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Review article

Centrally mediated disorders of gastrointestinal pain: An update with Rome V and a latin american perspective

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Abstract

Introduction: Disorders of gut-brain interaction are common conditions in which gastrointestinal symptoms result from abnormalities in motility, visceral hypersensitivity, mucosal and immune dysfunction, alterations in the microbiota, and impaired central processing. Rome V includes a category in which the contribution of the central nervous system predominates, with pain, rather than luminal dysfunction, defining the disorder. **Results:** Centrally mediated disorders of gastrointestinal pain comprise three entities: centrally mediated abdominal pain syndrome, adult abdominal migraine, and narcotic bowel syndrome. The most far-reaching change concerns the first of these. Under Rome IV, pain was required to be continuous and unrelated to food intake, defecation, or menstruation; if it also fluctuated with meals, the patient was excluded from the diagnosis and was often classified as having irritable bowel syndrome. Rome V retains this profile while adding a category in which pain may vary in response to physiological events without ceasing to be continuous. The distinguishing feature is therefore pain continuity rather than its relationship to such events. Conversely, pain associated with irritable bowel syndrome is required to be noncontinuous. Diagnostic criteria, epidemiology, mechanisms, diagnosis, and treatment are reviewed. **Conclusions:** Centrally mediated disorders of gastrointestinal pain require a shift in clinical reasoning: rather than searching for a luminal lesion, the focus should be placed on treating an identifiable central mechanism. However, the evidence is heterogeneous and derives from small uncontrolled series; therefore, recommendations should be presented accordingly. In Latin America, prevalent organic disease must be excluded, and management should be tailored to the availability of neuromodulators, psychogastroenterology services, and opioid tapering programs.

Keywords

Abdominal pain; gut-brain axis; hyperalgesia; opioid-related disorders; migraine disorders.

INTRODUCTION

The publication of Rome V comes at a time when gastroenterology can no longer sustain a dichotomous distinction between "organic" and "functional" disorders. This statement applies to all disorders of gut-brain interaction (DGBI) but is particularly relevant to patients whose predominant symptom is chronic abdominal pain without a known luminal cause or identifiable correlate. These patients are easily recognized. They are the individuals who have undergone repeated endoscopies and computed tomography scans, and even laparoscopies, without findings that explain their symptoms. They ultimately present to the gastroenterologist's office with more tests than explanations[1,2].

Rome V recommends definitively replacing the term *functional gastrointestinal disorders* with *disorders of gut-brain interaction* (DGBI). Although the term *functional* has historically been useful, it has carried a significant stigma. For some physicians and patients, it may suggest the absence of organic disease and attribute symptoms to a purely psychological phenomenon or, in the worst-case scenario,

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imply that the symptom is illegitimate[1]. No category is more affected by this change than centrally mediated disorders of gastrointestinal pain, in which the term *functional* has served for decades to close the case rather than initiate a targeted therapeutic plan.

This review describes the changes introduced by Rome V in the category of centrally mediated disorders of gastrointestinal pain, summarizes the available evidence, and discusses how it can be applied in the Latin American context.

METHODS

A narrative search was conducted in MEDLINE/PubMed, Embase, LILACS, and SciELO from January 2016 through July 2026, without language restrictions. The search terms included "centrally mediated abdominal pain," "abdominal migraine," "narcotic bowel syndrome," "opioid-induced hyperalgesia," and "disorders of gut-brain interaction." The Rome V special issue, systematic reviews, and population-based prevalence studies were prioritized. The domains of the SANRA scale were followed[3].

RESULTS

What changes in the category of centrally mediated disorders of gastrointestinal pain with Rome V?

In Rome IV, *functional abdominal pain syndrome* was renamed *centrally mediated disorders of gastrointestinal pain* to reflect the predominant contribution of the central nervous system rather than the intestine[2]. Rome V retains this framework and organizes it into three entities: centrally mediated abdominal pain syndrome; abdominal migraine, now formally recognized in adults; and narcotic bowel syndrome, or opioid-induced gastrointestinal hyperalgesia (NBS) (**Table 1**)[1,2].

