal dietary assessment and management, although evidence for specific agents is limited. This consensus recommends a symptom-based approach to the adjunctive use of treatments established in the overlapping disorders of gut-brain interaction, persistent pain, and psychiatric disorders, which commonly overlap with IGP. Psychological support is recommended early, with mental health clinicians forming a core part of the treatment team in 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.

The authors have identified several key areas for future development in this document. These include: the need to broaden our current research focus to elucidate the complex pathophysiology of symptom genesis in IGP, beyond delayed gastric emptying, to develop novel therapeutic targets; defining a combination of testing modalities which phenotype the sensorimotor pathology of IGP with greater accuracy; the need for medically endorsed patient educational material to help combat the impact of online misinformation; and the need for a shift in funding models in public hospitals in Australia, to facilitate multidisciplinary care including mental health clinicians as national standard of care in these complex disorders.

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 idiopathic gastroparesis, to incorporate the many pathophysiological mechanisms now established, and the recognition of IGP as a sensorimotor disorder. Employing this understanding will enable us to re-focus research towards the development of novel targeted therapies, to improve the lives of individuals living with this challenging disorder, long overdue.

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

Special thanks are extended to the GESA Luminal Faculty Patient Advocacy Group for your vital feedback incorporated into this document to ensure representation of the patient experience.

Jane Andrews, Gastroenterologist
Jamee Barugh, Nutrition
Andrew Buckle, Gastroenterologist
Chris Cederwell, 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, Nutrition
Lee Martin, Nutrition (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, Nutrition
- Abhinav Vasudevan, Gastroenterologist

APPENDICES

Appendix 1

Table 5. Nutritional outcomes of enteral feeding in gastroparesis: Evidence summary

Author, year, location	Population and study design	Population
Gallo, 2023, Australia (abstract only) (79)	Disorders of the Gut Brain Interaction Retrospective N=15	6 (40%) patients experienced weight gain post tube insertion, six (40%) had no weight change and three (20%) experienced weight loss.
Martin, 2023, UK (abstract only) (80)	Disorders of the Gut Brain Interaction Retrospective N= 15	8/15 continued long term enteral feeding (median 4.3 years) although 3 (out of 6 at admission) remained underweight (BMI <18.5kg/m^2. 7/15 discontinued enteral feeding after a median 0.3 (IQR 0-1.5) years and 1 (out of 3 at admission) remained underweight.
Strijbos, 2019, Nethe lands (64)	Gastroparesis Retrospective, n= 86 Diabetes 26%, post-surgical 27%, idiopathic 38%, generalised motility disorder 8%	36/86 commenced 3 months of nasoduodenal enteral feeding after not responding to diet and prokinetic therapy. Weight gain occurred regardless of symptom improvement (17/36 were symptomatic responders, gaining a mean 2.5 kg (p = 0.018) from baseline, compared to19/36 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 and this did not differ between those whose symptoms responded to PEG-J or not.

Appendix 2

Table 6. Meal Plan Summary: soft, small particle diet

Meal	Food	Nutrients (kJ, Protein, Carbs, Fat, Fibre)
Breakfast	Quick oats (1/2 cup dry) Low-fat milk (3/4 cup) Mashed banana (1/2 banana) Nut spread (2 tsp)	1.5MJ 15g protein 45g CHO 12g fat 6 g fibre
Morning Tea	Greek yogurt (1/2 cup, regular fat) Stewed apples (1/2 cup)	1.1MJ 9g prot 39g CHO 7g fat 2g fibre
Lunch	Lean minced beef (3/4 cup cooked) Mashed potatoes (1/2 cup) Mashed Pumpkin (1/4 cup mashed) Mashed Carrots (1/4 cup mashed), Gravy (1 tbsp)	2.0 MJ 35g prot 20g CHO 28g fat 5 g fibre
Afternoon Tea	Whole-meal bread (1 slice) Hommus 2 tbsp	0.4MJ 4.5 g prot 14g CHO 2g fat 3 g fibre
Dinner	Baked White fish (85g) Mashed sweet potato (3/4 cup) Mashed broccoli (1/2 cup) Olive oil (1 tsp)	1.44MJ 25g prot 37g CHO 6g Fat 7g fibre
Supper	Greek yogurt (1/2 cup, plain, 2%) Peaches in juice (1/2 cup)	.9 MJ 7g protein 38g CHO, 6 fat 1g fibre

