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All Roads Lead to Rome: Update on Rome III Criteria and New Treatment Options

David Q. Shih, MD[1,2] and **Lola Y. Kwan, MD**[1]

[1]Cedars-Sinai Inflammatory Bowel Disease Center, Los Angeles, California, Los Angeles

[2]Department of Medicine, Division of Digestive Diseases, University of California, Los Angeles

Abstract

The recently published Rome III criteria reflect current understanding of functional gastrointestinal disorders. These criteria include definitions of these conditions and their pathophysiologic subtypes and offer guidelines for their management. At the 2006 Annual Scientific Meeting of the American College of Gastroenterology, a panel of experts discussed these criteria as they pertain to irritable bowel syndrome, functional dyspepsia, and chronic constipation. This article reviews the panel's findings, highlights the differences between the Rome II and III criteria, and summarizes best treatment options currently available to practitioners and their patients.

Functional gastrointestinal (GI) disorders, such as irritable bowel syndrome (IBS), functional dyspepsia, and chronic constipation, pose an extensive healthcare burden and negatively affect quality of life. The total cost of caring for an IBS patient may reach thousands of dollars; in fact, symptoms of IBS are second only to the common cold for causing work absenteeism. 1 Similarly, patients and their insurers spend considerable amounts of money for the diagnosis and treatment of chronic constipation, an uncomfortable and distressing condition for which few satisfactory therapies are available.2,3

Over the past 15 years, the evolving definition of functional bowel disorders has been driven by advanced understanding of symptom patterns. Currently, functional GI disorders are classified into six major domains according to anatomical distribution, from the esophagus to anorectum. These disorders are believed to be caused by disturbed motor and sensory function, altered immune function and inflammation, and dysregulation of the central and enteric nervous systems. Consensus-based definitions spearheaded by the Rome committee for various functional GI disorders were launched in May of this year during Digestive Disease Week 2006.

During the annual scientific meeting of the American College of Gastroenterology, held in Las Vegas, Nevada, October 20–25, 2006, a panel of experts reviewed the Rome III criteria and their recommended therapeutic interventions. Participants included Nicholas J. Talley, MD, PhD, FACG, Professor of Medicine at the Mayo Clinic College of Medicine, Rochester; Minnesota; Charlene M. Prather, MD, MPH, Associate Professor of Internal Medicine at St. Louis University, Missouri; and Satish S.C. Rao, MD, PhD, FACG, Professor of Internal Medicine and Director of Neurogastroenterology and GI Motility at the University of Iowa Hospital and Clinic, Iowa City.

Irritable Bowel Syndrome

Irritable bowel syndrome affects about 10%–20% of adolescents and adults; it predominantly occurs in females. 4,5 As with other functional GI disorders, treating the symptoms of IBS is expensive in terms of dollars and patient discomfort and anguish.6,7

Rome III Criteria for IBS

Irritable bowel syndrome is characterized by abdominal pain or discomfort associated with disturbed defecation or a change in bowel habit (Table 1).8 Based upon bowel patterns at a particular point in time, the disorder may be categorized further into four groups (Table 2).8 An individual with symptoms that do not correlate with either diarrhea- or constipation-predominant IBS is categorized as having either mixed IBS (meeting criteria for both diarrhea- and constipation-predominant IBS) or unsubtyped IBS (insufficient abnormality of stool consistency to meet any of the three types). Alarming symptoms may suggest involvement of structural or biochemical abnormalities, yet such findings are not required as exclusionary criteria for the condition's diagnosis.8

Historically, variations in patient and physician perception have made the diagnosis of "constipation" and "diarrhea" difficult. For example, straining to defecate may occur with soft or watery stools; on the other hand, the stool may be solid, yet defecation is frequent.9 Stool form reflects intestinal transit time, and the Rome committee recommends use of the Bristol Stool Form Scale to classify patients into the four IBS subtypes (Figure 1).8–10 This scale considers constipation to be related to IBS types 1 and 2 and diarrhea to be linked with IBS types 6 and 7.8,10

Pathophysiology

Mechanisms that explain the presence of visceral hypersensitivity and GI motor disturbances in IBS are emerging. Some recent studies point to postinfectious changes, inflammation, bacterial overgrowth, and neurotransmitter alteration as potential mechanisms of IBS.

