Inside Out: Gastroenterology & Hepatology Workshop

Saturday 25 October 2025
Clinical Skills Development Service | RBWH

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Controversies in disorders of gut-brain interactions: Essentials for GPs

Dr Trina Kellar
Lead, Neurogastroenterology Service | RBWH

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Royal Brisbane and Women's Hospital
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The University of Queensland
AUSTRALIA

QLD PelvicFloor Centre

Disorders of Gut-Brain Interaction: Core Skills & Controversies in 2025

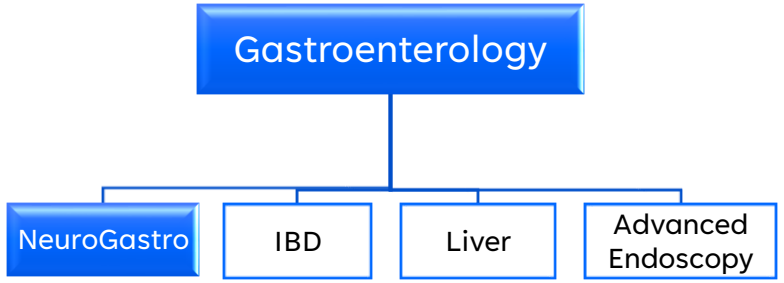
Dr Trina Kellar

Royal Brisbane & Women's Hospital - Neurogastroenterology Unit

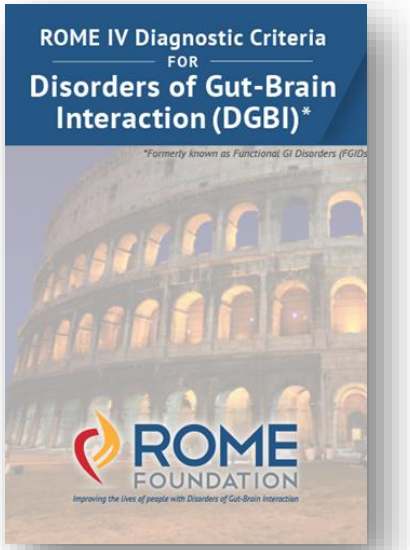
Overview

- Neurogastroenterology & DGBI
- Eating disorders + DGBI
- Idiopathic Gastroparesis
- EDS, SMAS, MALS, PoTS, MCAS
- Therapeutic overview

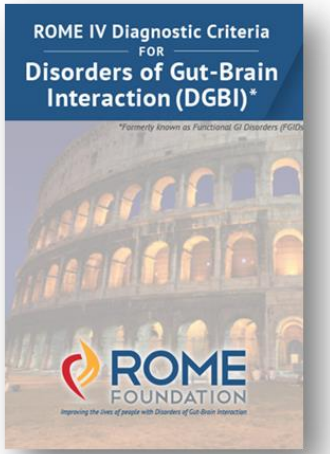
What is NeuroGastroenterology?



- NeuroGastroenterology:
  1. Disorders of Gut-Brain Interaction (DGBI)
     - *aka*. functional gut disorders, gut-brain disorders
     - Rome IV diagnostic criteria
  2. Motility disorders



- Multidisciplinary team RBWH
  - GE, dietitians, mental health, QuEDS, persistent pain
  - +/- sub-specialties, community providers
- Multidisciplinary team QLD Pelvic Floor Centre



theromefoundation.org/rome-iv/rome-iv-criteria

Table 2. Functional Gastrointestinal Disorders: Disorders of Gut–Brain Interaction

A. Esophageal Disorders

A1. Functional chest pain
A2. Functional heartburn
A3. Reflux hypersensitivity
A4. Globus
A5. Functional dysphagia

B. Gastroduodenal Disorders

B1. Functional dyspepsia
  B1a. Postprandial distress syndrome (PDS)
  B1b. Epigastric pain syndrome (EPS)
B2. Belching disorders
  B2a. Excessive supragastric belching
  B2b. Excessive gastric belching
B3. Nausea and vomiting disorders
  B3a. Chronic nausea vomiting syndrome (CNVS)
  B3b. Cyclic vomiting syndrome (CVS)
  B3c. Cannabinoid hyperemesis syndrome (CHS)
B4. Rumination syndrome

C. Bowel Disorders

C1. Irritable bowel syndrome (IBS)
  IBS with predominant constipation (IBS-C)
  IBS with predominant diarrhea (IBS-D)
  IBS with mixed bowel habits (IBS-M)
  IBS unclassified (IBS-U)
C2. Functional constipation
C3. Functional diarrhea
C4. Functional abdominal bloating/distension
C5. Unspecified functional bowel disorder
C6. Opioid-induced constipation

D. Centrally Mediated Disorders of Gastrointestinal Pain

D1. Centrally mediated abdominal pain syndrome (CAPS)
D2. Narcotic bowel syndrome (NBS)/
  Opioid-induced GI hyperalgesia

E. Gallbladder and Sphincter of Oddi (SO) Disorders

E1. Biliary pain
  E1a. Functional gallbladder disorder
  E1b. Functional biliary SO disorder
E2. Functional pancreatic SO disorder

F. Anorectal Disorders

F1. Fecal incontinence
F2. Functional anorectal pain
  F2a. Levator ani syndrome
  F2b. Unspecified functional anorectal pain
  F2c. Proctalgia fugax
F3. Functional defecation disorders
  F3a. Inadequate defecatory propulsion
  F3b. Dyssynergic defecation

Rome IV Classification: Functional Dyspepsia

B1a. Postprandial Distress Syndrome (PDS)

*Diagnostic criteria** *Must include **one or both** of the following at least 3 days a week:*

1. Bothersome postprandial fullness (i.e., severe enough to impact on usual activities)
2. Bothersome early satiation (i.e., severe enough to prevent finishing a regular size meal)

No evidence of organic, systemic, or metabolic disease that is likely to explain the symptoms on routine investigations (including at upper endoscopy)

*Criteria fulfilled for the last 3 months with symptom onset at least 6 months prior to diagnosis

Supportive criteria

1. Postprandial epigastric pain or burning, epigastric bloating, excessive belching, and nausea can also be present
2. Vomiting warrants consideration of another disorder
3. Heartburn is not a dyspeptic symptom but may often co-exist
4. Symptoms that are relieved by evacuation of feces or gas should generally not be considered as part of dyspepsia
5. Other individual digestive symptoms or groups of symptoms (e.g., from GERD and IBS) may co-exist with PDS

B1b. Epigastric Pain Syndrome (EPS)

*Diagnostic criteria** *Must include **one or both** of the following symptoms at least 1 day a week:*

1. Bothersome epigastric pain (i.e., severe enough to impact on usual activities)
2. Bothersome epigastric burning (i.e., severe enough to impact on usual activities)

No evidence of organic, systemic, or metabolic disease that is likely to explain the symptoms on routine investigations (including at upper endoscopy).

*Criteria fulfilled for the last 3 months with symptom onset at least 6 months prior to diagnosis

Supportive criteria

1. Pain may be induced by ingestion of a meal, relieved by ingestion of a meal, or may occur while fasting
2. Postprandial epigastric bloating, belching, and nausea can also be present
3. Persistent vomiting likely suggests another disorder
4. Heartburn is not a dyspeptic symptom but may often co-exist
5. The pain does not fulfill biliary pain criteria
6. Symptoms that are relieved by evacuation of feces or gas generally should not be considered as part of dyspepsia
7. Other digestive symptoms (such as from GERD and IBS) may co-exist with EPS

B3a. Chronic Nausea Vomiting Syndrome (CNVS)

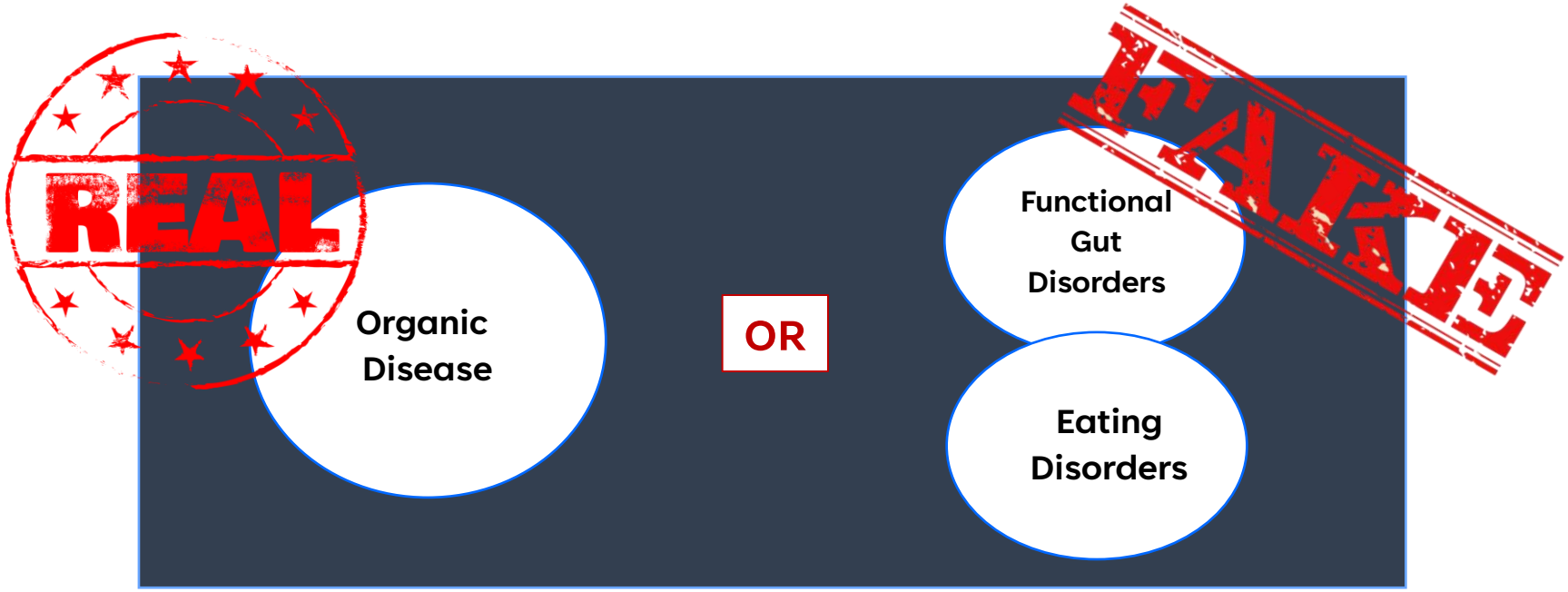
*Diagnostic criteria** *Must include **all** of the following:*

1. Bothersome (i.e., severe enough to impact on usual activities) nausea, occurring at least 1 day per week and/or one or more vomiting episodes per week
2. Self-induced vomiting, eating disorders, regurgitation, or rumination are excluded
3. No evidence of organic, systemic, or metabolic diseases likely to explain the symptoms on routine investigations (including at upper endoscopy)