Two changes have immediate consequences for clinical practice. The first affects centrally mediated abdominal pain syndrome, which now encompasses two forms. In the first, inherited from Rome IV, continuous pain is unrelated to food intake, defecation, or menstruation. In the second, new in Rome V, pain may worsen after eating or partially improve after defecation but remains continuous. According to the working group, this second form is considerably more common than the first. Until now, it either fell outside the classification or was labeled as irritable bowel syndrome[4]. The second change is reciprocal. The Rome V criteria for irritable bowel syndrome now require pain or discomfort to be noncontinuous, specifically to delineate the boundary between irritable bowel syndrome and centrally mediated pain[4,5].

This adjustment has already been tested. In the first independent validation of the Rome V criteria, conducted in 726 patients referred from primary care to a specialty clinic, sensitivity was 66.1% (95% confidence interval [CI], 62.1%-69.9%) and specificity was 80.1% (95% CI, 72.4%-86.5%), compared with sensitivities of 78.9% (95% CI, 75.4%-82.1%) with Rome IV and 87.5% (95% CI, 84.4%-90.3%) with Rome III. The negative likelihood ratio for Rome V was 0.42 (95% CI, 0.37-0.49). Thus, failure to meet the criteria only slightly reduces the likelihood that a patient actually has irritable bowel syndrome. Of the 200 patients who met the reference standard but were not identified, 142 (71%) were excluded because they reported continuous daily pain[6]. The authors caution that these patients probably do not have centrally mediated

Table 1. Centrally mediated disorders of gastrointestinal pain in Rome V

Entity	Defining feature	Novelty in Rome V	Clinical implication
Centrally mediated abdominal pain	Continuous or nearly continuous pain with an impact on functioning	The classic form is retained, with no relationship to physiological events. A new form is added in which pain changes with food intake or defecation but remains continuous.	Captures a group that previously lacked a diagnostic category
Adult abdominal migraine	Stereotypical paroxysmal episodes with symptom-free intervals	Formally recognized in adults	Opens the way to acute and prophylactic treatment based on a migraine-oriented approach
Narcotic bowel syndrome or opioid-induced gastrointestinal hyperalgesia	Pain that worsens as the opioid dose increases	Further established as an entity distinct from opioid-induced constipation	Treatment consists of planned opioid withdrawal

Table prepared by the authors based on: Drossman DA, et al. *Gastroenterology.* 2026;170(6):1083-1098[1]; Fukudo S, et al. *Gastroenterology.* 2026;170(6):1283-1302[2]; Lembo A, et al. *Gastroenterology.* 2026:S0016-5085(26)07065-4[4].

abdominal pain, in which the pain must be severe and constitute the predominant symptom. Instead, they would be classified as having an unspecified intestinal disorder, a category of limited clinical utility that also excludes them from irritable bowel syndrome trials. The boundary between the two diagnoses therefore remains unresolved. The authors themselves call for simultaneous validation in different countries using identical methodology, given that the clinical phenotype may vary by region.

Centrally mediated abdominal pain syndrome

This syndrome is defined by continuous or nearly continuous abdominal pain that affects daily functioning, is not explained by another structural disorder or disorder of gut-brain interaction, and has no or only occasional relationship to physiological events such as food intake, defecation, or menstruation. The research criteria retain a duration of six months. The clinical criteria, formalized by the Rome Foundation in 2022 and incorporated into the Rome V criteria, allow diagnosis after eight weeks of symptoms when the symptom is sufficiently bothersome and alternative diagnoses have been reasonably excluded[1,7].

Regarding epidemiology, in the Rome Foundation global study using Rome IV criteria, the prevalence of centrally mediated abdominal pain syndrome was 0.02% in internet-based surveys and 0.05% in household surveys[8]. The corresponding Rome IV chapter reported prevalences ranging from 0.5% to 2.1%[9], based on a 1993 U.S. population-based survey that used earlier definitions[10]. The discrepancy is attributable to stricter diagnostic criteria and changes in the definition over time rather than to differences between the populations studied.

Rome V describes mechanisms of hyperalgesia, allodynia, and sensitization at different levels of the neuraxis. The central pain filter, modulated by serotonergic and noradrenergic systems, can amplify or attenuate signals ascending from the gut[11,12]. In centrally mediated abdominal pain syndrome, the imbalance favors amplification. Descending inhibitory modulation fails, and pain no longer requires a proportionate peripheral stimulus to persist. Anxiety, depression, trauma, catastrophizing, and hypervigilance may increase symptom burden, lower perception thresholds, and reduce the therapeutic response[13]. These factors are part of the underlying mechanism and should therefore be therapeutic targets.