Nutritional analysis: Energy: 7.3MJ, Protein: 97g (22.6% of energy), Carbohydrates: 188 g (42.71% of energy), Fat: 60g (30% of energy), Fibre: 24g, Iron:11 mg, B12: 7mg, Zinc: 14.0 mg, Folate: 399 µg, Vitamin C: 139 mg, Calcium: 1,013 mg.

Table 7. Meal Plan Summary: Texture modified diet which includes liquids

Meal	Foods	Nutrients (per meal)
Breakfast	Scrambled eggs (2 eggs) Whole meal bread (1 slice)	1.3MJ 18g protein 15g carbs 19g fat 2g fibre
Morning Tea	Smoothie: Skim milk powder (10g) Mashed banana (1/2 banana) Greek yogurt (1/2 cup, plain, 2%) Low-fat milk (1/2 cup)	1.1MJ 20g protein 34g carbs 5g fat 4g fibre
Lunch	Minced meat (1/3 cup cooked), Mashed potatoes (1/2 cup), Mashed Pumpkin (1/4 cup mashed), Mashed Carrots (1/4 cup mashed),	1.5MJ 17 g protein, 20g carbs 22g fat g fibre
Afternoon Tea	Sustagen (250ml)	0.9 kJ 13g protein 30g carbs 5 g fat 0 g fibre
Dinner	Puree soup made of: Mashed sweet potato (3/4 cup), Mashed broccoli (1/2 cup), Olive oil (1 tsp), Purée chicken (1/4 cup) Purée spinach (1/4 cup)	1.7MJ 25g protein 27g carbs 21g fat 8 g fibre
Supper	Purée fruit (3/4 cup)	0.7MJ 6g protein 35g carbs 1g fat 6g fibre

Nutritional analysis: Energy: 7.2MJ, Protein: 92g (22% energy), Carbs: 160g (44% energy), Fat: 74g (37% energy), Fibre: 24g, Iron: 15mg, B12: 6.7ug, Zinc: 10mg, Folate: 221ug, Vitamin C: 99mg, Calcium: 1151mg.

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, Moses PL, Almansa C, Bennett D, 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, Barbara G, Morselli-Labate AM, Monetti N, 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, Hasler WL, Abell TL, Unalp-Arida A, 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, Abell TL, Bernard CE, Koch KL, 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, Mulak A, Serra J, Enck P, 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, Drug V, Mulak A, Serra J, 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, Vaughn VM, Petrey J, Greer K, et al. ACG Clinical Guideline: Gastroparesis. Am J Gastroenterol. 2022;117(8):1197-220.
20. Schol J, Huang IH, Carbone F, Fernandez LMB, Gourcerol G, Ho V, et al. Rome Foundation and international neurogastroenterology and motility societies' consensus on idiopathic gastroparesis. The Lancet Gastroenterology & Hepatology. 2025;10(1):68-81.