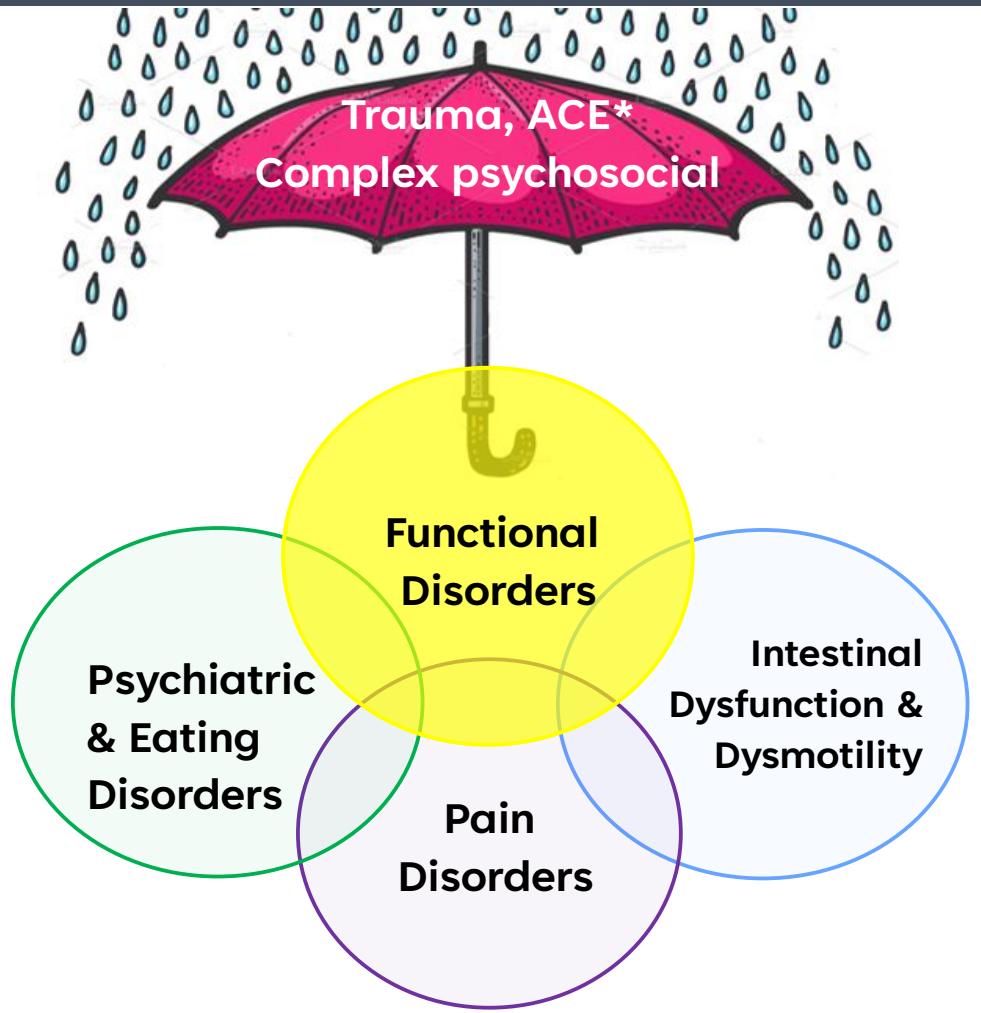
*Criteria fulfilled for the last 3 months with symptom onset at least 6 months prior to diagnosis

theromefoundation.org/rome-iv/rome-iv-criteria

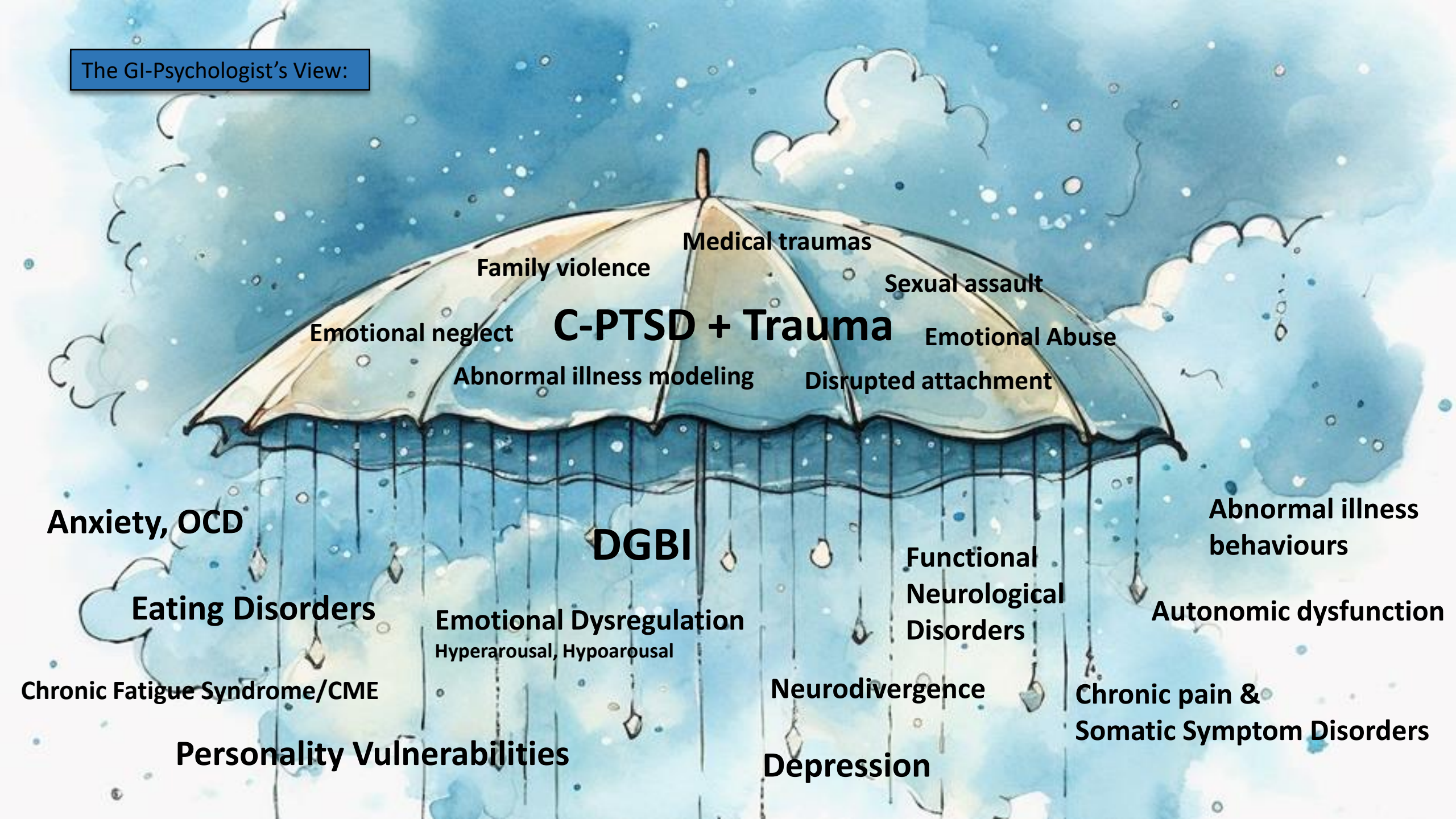
Understanding Functional Disorders: Move past the stigma



It’s all physiology, it’s all real.



*Adverse childhood experiences (ACE)


The GI-Psychologist's View:
Medical traumas
Family violence
Sexual assault
C-PTSD + Trauma
Emotional neglect
Emotional Abuse
Abnormal illness modeling
Disrupted attachment
Anxiety, OCD
DGBI
Abnormal illness behaviours
Functional Neurological Disorders
Eating Disorders
Emotional Dysregulation
Hyperarousal, Hypoarousal
Autonomic dysfunction
Chronic Fatigue Syndrome/CME
Neurodivergence
Chronic pain & Somatic Symptom Disorders
Personality Vulnerabilities
Depression

Disorders of Gut-Brain Interaction: Pathophysiology

Visceral Hypersensitivity and Pain

Bio-psychosocial model of disease:

- Predisposition (in utero, genetic, ACE)
- Trigger (injury, inflammation, stress)

↓

Complex neuro-immune interplay:

- Nervous system (enteric NS + CNS)
  Endocrine system (HPA, others)
  Immune system (intestinal + systemic)
- Microbiome (direct + metabolites)
  Mucosal barrier dysfunction
  Food antigens; substances

↓

- Peripheral + central pain sensitization
- Visceral hypersensitivity, hyperalgesia

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- Symptom perception & response
- Perpetuating factors (biopsychosocial)