Adult abdominal migraine

Abdominal migraine is incorporated into the category of centrally mediated disorders of gastrointestinal pain in adults. Its conceptual value lies in recognizing that some gastrointestinal syndromes are episodic, with symptom-free intervals, and are more closely related to central neural circuits than to a persistent intestinal disorder[2]. Diagnosis requires paroxysmal episodes of severe abdominal pain, either periumbilical or diffuse, lasting at least one hour and separated by weeks or months. The episodes must be disabling and stereotypical for the individual patient and accompanied by at least two of the following: anorexia, nausea, vomiting, headache, photophobia, or pallor. Between episodes, the patient is asymptomatic. This distinguishes abdominal migraine from centrally mediated abdominal pain syndrome and irritable bowel syndrome[1,2].

Evidence in adults is limited. A systematic review conducted according to *PRISMA* included 662 patients from 63 studies, most of which were case reports and case series. The population was defined according to ICHD-3 criteria rather than Rome criteria, and only 33 patients were adults. Among these patients, the median age at onset was 31.0 years (interquartile range [IQR], 19.0-48.0), and the median age at diagnosis was 40.0 years (IQR, 27.0-52.0), with a median diagnostic delay of 4.0 years (IQR, 2.0-8.0). The finding with the greatest clinical relevance is a history of migraine. 27 of the 33 adults (82%) had a personal or family history of migraine, compared with 46.8% in the overall cohort. Associated symptoms included nausea in 66.1%, vomiting in 53.6%, and headache in 47.1% of 448 documented cases. The population was predominantly pediatric. Reported treatment response rates were 98.0% for triptans (50/51), 95.0% for anticonvulsants (19/20), 92.8% for antihistamines (64/69), 100% for β-blockers (12/12), and 88.9% for tricyclics (8/9). These figures were also not stratified by age and, according to the authors, reflect substantial publication and selection biases. Therefore, they do not provide a sufficiently robust basis for recommending any of these therapies as first-line treatment[14].

Narcotic bowel syndrome and opioid-induced hyperalgesia

Narcotic bowel syndrome describes a clinical paradox: chronic or recurrent abdominal pain that worsens, or at least does not improve, as the opioid dose increases. The trajectory of the pain is what guides the diagnosis: progressive dose escalation, increasingly shorter periods of relief, and increasing baseline pain, with exclusion of a structural cause[1,2]. Proposed mechanisms include opioid-induced hyperalgesia mediated by bimodal opioid regulatory systems, glial neuroinflammation, descending facilitation, and interaction with the N-methyl-D-aspartate (NMDA) receptor at the spinal level. None of these mechanisms has been validated as a clinical biomarker[15]. It should be distinguished from opioid-induced constipation and

cannabinoid hyperemesis syndrome, which Rome V classifies among gastroduodenal disorders[16].

Treatment is counterintuitive for patients and requires careful explanation beforehand. The opioid, perceived as the only source of relief, is the agent that triggers and perpetuates the pain. The described strategy combines a therapeutic alliance, education about the underlying mechanism, planned and gradual opioid withdrawal, initiation of a maintenance neuromodulator before dose reduction, symptomatic management of withdrawal, and behavioral therapy[15,17]. The evidence comes from uncontrolled case series and cohort studies. Buprenorphine-naloxone has been described only in case reports and cannot be recommended as a standard treatment.

Positive diagnosis and rational use of diagnostic testing

A positive diagnosis is far removed from diagnosing for convenience. It means conducting a focused evaluation. The medical record should identify a symptom pattern and its relationship to food intake, bowel movements, posture, sleep, medications, previous infections, surgery, cannabis or opioid use, comorbidities, and alarm features. The physical examination retains both clinical and therapeutic value[1]. In a patient with chronic abdominal pain, three questions should structure the evaluation before any diagnostic testing is ordered. First, is there opioid exposure accompanied by dose escalation and progressively shorter periods of relief? If so, the likely diagnosis is narcotic bowel syndrome, and the rest of the diagnostic reasoning changes. Second, is the pain episodic or continuous? Stereotypical episodes separated by symptom-free intervals suggest abdominal migraine. Third, if the pain is continuous, does it change with food intake or defecation? This distinction separates the two forms of centrally mediated abdominal pain syndrome (**Table 2**).