21. Balshem H, Helfand M, Schünemann HJ, Oxman AD, Kunz R, Brozek J, 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, Mustafa RA, Rylance J, Foroutan F, et al. Core GRADE 1: overview of the Core GRADE approach. BMJ. 2025;389:e081903.
23. Guyatt G, Zeng L, Brignardello-Petersen R, Prasad M, De Beer H, Murad MH, 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, De Beer H, Prasad M, Murad MH, et al. Core GRADE 3: rating certainty of evidence-assessing inconsistency. BMJ. 2025;389:e081905.
25. Guyatt G, Wang Y, Eachempati P, Iorio A, Murad MH, Hultcrantz M, 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, Owen A, Agoritsas T, Murad MH, et al. Core GRADE 5: rating certainty of evidence-assessing indirectness. BMJ. 2025;389:e083865.
27. Guyatt G, Yao L, Murad MH, Hultcrantz M, Agoritsas T, De Beer H, et al. Core GRADE 6: presenting the evidence in summary of findings tables. BMJ. 2025;389:e083866.
28. Guyatt G, Vandvik PO, Iorio A, Agarwal A, Yao L, Eachempati P, 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, Abell TL, Koch KL, McCallum RW, 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, Fontaine S, Bruley Des Varannes S, Zerbib F, 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, Lahr C, Lammers W, Windsor JA, 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, Waite S, Schamberg G, Daker 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, Ramsey FV, Diaz JR, Bennett PA, 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? Nature Reviews Immunology. 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, Stengel A, Giel KE, Zipfel S, et al. Which Symptoms, Complaints and Complications of the Gastrointestinal Tract Occur in Patients With Eating Disorders? A Systematic Review and Quantitative Analysis. Frontiers in Psychiatry. 2020;11.
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. European Eating Disorders Review. 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, Dupont C, Aupetit A, Achamrah N, 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: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. Pasricha PJ, Grover M, Yates KP, Abell TL, Bernard CE, Koch KL, et al. Functional Dyspepsia and Gastroparesis in Tertiary Care are Interchangeable Syndromes With Common Clinical and Pathologic Features. Gastroenterology. 2021;160(6):2006-17.
57. Innovation NAfC. Gastric emptying scintigraphy: Clinical Practice Guide. Sydney: ACI; 2023.
58. Carson DA, Bhat S, Hayes TCL, Gharibans AA, Andrews CN, O'Grady G, 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.
59. Oka A, Awoniyi M, Hasegawa N, Yoshida Y, Tobita H, Ishimura N, et al. Superior mesenteric artery syndrome: Diagnosis and management. World J Clin Cases. 2023;11(15):3369-84.

60. 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. Rev Esp Enferm Dig. 2003;95(7):485-9, 0-4.
61. 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.
62. Terlouw LG, Moelker A, Abrahamsen J, Acosta S, Bakker OJ, Baumgartner I, 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.
63. Parkman HP, Yates KP, Hasler WL, Nguyan L, Pasricha PJ, Snape WJ, et al. Dietary intake and nutritional deficiencies in patients with diabetic or idiopathic gastroparesis. Gastroenterology. 2011;141(2):486-98, 98.e1-7.
64. Strijbos D, Keszthelyi D, Smeets FG, Kruimel J, Gilissen LP, de Ridder R, 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.
65. Limketkai BN, LeBrett W, Lin L, Shah ND. Nutritional approaches for gastroparesis. Lancet Gastroenterol Hepatol. 2020;5(11):1017-26.
66. 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.
67. 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.
68. Paine P, McMahon M, Farrer K, Overshott R, Lal S. Jejunal feeding: when is it the right thing to do? Frontline Gastroenterol. 2020;11(5):397-403.
69. 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.
70. Mogilevski T, Rosella S, Nguyen A, Fitzpatrick J, Parker F, Halmos EP, 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.
71. 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.
72. 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.
73. 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.
74. 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.
75. Serra J, Pohl D, Azpiroz F, Chiarioni G, Ducrotté P, Gourcerol G, et al. European society of neurogastroenterology and motility guidelines on functional constipation in adults. Neurogastroenterol Motil. 2020;32(2):e13762.
76. 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.