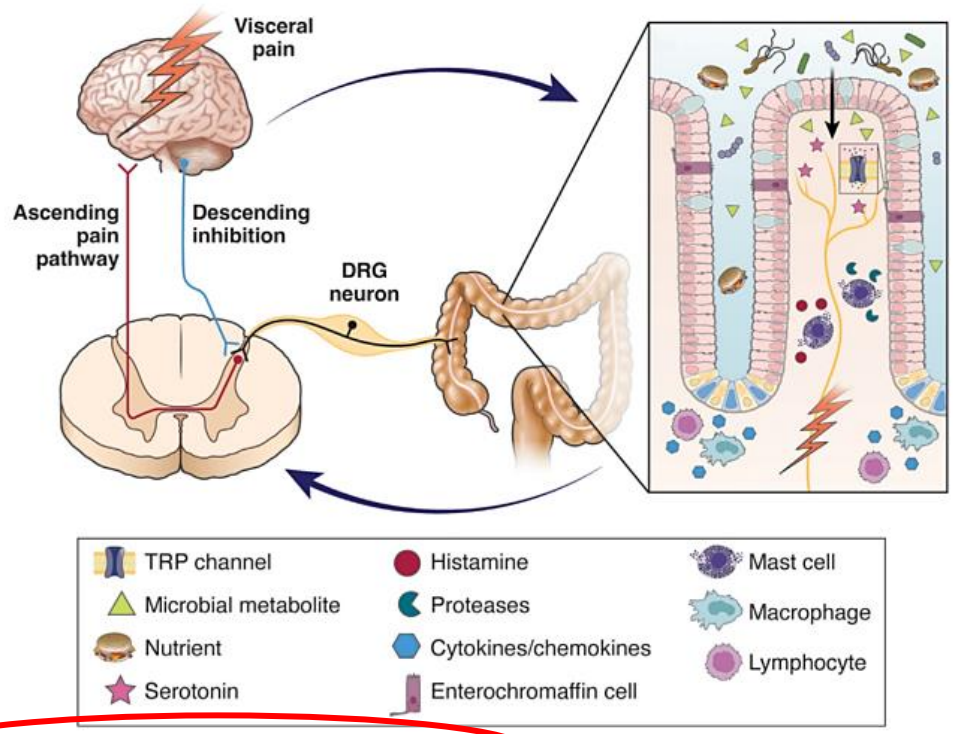
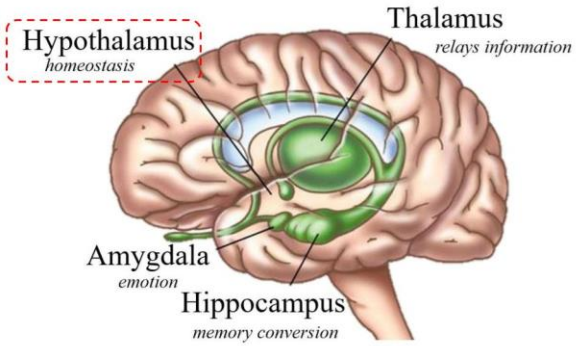
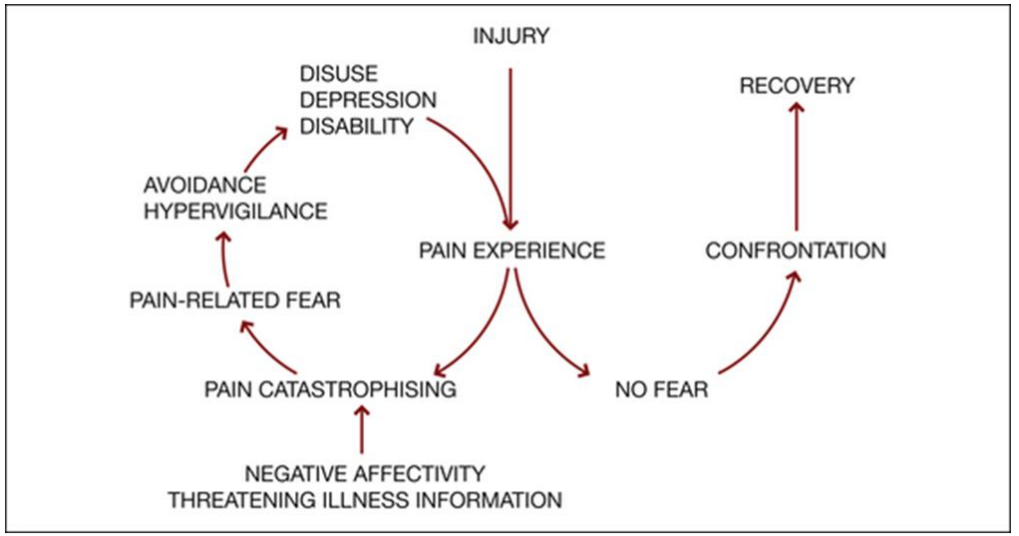


Figure 1. Chronic visceral pain is a disorder of the gut-brain axis. Nociceptors have cell bodies that lie in the dorsal root ganglia (DRG) and pseudounipolar axons that connect the intestine and the spinal cord. These synapse with second-order neurons in the spinal cord and with central ascending pathways thereafter. Nociceptive neurotransmission in the spinal cord is modulated by descending pathways. (*Inset*) At the level of the mucosa, nociceptive terminals are both mechanosensitive and chemosensitive and are stimulated by luminal factors (eg, microbial products and nutrients) as well as by host mediators released due to infection, inflammation, or tissue damage (eg, serotonin, histamine, proteases, chemokines, and cytokines). These mediators can act indirectly via the epithelium/enterochromaffin cells or can stimulate nociceptors directly if there is a breakdown in the mucosal barrier. This results in sensitization of ion channels such as TRP, resulting in increased visceral pain.

The Limbic System

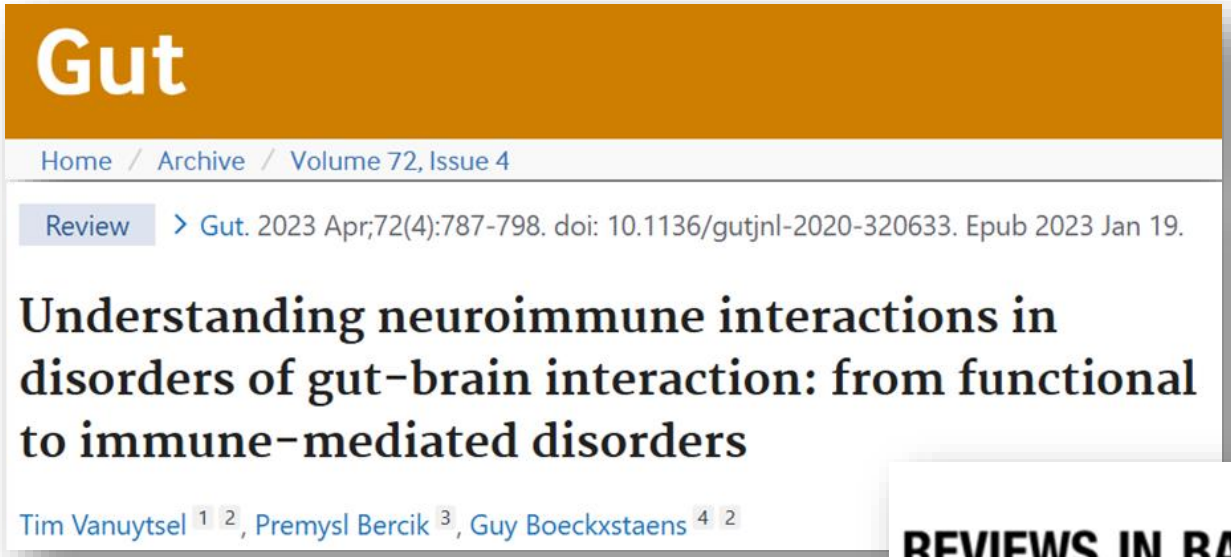


Symptom-Response Cycle:



Leeuw M, et al. 2007.

https://www.thereignofthebrain.com/resources/the-limbic-system-hypothalamus



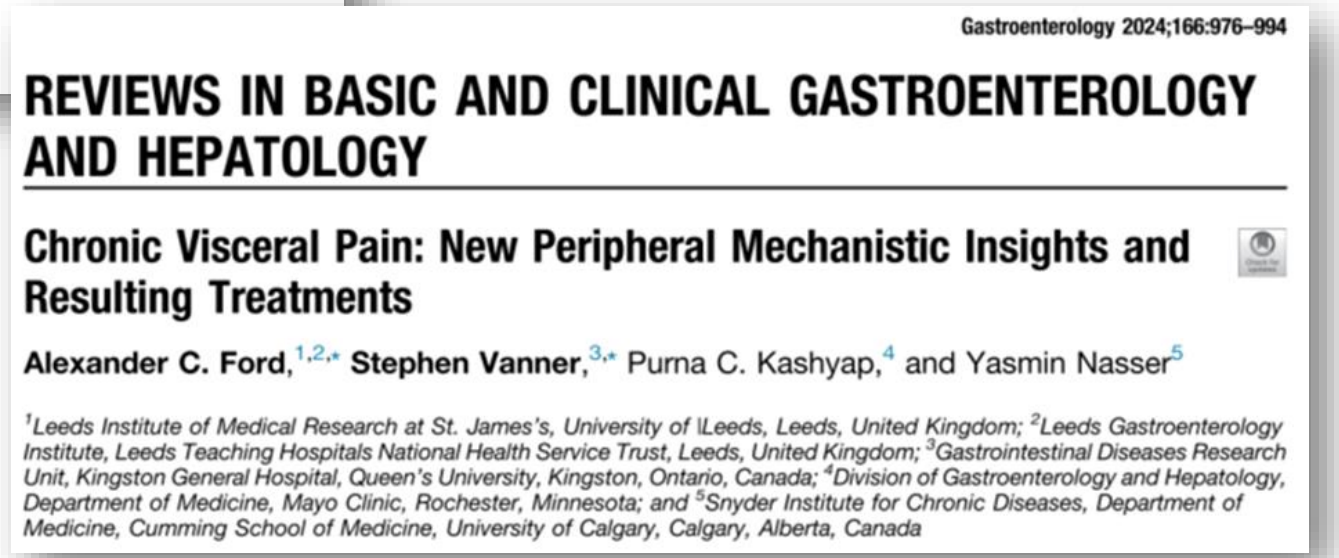
Gut

Home / Archive / Volume 72, Issue 4

Review > Gut. 2023 Apr;72(4):787-798. doi: 10.1136/gutjnl-2020-320633. Epub 2023 Jan 19.

Understanding neuroimmune interactions in disorders of gut-brain interaction: from functional to immune-mediated disorders

Tim Vanuytsel [1 2], Premysl Bercik [3], Guy Boeckxstaens [4 2]



Gastroenterology 2024;166:976–994

REVIEWS IN BASIC AND CLINICAL GASTROENTEROLOGY AND HEPATOLOGY

Chronic Visceral Pain: New Peripheral Mechanistic Insights and Resulting Treatments

Alexander C. Ford,[1,2,*] Stephen Vanner,[3,*] Purna C. Kashyap,[4] and Yasmin Nasser[5]

[1]Leeds Institute of Medical Research at St. James's, University of \Leeds, Leeds, United Kingdom; [2]Leeds Gastroenterology Institute, Leeds Teaching Hospitals National Health Service Trust, Leeds, United Kingdom; [3]Gastrointestinal Diseases Research Unit, Kingston General Hospital, Queen's University, Kingston, Ontario, Canada; [4]Division of Gastroenterology and Hepatology, Department of Medicine, Mayo Clinic, Rochester, Minnesota; and [5]Snyder Institute for Chronic Diseases, Department of Medicine, Cumming School of Medicine, University of Calgary, Calgary, Alberta, Canada

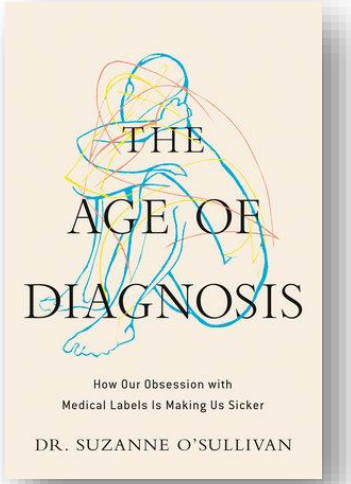
Concerning Trends & Controversies

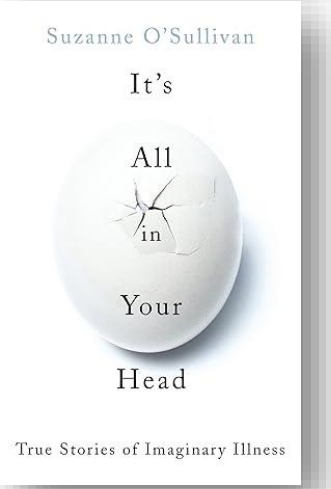
Concerning Trends

- International spike in:
  - Over-medicalization
  - Diagnoses of uncertain significance, self-diagnosis
  - Non-evidence based invasive treatments
  - Young adults on artificial nutrition support [Lal]
  - Online **misinformation**, social pressure, illness identity

- Diagnostic trends:
  - DGBI with severe disordered eating ('eating disorder excluded')
  - Hypermobility spectrum disorders incl Ehlers Danlos syndrome (EDS)
  - Vascular compression syndromes incl SMA and MAL syndromes
  - Postural tachycardia syndrome (PoTS)
  - Mast cell activation syndrome (MCAS)

In vulnerable individuals, high risk of iatrogenic harm.