When the symptom pattern is typical, there are no alarm features, a reasonable basic evaluation has been completed, and no relevant abnormalities are identified, repeating diagnostic tests may reinforce the cycle of fear, hypervigilance, and unnecessary resource use that perpetuates these disorders[1,13]. In this category, the harm caused by excessive testing may be greater than in other conditions. Each additional normal test confirms to the patient that no one has found "the cause" and reinforces further diagnostic seeking. The statement "your tests are normal" is rarely sufficient. A more useful formulation would be: "Your symptoms are consistent with a disorder of gut-brain interaction. This means that your gastrointestinal tract and nervous system are processing signals with increased sensitivity, and the condition can be treated in a stepwise manner"[13].

Treatment

Neuromodulators have emerged as a valuable therapeutic option, but they should not be used indiscriminately. In painful DGBI, tricyclic antidepressants (TCAs) are an important option. Nortriptyline and desipramine are generally better tolerated than amitriptyline or imipramine because they have less antihistaminic and anticholinergic activity. Selective serotonin reuptake inhibitors are less useful as analgesic monotherapy but may be beneficial when anxiety is a prominent component[13,17]. In these disorders,

Table 2. Discriminating questions in a patient with chronic abdominal pain

Question	Answer	Clinical guidance
1. Is there opioid exposure accompanied by dose escalation and progressively shorter periods of relief?	Yes	Narcotic bowel syndrome; prioritize planned opioid withdrawal before pursuing other diagnoses
2. Is the pain episodic or continuous?	Episodic, stereotypical, with symptom-free intervals	Adult abdominal migraine; assess for a personal or family history of migraine.
3. If continuous, is the pain related to food intake, defecation, or menstruation?	No, or only occasionally	Centrally mediated abdominal pain, classic form.
4. If continuous, does the pain change with these events while remaining continuous?	Yes	Centrally mediated abdominal pain, form incorporated into Rome V; determine whether the patient has previously been labeled as having irritable bowel syndrome.
5. Are there red flags or relevant regional epidemiologic risk factors?	Yes	Targeted evaluation before establishing the diagnosis.

Table prepared by the authors based on: Drossman DA, et al. *Gastroenterology.* 2026;170(6):1083-1098[1]; Fukudo S, et al. *Gastroenterology.* 2026;170(6):1283-1302[2]; Lembo A, et al. *Gastroenterology.* 2026:S0016-5085(26)07065-4[4].

neuromodulators constitute first-line treatment because the therapeutic target is central pain processing. When the response to a single agent is partial, the Rome Foundation report proposes combining two agents with different mechanisms of action at moderate doses rather than escalating the dose of a single agent. Serotonin-norepinephrine reuptake inhibitors (SNRIs) are a reasonable alternative when somatic pain and affective comorbidity coexist, although their specific evaluation in chronic gastrointestinal pain is more limited (**Table 3**)[17].

Gut-brain therapies include gut-directed cognitive behavioral therapy, hypnotherapy, mindfulness, and other interventions aimed at reducing visceral fear, hypervigilance, avoidance, anxiety, and a diminished sense of control. Their advantages include safety, persistence of benefit beyond the intervention period, and potential cost-effectiveness. Their limitations include availability, treatment duration, patient motivation, and access to trained therapists[13]. A cross-cutting principle in this category is to redefine the treatment outcome. The goal will rarely be complete elimination of pain. Instead, it should be recovery of function, fewer emergency visits, opioid withdrawal, and the ability to return to work, sleep, and eat without constant monitoring. Making this explicit from the first visit helps prevent treatment discontinuation.

Applicability in Latin America

When establishing a positive diagnosis of a centrally mediated disorder, regionally prevalent organic disease should not be overlooked. Chagas disease, caused by *Trypanosoma cruzi*, is a key example. It primarily affects countries in Central and South America, and its chronic manifestations may involve the heart, esophagus, colon, and nervous system[18]. An estimated 6 to 7 million people are affected worldwide[19]. In a patient with continuous chronic abdominal pain, longstanding constipation, megacolon, or esophageal dilation, the epidemiologic history, place of birth, rural residence, migration from an endemic area, and previous blood transfusions alter the sequence of the evaluation. Manometric characterization should be performed according to the current criteria of the Chicago Classification[20]. A reasonable diagnostic workup differs in Bogotá, Mexico City, or São Paulo from that in a European center.