77. Tjeerdsma HC, Smout AJ, Akkermans LM. Voluntary suppression of defecation delays gastric emptying. Dig Dis Sci. 1993;38(5):832-6.
78. 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.
79. Gallo R, Armstrong E, Griffin H, Russell D, Hebbard G. Impact of enteral tube feeding for individuals with disorders of the gut brain interaction. Clinical Nutrition ESPEN. 2023;58:604.
80. Martin LD, Wang X, Day C, Aljanahi A, Emmanuel AV, Gabe SM, et al. Enteral tube feeding in functional gastrointestinal disorders: A game of chance? Clinical Nutrition ESPEN. 2023;57:849.
81. 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.
82. Janssen P, Harris MS, Jones M, Masaoka T, Farré R, Törnblom H, 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.
83. 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.
84. 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.
85. Pittayanon R, Yuan Y, Bollegala NP, Khanna R, Lacy BE, Andrews CN, et al. Prokinetics for Functional Dyspepsia: A Systematic Review and Meta-Analysis of Randomized Control Trials. Am J Gastroenterol. 2019;114(2):233-43.
86. 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.
87. Camilleri M, Parkman HP, Shafi MA, Abell TL, Gerson L. Clinical guideline: management of gastroparesis. Am J Gastroenterol. 2013;108(1):18-37; quiz 8.
88. 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.
89. 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.
90. Carbone F, Van den Houte K, Clevers E, Andrews CN, Papathanasopoulos A, Holvoet L, et al. Prucalopride in Gastroparesis: A Randomized Placebo-Controlled Crossover Study. Am J Gastroenterol. 2019;114(8):1265-74.
91. Bailey F, Davies A. The misuse/abuse of antihistamine antiemetic medication (cyclizine) by cancer patients. Palliat Med. 2008;22(7):869-71.
92. 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.
93. 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.
94. Simmons K, Parkman HP. Granisetron transdermal system improves refractory nausea and vomiting in gastroparesis. Dig Dis Sci. 2014;59(6):1231-4.
95. Pasricha PJ, Yates KP, Sarosiek I, McCallum RW, Abell TL, Koch KL, 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.
96. Parkman HP, Van Natta ML, Abell TL, McCallum RW, Sarosiek I, Nguyen L, et al. Effect of nortriptyline on symptoms of idiopathic gastroparesis: the NORIG randomized clinical trial. JAMA. 2013;310(24):2640-9.

97. 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.
98. Lacy BE, Saito YA, Camilleri M, Bouras E, DiBaise JK, Herrick LM, et al. Effects of Antidepressants on Gastric Function in Patients with Functional Dyspepsia. Am J Gastroenterol. 2018;113(2):216-24.
99. Talley NJ, Locke GR, Saito YA, Almazar AE, Bouras EP, Howden CW, et al. Effect of Amitriptyline and Escitalopram on Functional Dyspepsia: A Multicenter, Randomized Controlled Study. Gastroenterology. 2015;149(2):340-9.e2.
100. 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.
101. 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.
102. Parkman HP, Yates KP, Sarosiek I, Bulat RS, Abell TL, Koch KL, 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.
103. 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.
104. Jehangir A, Parkman HP. Cannabinoid Use in Patients With Gastroparesis and Related Disorders: Prevalence and Benefit. Am J Gastroenterol. 2019;114(6):945-53.
105. Parkman HP, Sharkey EP, Nguyen LA, Yates KP, Abell TL, Hasler WL, et al. Marijuana Use in Patients with Symptoms of Gastroparesis: Prevalence, Patient Characteristics, and Perceived Benefit. Dig Dis Sci. 2020;65(8):2311-20.
106. Camilleri M, Zheng T. Cannabinoids and the Gastrointestinal Tract. Clin Gastroenterol Hepatol. 2023;21(13):3217-29.
107. Kilani Y, Gonzalez Mosquera DA, Puelo PC, Aldiabat M, Ruffle JK, Madi MY, et al. The Impact of Cannabis Use in Gastroparesis: A Propensity-Matched Analysis of 41,374 Gastroparesis Patients. Am J Gastroenterol. 2025.
108. Parkman HP, Xin Y, Wilson LA, Burton-Murray H, McCallum RW, Sarosiek I, et al. Characterization of Patients With Symptoms of Gastroparesis Having Frequent Emergency Department Visits and Hospitalizations. Clin Gastroenterol Hepatol. 2025.
109. Zheng T, BouSaba J, Taylor A, Dilmaghani S, Busciglio I, Carlson P, 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.
110. 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.
111. 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.
112. 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.
113. 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.
114. 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.
115. Ballou S, Vasant DH, Guadagnoli L, Reed B, Chiarioni G, Ten Cate 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.