Concerning Trends: An International Problem

Special Issue: Social Media and Mental Health

The tic in TikTok and (where) all systems go: Mass social media induced illness and Munchausen's by internet as explanatory models for social media associated abnormal illness behavior

Clinical Child Psychology and Psychiatry
2023, Vol. 28(1) 270–278

DOI: 10.1177/13591045221098522
journals.sagepub.com/home/ccp
SAGE

British Journal of Medical Psychology

Abnormal treatment behaviour

B. Singh, K. Nunn, J. Martin, J. Yates

First published: December 1981 | https://doi.org/10.1111/j.2044-8341.1981.tb01471.x

JGH Journal of Gastroenterology and Hepatology
JGHF
doi:10.1111/jgh.16698

REVIEW ARTICLE

Role of social media in the presentation of disorders of gut–brain interaction: Review and recommendations

Michael R Salzberg,* Hannah Kim,† Chamara Basnayake,‡ Darcy Holt§ and Michael A Kamm¶

*Department of Psychiatry, Royal Melbourne Hospital, University of Melbourne, †Medical Stream Lead, Eating Disorders, Orygen Specialist Program, ‡St Vincent's Hospital Melbourne, University of Melbourne, §Clinical Nutrition and Metabolism Unit and Department of Gastroenterology and Hepatology, Monash Health, Monash University, ¶St Vincent's Hospital and University of Melbourne, Melbourne, Victoria, Australia

Slide courtesy of Neurogastroenterologist Dr Charlotte Daker, Auckland

Correct Information: Crash Course

DGBI & Eating Disorders Overlap

Gut-brain disorders
Motility disorders
Eating Disorders
} Co-Exist

ED & DGBI

- No single test reliably differentiates ED from gastroparesis or DGBI
  - They co-exist

- 42-98% ED pts meet DGBI criteria [Hanel]
  - Satiety, distension, pain, nausea, regurgitation, heartburn, bloating, dysphagia [Salvioli]
  - Equal across different ED types [Boyd]
- 20-50% have delayed gastric emptying study [Riedlinger;Schalla]
  - Similar rates AN, BN, ARFID [Staller]
- 2/3 delayed colonic transit [Chiarioni]

- Up to 77% Gastroparesis *screen* positive for ARFID[Hollis]
- 20-43% Neurogastro clinic Pts meet ED criteria [Murray]

DGBI in Eating Disorders:

- Functional dyspepsia
  - Post-prandial distress syndrome
  - Epigastric pain syndrome
- Chronic nausea vomiting syndrome
- Rumination syndrome
- Functional dysphagia
- Functional bloating
- Functional constipation, IBS
- Defecatory dysfunction (mechanical, functional)

Avoidant Restrictive Food Intake Disorder (ARFID)

ARFID summary:

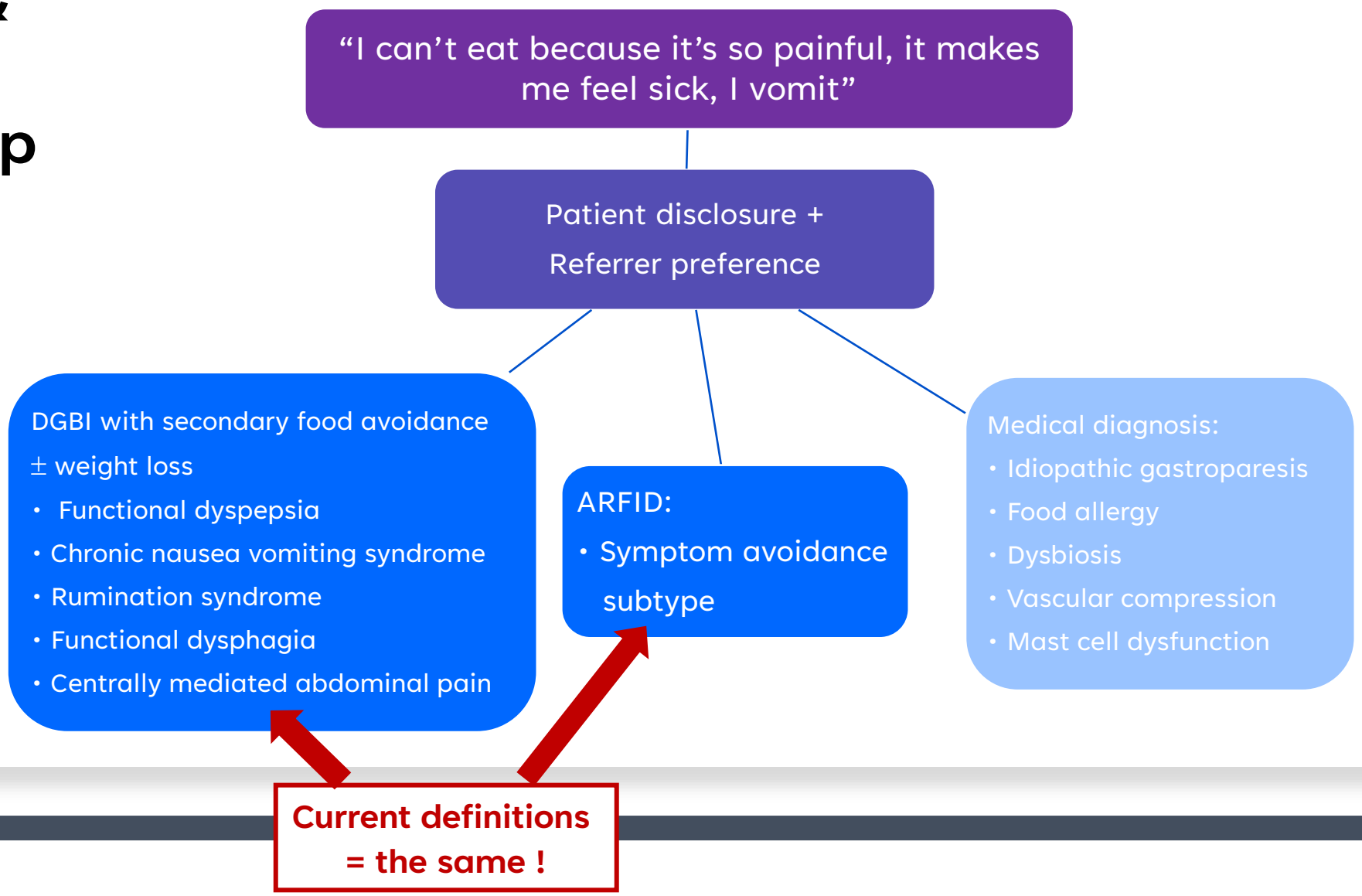
- Severely restricted oral intake
- Any BMI
- Not about body weight or shape
- 3 Sub-types ("I don't eat because…")
    1. To avoid symptoms
    2. No appetite
    3. Sensory aversions
- Not ***explained*** *by* medical condition

DSM-V (Revised 2013)

Avoidant-restrictive food intake disorder

- Restriction of food intake (frequency, volume, or variety) warranting independent attention and not due to lack of available food or cultural practices.
- Develops due to one or more of the following: (1) fear of aversive consequences (eg, choking, nausea, or diarrhoea), (2) lack of interest in eating or a low appetite, or (3) sensitivity to sensory characteristics of food.
- Associated with one or more of the following (required for diagnosis): (1) medical consequences such as weight loss, low weight, inability to gain weight or grow, malnutrition, dependence on supplemental nutrition, or (2) psychosocial impairments (eg, difficulty with social eating, significant distress around eating, or family impairments).

DGBI & ARFID Overlap



ED-DGBI Treatment Approach

- Bio-psychosocial model of care
  - Joint care, or refer to MDT; practice within your scope
- Least invasive investigation and treatment possible
  - Oral nutrition support; NGT if indicated; avoid other
  - Long-term tube feeding in DGBI not shown to improve weight or Sx
- Treat reflux, constipation
- Prokinetics, anti-emetics
- Neuromodulators for functional GI symptoms
- Treat comorbid psychiatric, persistent pain

Atkins et al. Journal of Eating Disorders (2023) 11:20
https://doi.org/10.1186/s40337-022-00731-6

Journal of Eating Disorders

REVIEW Open Access

Assessment and management of disorders of gut–brain interaction in patients with eating disorders

Micaela Atkins[1,2*], Helen Burton Murray[2,3†] and Kyle Staller[2,4†]

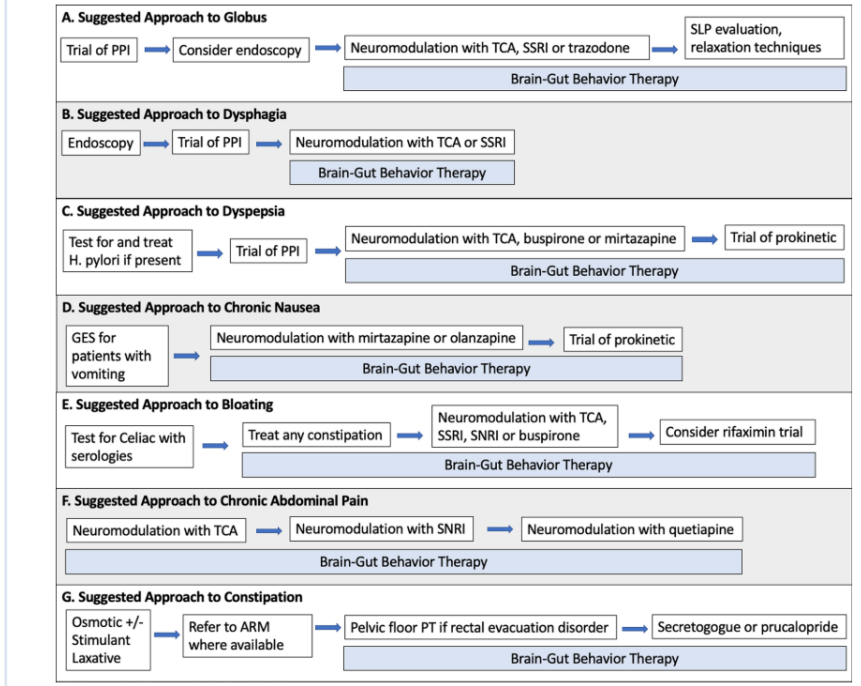


Fig. 1 Suggested management for DGBI symptoms. PPI = proton pump inhibitor, TCA = tricyclic antidepressant, SSRI = selective serotonin reuptake inhibitor, SLP = speech language pathology, GES = gastric emptying scan, SNRI = serotonin norepinephrine reuptake inhibitor, ARM = anorectal manometry, PT = physical therapy