Rome V recognizes the importance of physiological testing, psychogastroenterology, specialized nutritional care, and multidimensional models. However, in many countries, these resources are concentrated in a small number of centers. The consequence should not be abandonment of Rome V but rather its use within a stepwise framework[21,22]. For the category of centrally mediated disorders of gastrointestinal pain, a stepwise approach is more feasible than it may appear because treatment depends primarily on the time devoted to the clinical encounter, the availability of neuromodulators, and continuity of follow-up. The actual bottleneck is psychogastroenterology. Gut-directed cognitive behavioral therapy and hypnotherapy have supporting evidence, but there are very few trained therapists in the region.

Narcotic bowel syndrome warrants particular consideration. Opioid consumption per capita in Latin America is substantially lower than in North America. This likely explains why narcotic bowel syndrome is infrequently

Table 3. Therapeutic approach by entity

Entity	First-line therapy	Alternatives or adjunctive therapies	Precautions
Centrally mediated abdominal pain	Low-dose tricyclic antidepressant, titrated according to response and tolerability	Augmentation with a second agent with a different mechanism of action; SNRI if somatic pain or affective comorbidity is present; gut-brain behavioral therapies	Anticholinergic effects and sedation; favor combination therapy over escalating the dose of a single agent
Adult abdominal migraine	Acute treatment of the episode using a migraine-oriented approach	Prophylaxis with a β-blocker, anticonvulsant, antihistamine, or tricyclic antidepressant if episodes are frequent	Evidence is derived from case reports and case series, predominantly in pediatric populations and without age-specific stratification; cardiovascular contraindications to triptans
Narcotic bowel syndrome	Planned, gradual opioid withdrawal, with prior explanation of the underlying mechanism	Neuromodulator initiated before dose reduction; symptomatic management of withdrawal; behavioral therapy	Requires a therapeutic alliance and close follow-up; high risk of treatment discontinuation; buprenorphine-naloxone has been described only in case reports

SNRIs: serotonin-norepinephrine reuptake inhibitors. Table prepared by the authors based on: Drossman DA, et al. *Gastroenterology.* 2026;170(6):1083-1098[1]; Fukudo S, et al. *Gastroenterology.* 2026;170(6):1283-1302[2]; Ding K, et al. *Front Neurol.* 2026;17:1760307[14]; Farmer AD, et al. *Lancet Gastroenterol Hepatol.* 2017;2(5):361-368[15]; Drossman DA, et al. *Gastroenterology.* 2018;154(4):1140-1171.e1[17].

recognized in the region, but it does not mean that the condition does not occur. It also coexists with the opposite problem: undertreatment of cancer pain. Both phenomena require local data that are currently unavailable.

CONCLUSIONS

Centrally mediated disorders of gastrointestinal pain represent the setting in which the conceptual proposal of Rome V is most rigorously tested because these disorders lack both a biomarker and a demonstrable lesion. Diagnosis therefore rests entirely on the characteristics of the pain. Recognizing that continuous pain may change with food intake or defecation fills a genuine gap in the classification. Conversely, requiring that pain in irritable bowel syndrome not be continuous makes explicit a boundary that was previously drawn according to each clinician's individual judgment.

In summary, Rome V does not seek to eliminate the complexity of DGBI. Rather, it provides a framework for organizing it: to stop relentlessly searching for an absent lesion and begin treatment with clear communication and multidisciplinary tools because the gut-brain axis is indeed affected and requires management. At the same time, it is important to be explicit about what remains unknown. There are no biomarkers. Therapeutic evidence for adult abdominal migraine and narcotic bowel syndrome comes from case reports and case series without control groups. The diagnostic performance of the new criteria has been evaluated only at a single center, with unfavorable results for sensitivity. Stating this level of evidence allows clinicians to distinguish situations in which they can act with confidence from those in which they are testing a treatment that must be monitored and discontinued if it proves ineffective.

Conflicts of interest

The authors declare no conflicts of interest.

Use of artificial intelligence

Claude (Anthropic) was used as an assistance tool for the critical revision of the manuscript and for verification of data and references against primary sources. The authors reviewed, verified, and edited all content produced with this assistance and assume full responsibility for the accuracy and integrity of the manuscript and its conclusions. The tool is not listed as an author.

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