116. 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.
117. Knowles SR, Keefer L, Mikocka-Walus AA. Psychogastroenterology for Adults: A Handbook for Mental Health Professionals: Taylor & Francis; 2019.
118. Goodoory VC, Khasawneh M, Thakur ER, Everitt HA, Gudleski GD, Lackner JM, 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.
119. Dendrinos E, Kim H, Hubik D, Green J, Yang L, Stanley A, et al. Clinical outcomes of patients seen by psychiatrists in a multidisciplinary clinic for disorders of gut-brain interaction. Intern Med J. 2025.
120. Simons M, Cline M. Scoping review: the social and emotional impacts of gastroparesis. Transl Gastroenterol Hepatol. 2024;9:42.
121. 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.
122. 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.
123. Woodhouse S, Hebbard G, Knowles SR. Psychological controversies in gastroparesis: A systematic review. World J Gastroenterol. 2017;23(7):1298-309.
124. 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.
125. 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.
126. Treisman GJ, King JE. Chapter 27 - Psychiatric aspects of gastroparesis. In: McCallum RW, Parkman HP, editors. Gastroparesis: Academic Press; 2021. p. 377-88.
127. Kellar T, Basnayake C, Burgell RE, Kamm M, Kim HW, Lane K, 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.
128. Keightley P, Knowles SR. Psychological assessment, formulation, and intervention planning in gastrointestinal cohorts with psychological issues. In: Knowles SR, Keefer L, Mikocka-Walus AA, editors. Psychogastroenterology for Adults: A Handbook for Mental Health Professionals: Taylor & Francis Group; 2019. p. 172-90.
129. Weusten B, Barret M, Bredenoord AJ, Familiari P, Gonzalez JM, van Hooft JE, et al. Endoscopic management of gastrointestinal motility disorders - part 2: European Society of Gastrointestinal Endoscopy (ESGE) Guideline. Endoscopy. 2020;52(7):600-14.
130. Arts J, Holvoet L, Caenepeel P, Bisschops R, Sifrim D, Verbeke K, 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.
131. Desprez C, Melchior C, Wuestenberghs F, Zalar A, Jacques J, Leroi AM, et al. Pyloric distensibility measurement predicts symptomatic response to intrapyloric botulinum toxin injection. Gastrointest Endosc. 2019;90(5):754-60.e1.
132. Khashab MA, Stein E, Clarke JO, Saxena P, Kumbhari V, Chander Roland B, et al. Gastric peroral endoscopic myotomy for refractory gastroparesis: first human endoscopic pyloromyotomy (with video). Gastrointest Endosc. 2013;78(5):764-8.
133. Vosoughi K, Ichkhanian Y, Benias P, Miller L, Aadam AA, Triggs JR, et al. Gastric per-oral endoscopic myotomy (G-POEM) for refractory gastroparesis: results from an international prospective trial. Gut. 2022;71(1):25.
134. Gregor L, Wo J, DeWitt J, Yim B, Siwiec R, Nowak T, 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.

135. Conchillo JM, Straathof JWA, Mujagic Z, Brouns JH, Bouvy ND, Keszthelyi D, et al. Gastric peroral endoscopic pyloromyotomy for decompensated gastroparesis: comprehensive motility analysis in relation to treatment outcomes. Endoscopy international open. 2021;9(02):E137-E44.
136. 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.
137. Tan J, Wu H, Yang X, Yang Y, Zhang L, Wang P, 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.
138. Abdelfatah MM, Noll A, Kapil N, Shah R, Li L, Nustas R, et al. Long-term Outcome of Gastric Per-Oral Endoscopic Pyloromyotomy in Treatment of Gastroparesis. Clin Gastroenterol Hepatol. 2021;19(4):816-24.
139. Martinek J, Hustak R, Mares J, Vackova Z, Spicak J, Kieslichova E, et al. Endoscopic pyloromyotomy for the treatment of severe and refractory gastroparesis: a pilot, randomised, sham-controlled trial. Gut. 2022;71(11):2170-8.
140. Gonzalez JM, Mion F, Pioche M, Garbay V, Baumstarck K, Boucekine 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.
141. Aziz M, Gangwani MK, Haghbin H, Dahiya DS, Sohail AH, Kamal F, 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.
142. McCallum RW, Sarosiek I, Parkman HP, Snape W, Brody F, Wo J, et al. Gastric electrical stimulation with Enterra therapy improves symptoms of idiopathic gastroparesis. Neurogastroenterol Motil. 2013;25(10):815-e636.