Artificial Nutrition Support in DGBI

GESA GUIDELINE

Management of Patients with Disorders of Gut-Brain Interaction and Altered (Reduced) Food Intake Behaviour

Short title: DGBI-induced altered food intake behaviour

Ayesha Shah[$*#@ 1,2], Jason P. Connor[*@3], Nicholas J. Talley[%$#@ 4], Andrew Day[+], Basil Almehdawy[*2], Chamara Basnayake[#5,6], Rebecca Burgell[#7], Sharon Carey[~&8,9], Charles Cock[#10], Charlotte Daker[+11], Kerith Duncanson[@$4], Vishal Kaushik[*12], Simon R. Knowles[#@14], Dan Kent[*14], Trina Kellar[*#2], Natasha Koloski[*@$1,2], Katherine Lane[*15], Neal Martin[*1,2], Nikhil Thapar[*>2,16,17], Paul Stanley[*1], Allison Malcom[#18,19], Kate Elizabeth Murphy[15], Gemma Sharp[20], Caroline Tuck[@13], Michael Jones[$, 21], Gerald Holtmann[*$@^1,2]

SMALL BOWEL AND NUTRITION

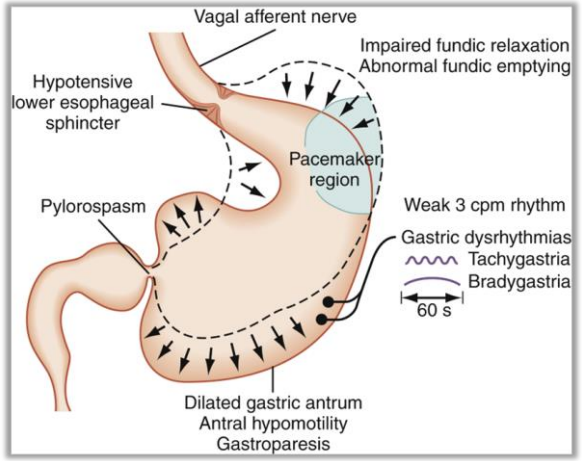
REVIEW

Jejunal feeding: when is it the right thing to do?

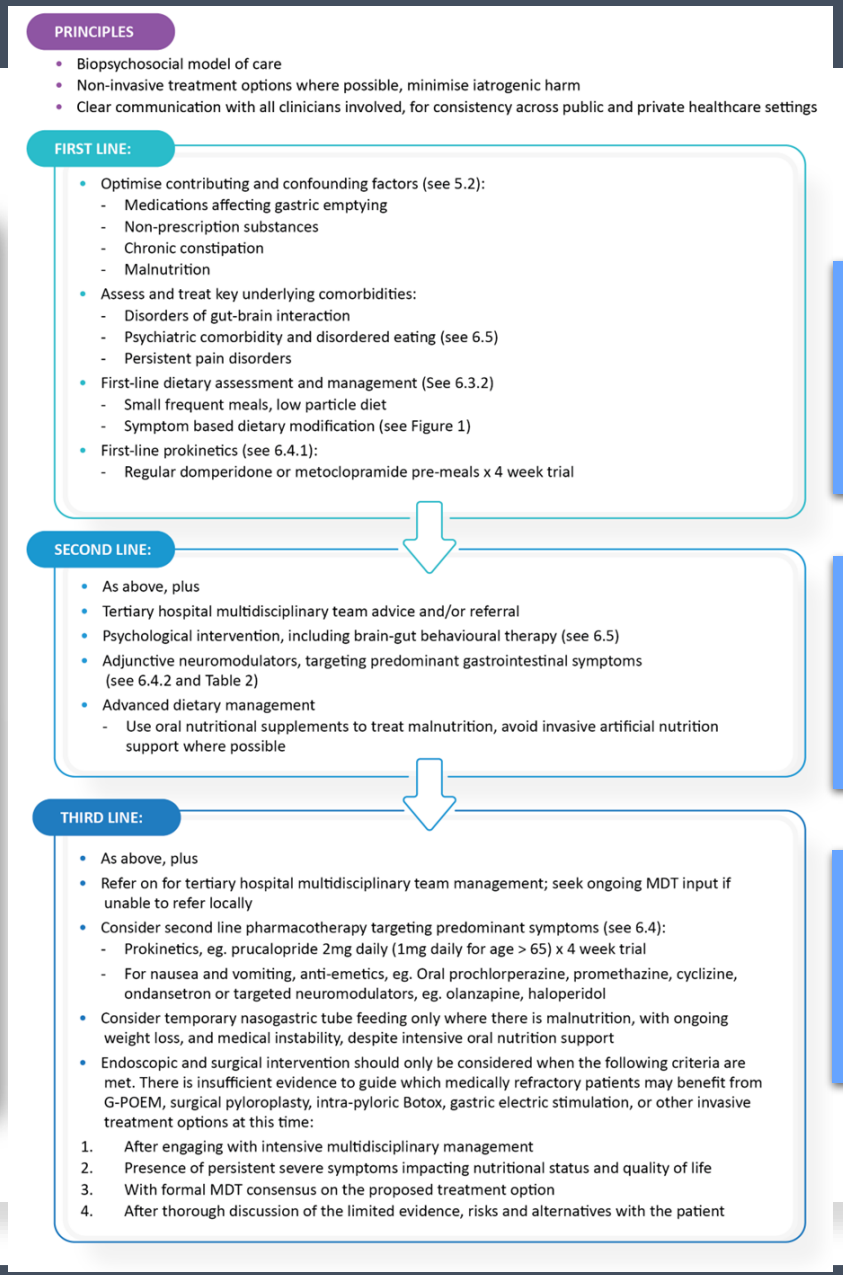
Peter Paine [1], Marie McMahon,[1] Kirstine Farrer,[2] Ross Overshott,[3] Simon Lal[4]

Idiopathic Gastroparesis: Update

- Much more than delayed gastric emptying
- Gastric emptying is a dynamic, physiological response
  - Acute illness, abdominal surgery, glycaemic and hormone fluctuations
  - Malnutrition, weight loss, eating disorders[Riedlinger, Schalla, Staller]
  - Medications, substances
    - Opioids, anticholinergics, antipsychotics, GLP-1 agonists, cannabinoids [Camilleri]
- Gastric emptying time correlates poorly with symptoms
  - Asymptomatic prevalence unknown
  - 40% variability over time



- "Functional Dyspepsia and gastroparesis likely represent an overlapping spectrum of sensorimotor abnormalities affecting the gastro-duodenum"
  - *Lal et al.* Position paper from ESCNM, ESNM, and Rome Foundation (July 2024)



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)

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 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)

Available @ GESA Clinical Resources Nov 2025: https://www.gesa.org.au/resources/clinical-practice-resources/

Hypermobility Spectrum Disorders

Neurogastroenterology

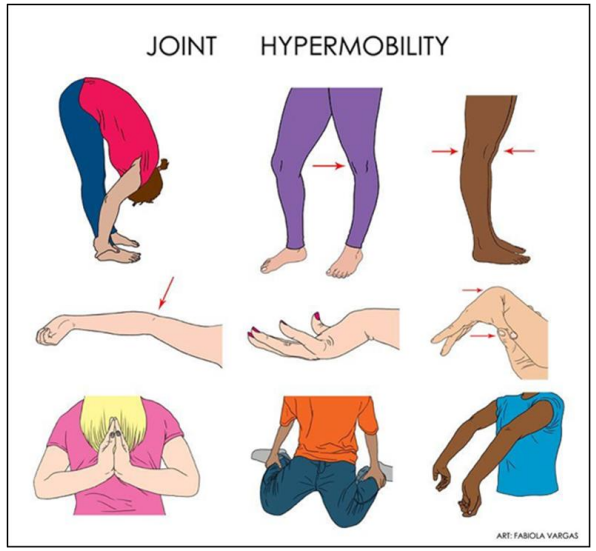
Review

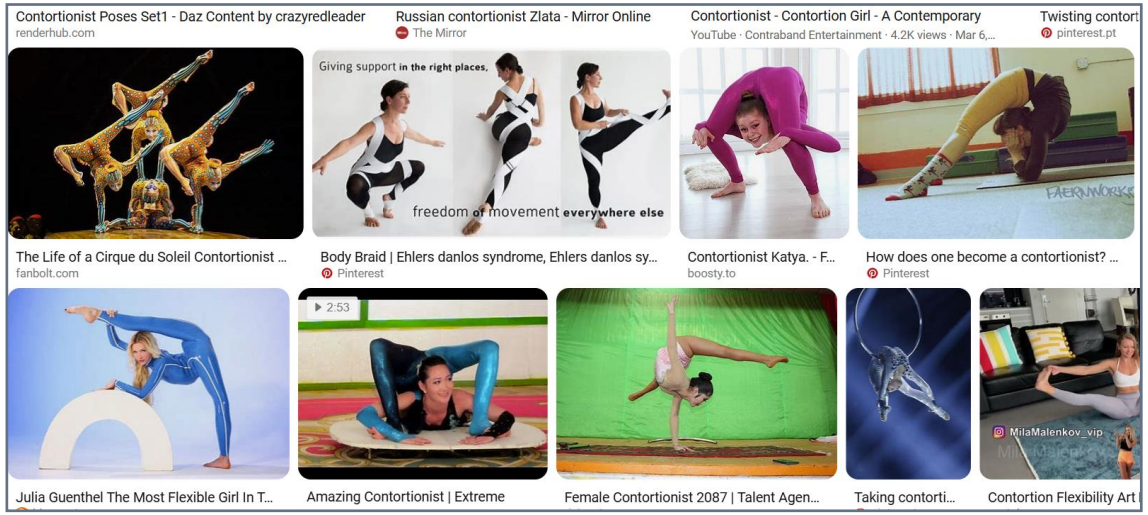
Gastrointestinal symptoms and nutritional issues in patients with hypermobility disorders: assessment, diagnosis and management

Ching Lam,[1] Gehanjali Amarasinghe,[2] Natalia Zarate-Lopez,[3] Asma Fikree,[4] Peter Byrne,[5] Sorena Kiani-Alikhan,[6] Simon Gabe,[2,7] Peter Paine [8]

Table 1 Hypermobility disorders

Condition	Features	Diagnosis
Joint hypermobility	► Either localised, peripheral or generalised. ► May be symptomatic or asymptomatic.	► Often not pathological.
Hypermobile spectrum disorder	► Generalised joint hypermobility (with allowances for age but not environmental factors). ► Varied connective tissue and other symptoms (unclear evidence for true association).	► Clinical constellation of symptoms but not fulfilling diagnostic criteria for hEDS. ► Genetic basis unknown. ► Molecular basis unknown. ► Pathogenesis of associated symptoms unknown.
Hypermobile Ehlers Danlos syndrome	► Generalised joint hypermobility (with allowances for age but not environmental factors). ► Clinical features suggestive of a connective tissue disorder. ► Female preponderance (unusual for a presumed autosomal dominant condition). ► Other associated symptoms (unclear evidence for true association).	► Clinical constellation of symptoms fulfilling diagnostic criteria for hEDS, some of which may suggest an acquired connective tissue disorder and some of which suggest a heritable connective tissue disorder. ► Genetic basis unknown. ► Molecular basis unknown. ► Pathogenesis of associated symptoms unknown.
Other Ehlers Danlos syndrome types	Joint hypermobility, skin hyperextensibility and tissue/blood vessel fragility.	► Clinical constellation of symptoms. ► Genetic basis known. ► Molecular basis known.
Joint hypermobility syndrome has now been incorporated into hypermobile spectrum disorder or hEDS.[1 2] hEDS, hypermobile Ehlers-Danlos syndrome.		





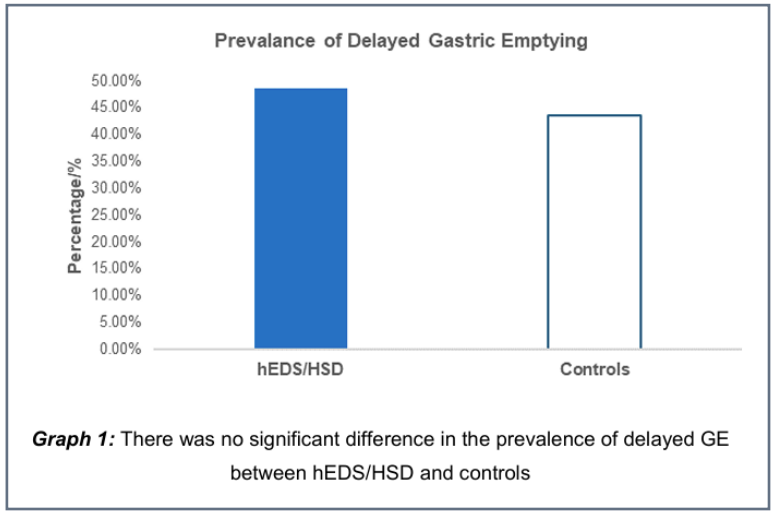
? When does it constitute disease

Hypermobility: GI Perspective

"After years of pain and undiagnosed symptoms a doctor finally diagnosed EDS and everything made sense, it's not in my head!!"

- Limited data, comorbid vs primary pathology?
  - Zero evidence EDS causes GI Sx - association vs causation
  - Population prevalence JH unknown (high % asymptomatic)
- More prevalent in hEDS: [Lam 2023]
  - Disorders of gut-brain interaction (2+ and severe)
  - Pain disorders, polypharmacy, opioid use
  - Avoidant restrictive food intake disorder (ARFID)
- Gastroparesis not increased [Upadhyaya]
- Early research, two phenotypes emerging:
  1. Severe CTD, volvulus, intussusception, but less pain
  2. Less hypermobile, more pain and hypersensitivity Sx

→ Biopsychosocial model: treat as DGBI + pain



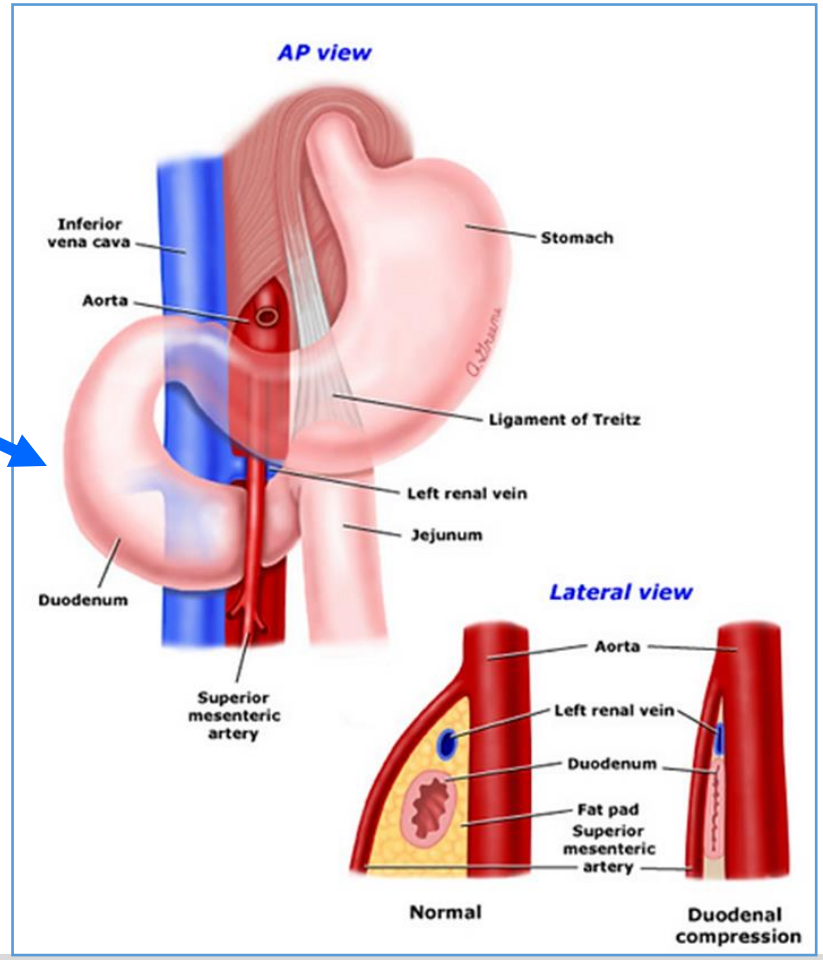
Graph 1: There was no significant difference in the prevalence of delayed GE between hEDS/HSD and controls

Baeza-Velasco C et al. Autism, Joint Hypermobility-Related Disorders and Pain. Front Psychiatry. 2018 Dec 7;9:656

Upadhyaya A et al. Delayed gastric emptying is not significantly increased in patients with hypermobile ehlers-danlos syndrome. Gut. 2024;73(Suppl 1):A289-A.

Superior Mesenteric Artery Syndrome (SMAS)

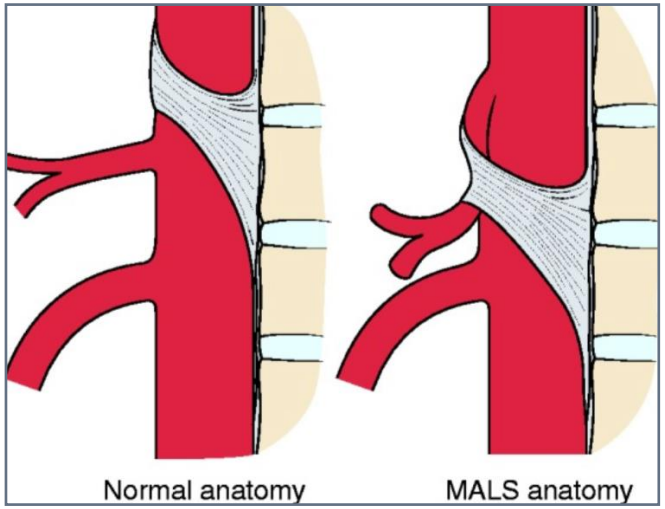
- Aetiology:
  - Acquired: severe weight loss, body cast, burns, adhesions
  - Congenital: short ligament Treitz, aberrant vessel
- Small bowel obstruction at 3rd part duodenum
- Luminal imaging ± endoscopy
  - 0.8% population prevalence loss of vascular angle asymptomatic [Rosa-Jimenez 2003]
- Management:
  - Weight restoration 75-80% success [Biank 2006, Wan 2020]
  - Lacking research in this cohort
  - Surgical: duodenojejunostomy, anterior transposition of D3
    - No long-term surgical outcome studies
    - Considered experimental



Oka et al World J Clin Cases 2023

Median Arcuate Ligament Syndrome (MALS)

- Coeliac artery compression by MAL
  - Usually atherosclerosis
  - Adult onset ‘idiopathic’ very controversial
  - Theory: anatomical variant - partial occlusion - no collateralization - intimal thickening occurs.
- Symptoms: foregut ischaemia (post-prandial, exertional)
- Inspiration/**expiration** vascular imaging:
  - Very poor symptom correlation
  - 4-7% asymptomatic population prevalence [Baskan 2015, Terlouw 2020]
- Surgical management:
  - No high quality longitudinal studies showing safety or efficacy of decompression ± cervical ganglionectomy in this cohort [Goodall 2020]
  - Surgical sham studies 18-57% placebo [Metz 2022]
  - Considered experimental

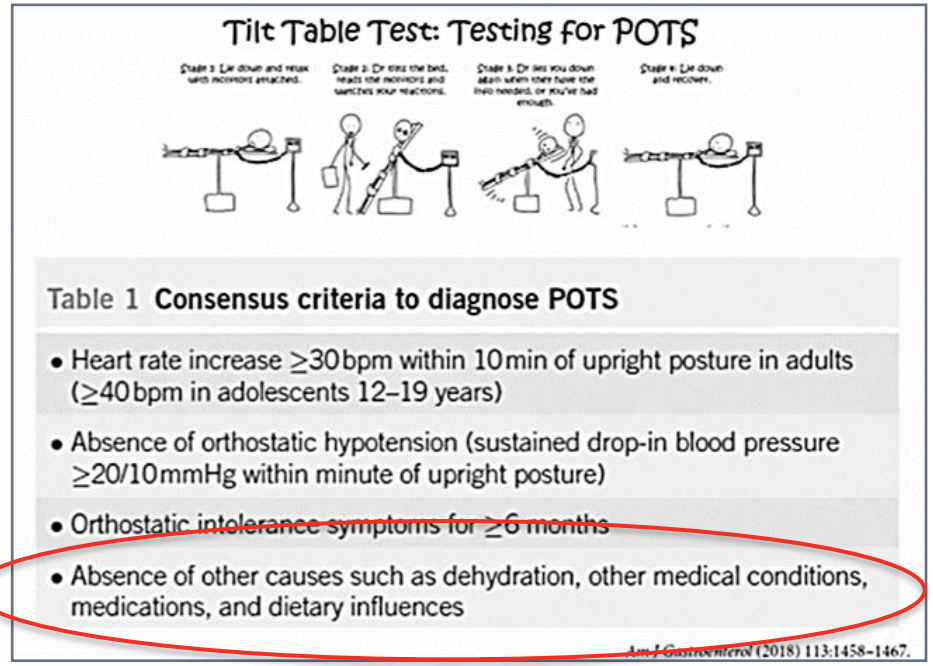


Recommendations	GRADE	Expert agreement
Recommendation 3 To exclude alternative diagnoses at least the following diagnostic tests must be performed: upper gastrointestinal endoscopy and abdominal imaging (CT scan/MRI scan). Depending on age and symptoms colonoscopy should be considered, but is mandatory in patients with diarrhoea.	1D	91%
Recommendation 4 A presumptive diagnosis of occlusive chronic mesenteric ischaemia is based on a combination of compatible history, significant mesenteric artery stenosis on radiological imaging and, preferably, a positive functional test. Results should be discussed in an expert multidisciplinary setting by at least a gastroenterologist, vascular surgeon and (interventional) radiologist.	1C	78%
Recommendation 14 CA compression in MALS can be diagnosed by inspiration/expiration duplex ultrasound, CTA or CE-MRA. In patients of younger age, suspected of having MALS, both duplex ultrasound and CE-MRA (</=2mm slices with 3D reconstructions) in inspiration and expiration and recommended imaging techniques	1D	74%
Recommendation 25 Patients with MALS might be considered for surgical coeliac artery release	2D	96%
Recommendation 26 In patients with MALS (and no preceeding adequate coeliac artery release) endovascular stenting of the CA is contraindicated	1D	100%

European Guidelines for Mesenteric Ischaemia (2020)

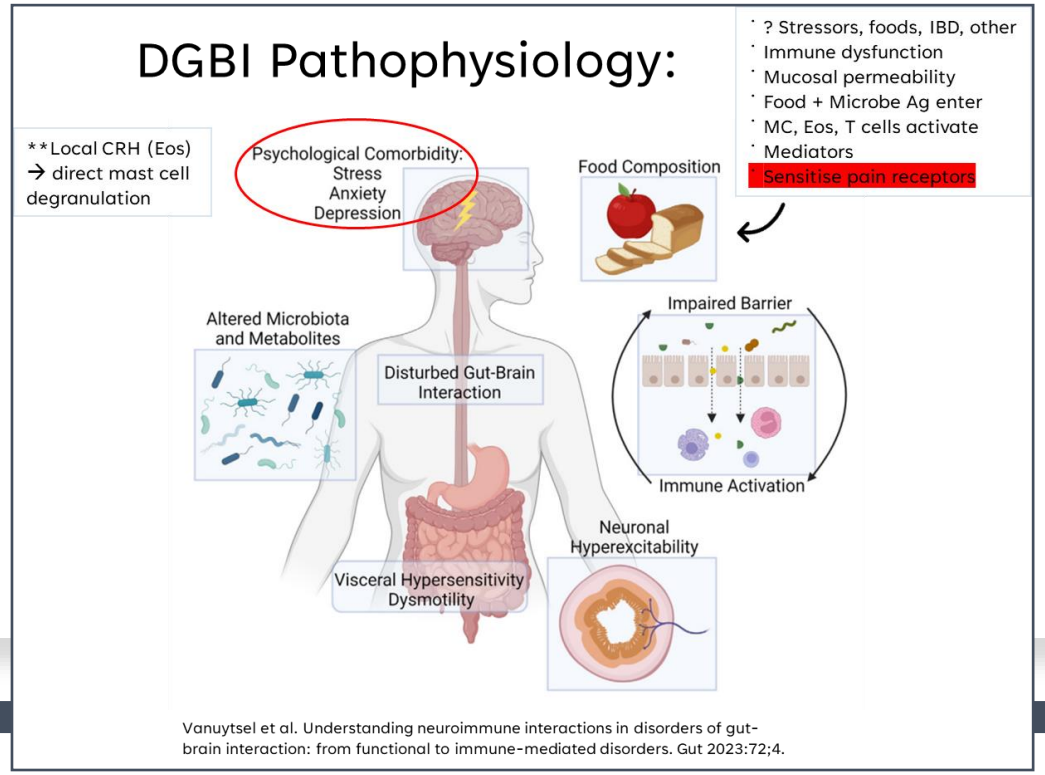
Postural Orthostatic Tachycardia Syndrome (POTS)

- Type of orthostatic intolerance
- Normal physiological response
  - Eating disorder admission criteria
  - Malnutrition, dehydration
  - Deconditioning
  - Opioids, anticholinergics
- Zero evidence PoTS causes GI symptoms
  - Secondary common
  - True orthostatic GI symptoms very rare
- Management:
  - Treat causes
  - Hydrate, high salt
  - Orthostatic tolerance training, compression stockings
  - Cardiac medications (limited evidence)



Mast Cell Activation Syndrome (MCAS)

- Immune system (incl mast cells, eosinophils) role in visceral hypersensitivity well established
- Part of DGBI pathophys, not separate diagnosis?
- Allergic phenotype → refer to Immunologist



Vanuytsel et al. Understanding neuroimmune interactions in disorders of gut-brain interaction: from functional to immune-mediated disorders. Gut 2023:72;4.

Table 4 Criteria for diagnosis of MCAS based on a consensus report by an international mastocytosis working group[36] and the AAAAI mast cell disorder work group report[37]

A	Typical signs of severe, recurrent (episodic) systemic MCA (often in form of anaphylaxis or crisis) (definition of systemic: involving at least two organ systems).
B	Objective laboratory evidence of MC activation: preferred marker: increase in serum tryptase level (baseline to baseline plus 20%+2 ng/mL)
C	Response to therapy with MC-stabilising agents, drugs directed against MC mediator production or drugs blocking mediator release or effects

All three criteria need to be fulfilled for diagnosis of MCAS.
AAAAI, American Academy of Allergy, Asthma & Immunology; MC, mast cell; MCAS, mast cell activation syndrome.

Gut
Home / Archive / Volume 72, Issue 4
Review > Gut. 2023 Apr;72(4):787-798. doi: 10.1136/gutjnl-2020-320633. Epub 2023 Jan 19.

Understanding neuroimmune interactions in disorders of gut-brain interaction: from functional to immune-mediated disorders

Tim Vanuytsel [1 2], Premysl Bercik [3], Guy Boeckxstaens [4 2]

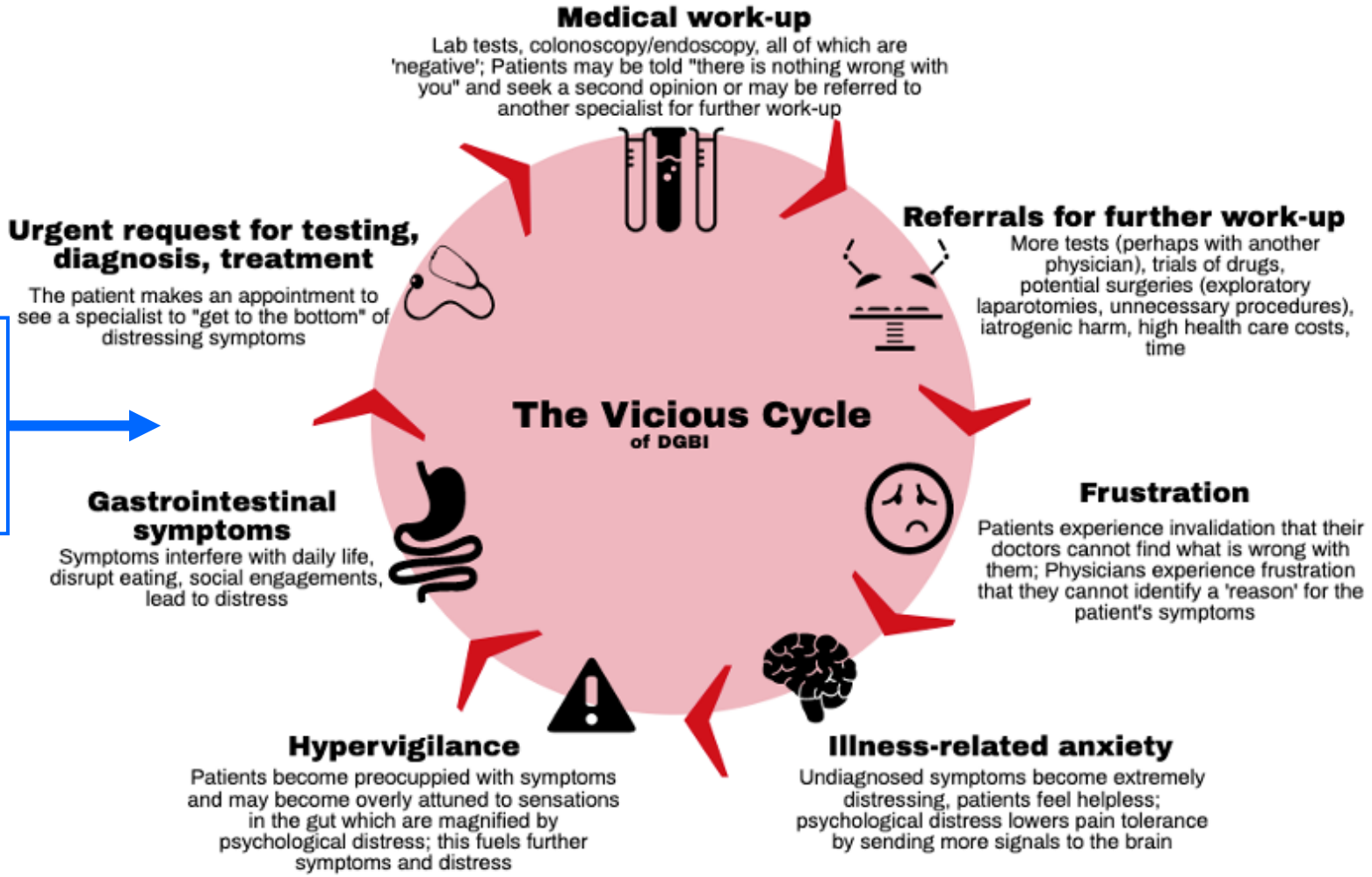
Treatment Approach:
Primum non nocere

- ✗ Tubes
- ✗ Lines
- ✗ Scalpels

Interplay Of Patient, Clinician And System Factors

Break the cycle:

1. Listen, empathise
2. Thorough assessment
3. Confident diagnosis
4. Management plan



Feingold & Drossman 2021

Treatment: Approach to the Complex Case

- First do no harm
- Least invasive treatment possible
- Biopsychosocial model of care
  - Neuromodulators, psychology, dietitian, physio
  - MDT is standard of care [Basnayake 2021]
- MDT *before* commencing artificial nutrition support
  - Nutritional rehabilitation, collaborate with eating disorder clinicians
- Avoid uncertain diagnoses
- Counter misinformation with logic and compassion
  - Prioritise education, support, collaborate with mental health clinicians
- Practice within your scope, collaborate early
  - Pain rehabilitation program MDT + other specialties
  - Know who you are referring to (NGE, versus interest in functional gut)

Step back from the noise,
acknowledge the unknown,
& focus on what ***is*** known.

Neuromodulators:

Mirtazapine

- Nausea, dyspepsia, low appetite, mood, sleep

Olanzapine, quetiapine, haloperidol, aripiprazole

- Nausea, mood, sleep

Tri/tetracyclic antidepressant

- Amitriptyline 10-50mg+ nocte
  - IBS-D, pain; anticholinergic SE useful (diarrhoea, sleep)
- Nortriptyline 10-50mg+ nocte
  - IBS-C, pain; less anticholinergic SE

SNRI

- Duloxetine, venlafaxine
  - Centrally mediated pain, widespread pain, mood disorder

Gabapentin, pregabalin

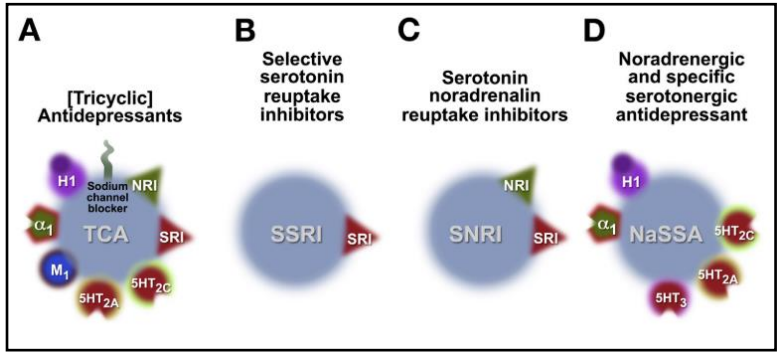
- Neuropathic pain, fibromyalgia

Review > Gastroenterology. 2018 Mar;154(4):1140-1171.e1.
doi: 10.1053/j.gastro.2017.11.279. Epub 2017 Dec 22.

Neuromodulators for Functional Gastrointestinal Disorders (Disorders of Gut-Brain Interaction): A Rome Foundation Working Team Report

Douglas A Drossman [1], Jan Tack [2], Alexander C Ford [3], Eva Szigethy [4], Hans Törnblom [5], Lukas Van Oudenhove [6]

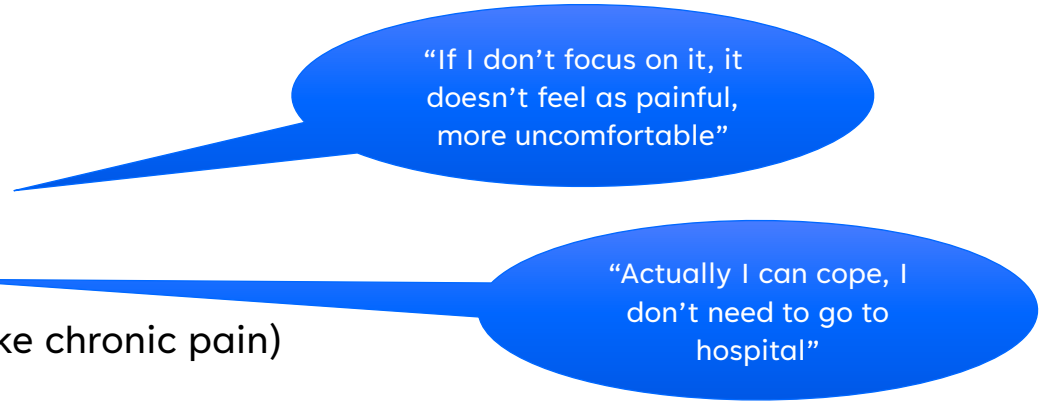
Neuromodulators: medications with CNS target



How does GI-Psychology work?

- Psychotherapy is a neuromodulator
- Psychotherapy targets:
  - The Cause: psychological and environmental factors that create and aggravate symptoms
  - The Effect: distress, mood, dysfunction caused by symptoms

- Modulates patient:
  - Experience of the symptoms
  - Response to the symptoms
  - Brain-gut connection (rewiring, like chronic pain)



Gut-directed Hypnotherapy

- Evidence based, delivered by GI-psychologists
- Treatment course, typically 6-10 sessions

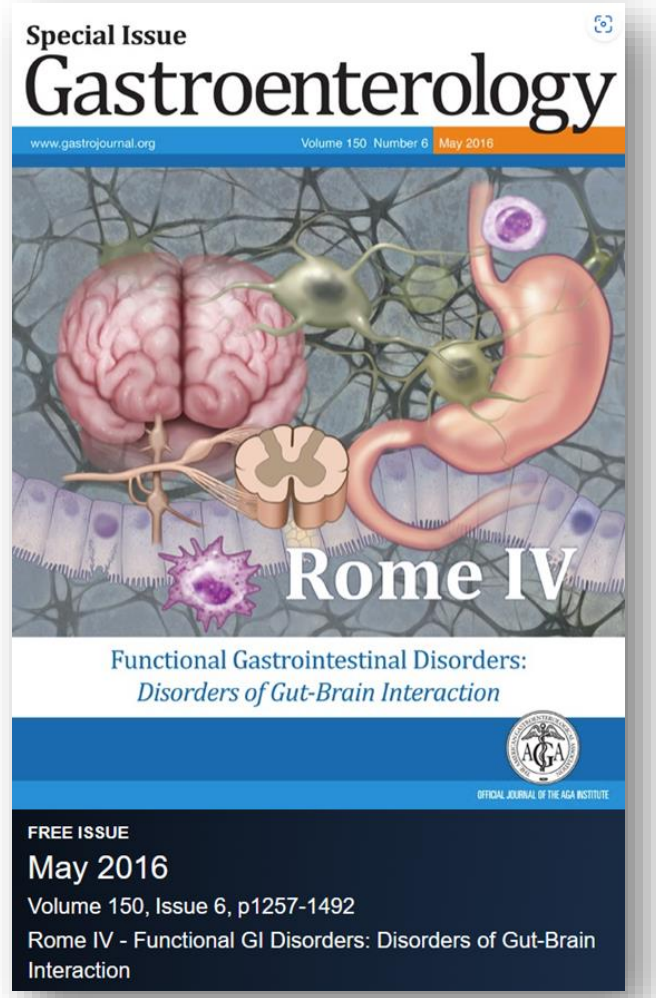
- Good candidates: Simpler cases
  Chronic functional nausea, bloating, pain
  Without major psychiatric comorbidity

- More complex psychological factors – GI psychotherapy +/- hypnotherapy

- More information:
  - The Gut Centre Gut Directed Hypnotherapy | The Gut Centre
  - Mind & Gut Clinic

Key Concepts

1. All functional disorders have a real physiological basis
2. High psychiatric comorbidity, vulnerable cohort
3. High risk iatrogenic harm
   - Avoid uncertain diagnoses, online misinformation
   - Practice within your scope
   - Least invasive testing and treatment
   - MDT before artificial nutrition
4. Need for cross-disciplinary research, education, services



Case: Ms TC

- 52 F RN, 30y intermittent nausea, vomiting, satiety, fullness, reflux

  → 5y now daily, most meals, constant nausea ‘tried every tablet, nothing works’

  → Progressive food intolerances ‘I’m allergic to everything’

  → Episodic weight loss 5-10kg, nadir BMI 17, slowly regains

- BG: chronic back pain, migraines; hysterectomy, appendicectomy, cholecystectomy, diagnostic laparoscopy for pelvic pain. Mild constipation with shift work, rare laxatives.
- Denies mental health or eating disorder diagnoses. Non smoker, no THC, no substance use.

- Medications: ondansetron 8mg TDS; PRN codeine, NSAIDs, sumatriptan, diazepam
- Investigations normal:
  - Blood; stool; imaging incl abdo/pelvis, biliary, small bowel, CNS
  - Gastroscopy & colonoscopy incl coeliac and lactase deficiency
  - Gastric emptying study → Dx Gastroparesis → NJ tube x 9months ‘I can’t eat anything’

Progress:

- Gastro diagnosis: chronic nausea vomiting syndrome
  - Multiple factors contributing to delayed gastric emptying (weight loss, medications)

- GI-Psychologist assessment:
  - Discloses issues with body image and restrictive behaviours now
  - And behaviours consistent with bulimia nervosa in teens; homelessness, domestic & family violence
  - No formal diagnosis, BN untreated ‘just got over it’

- Referred on for Psychiatry assessment:
  - New diagnosis complex PTSD
  - Meets criteria for ARFID, possible AN/P, for ongoing assessment

“The concept of the separation of mind and body is dominant and pervasive in Western thinking. This has had profound negative effects on research, patient-care and the patient-physician relationship.”

Prof. Douglas A Drossman
Gastroenterologist & Psychiatrist
Founder Rome Foundation

Thank you.

trina.kellar@health.qld.gov.au

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Irritable Bowel Syndrome (IBS)

Red flags

- Rectal bleeding
- Unintentional weight loss
- Nocturnal or progressive abdominal pain
- Aged > 40 years at first presentation, and symptoms persisting > 6 weeks (especially if female)

Background

About irritable bowel syndrome (IBS)

Assessment

The diagnosis of IBS is clinical – it is not a diagnosis of exclusion.

1. Take a history:
   - Symptoms of IBS – frequency, severity, triggers, exacerbating and relieving factors, impact on quality of life (QOL).
   - Red flags.
   - Psychological symptoms (e.g., stress, depressed or anxious mood).
   - Dietary habits (e.g., adequate fluid intake, fibre intake, irregular or inadequate meals, food triggers).
   - Family history of coeliac disease, inflammatory bowel disease (IBD), IBS, or colorectal or ovarian cancer.
2. Consider other diagnoses.
3. Screen patient for symptoms of stress, anxiety, or depression. Consider using the Kessler Psychological Distress Score (K10) or Depression Anxiety Stress Scale (DASS 21).
4. Examine the patient:
   - Examine abdomen, perianal area, and rectum, and consider gynaecological examination if pelvic condition suspected.
   - Follow recommended protocol and consider a chaperone.
5. Arrange investigations:
   - Consider FBC, ELFTs (include Ca 2+), ESR/CRP, iron studies, thyroid stimulating hormone (TSH), and coeliac serology and IgA level (see Coeliac Disease in Adults).
   - If infection suspected:
     - Consider faecal microscopy, culture and sensitivities (MCS) with PCR (e.g., for *Giardia, Clostridium difficile)*, ova, cysts and parasites (OCP).
     - If more acute, consider faecal viral PCR for norovirus, rotavirus, and adenovirus.
   - Consider faecal calprotectin – most helpful if result is negative (< 50 micrograms/g). May help differentiate IBS from IBD.