Postinfectious and Inflammatory IBS—IBS may follow a bout of bacterial gastritis. For example, Marshall and others11 reported a higher incidence of IBS among individuals exposed to municipal water contamination than among controls (27.5% vs 10.1%, respectively; $P < 0.01$). Furthermore, the incidence of IBS was even higher among individuals with clinically documented gastroenteritis than among controls (36.2% vs 10.1%; $P < 0.01$). Mearin et al12 found that the relative risk of developing IBS after *Salmonella* gastroenteritis increased by eightfold over the subsequent year, thereby supporting the role of postinfectious gastroenteritis in the development of IBS.

Some IBS patients also have subtle gut mucosal inflammation and immune activation. For example, an increase in mast cells, neutrophils, natural killer cells, eosinophils, and intraepithelial lymphocytes has been recorded among individuals with documented postinfectious and non-postinfectious IBS when compared with controls.13,14 Such increased immune activation also is manifested as a shift toward inflammatory cytokine profiles with an abnormal interleukin (IL)-10/IL-12 ratio and an increased level of IL-1β.15,16

Bacterial Overgrowth—The GI motor disturbances seen in IBS may lead to qualitative or quantitative changes in bacterial flora that promote bacterial overgrowth and increase bacterial fermentation/gas. This production of excess gas induces such symptoms as abdominal discomfort and bloating.17

The association between bacterial overgrowth and IBS is supported by studies showing improved IBS symptoms after antibiotic treatment. For example, a recent double-blind, randomized, placebo-controlled study by Pimentel et al18 assigned participants meeting the Rome I IBS criteria to receive either 400 mg of rifaximin three times daily for 10 days or placebo. The rifaximin-treated patients achieved a significant global IBS symptom improvement (36%) when compared with the placebo group (21%).

Promising evidence supports the role of bacterial overgrowth in IBS and eradication of such colonies for management of the disorder. However, the expert panel warned physicians to accept this approach cautiously, noting that more work to confirm these observations is needed.

Neurotransmitter Alteration and IBS—Alterations in neurotransmitters may lead to visceral hypersensitivity and GI motor disturbances. In particular, serotonin, or 5-hydroxytryptamine (5-HT), and its effects on the GI system are the subject of active research. Biologically, alterations in the levels or sensing mechanism of serotonin may lead to IBS, since this neurotransmitter is a major regulator of the peristaltic reflex and sensory relays in the gut.19

Two studies support this claim. Whereas Dunlop's team20 reported that the release of serotonin fell among patients with constipation-predominant IBS and rose in individuals with the diarrhea-predominant form of the disorder, Coates et al21 showed that IBS patients exhibited lower levels of gut mucosal serotonin and serotonin reuptake transporter than did controls.

Updates in IBS Treatment

Initial IBS management includes education, reassurance, and investigation of psychosocial issues.21

Helping Patients Help Themselves—In a randomized study, Robinson's team22 found that introducing a self-help guidebook to IBS patients resulted in a 60% reduction in their primary-care consultations ($P < 0.001$) and a drop in their perceived symptom severity ($P < 0.001$) when compared with controls.

The expert panel recommended the following resources for IBS patients:

- http://www.acg.gi.org/patientinfo/ibsrelief
- http://www.iffgd.org
- http://www.aboutibs.org
- http://www.med.unc.edu/medicine/fgidc

Pharmacologic Therapy—Drug therapy mainly targets IBS symptoms. The types of drugs available are divided into three main categories (Figure 2).23

Fiber supplements: According to anecdotal reports, some patients with diarrhea experience a firming of stools after using fiber supplements; however, some studies showed no clear benefit with use of these products.24–26 When prescribing a fiber supplement to patients with diarrhea, start at a low dose and progressively titrate the dose up as tolerated to minimize the bloating that patients frequently experience.

Loperamide: Loperamide is a μ-opioid-receptor agonist that reduces intestinal secretion, slows colonic transit, and increases resting anal sphincter tone.27 When compared with placebo, loperamide reduces diarrhea in IBS patients with this predominant symptom.25,26

Opioid-receptor activation reduces visceral pain via peripheral (spinal afferents) and central mechanisms; however, loperamide has not been shown to alter abdominal pain or other IBS symptoms when compared with placebo.25,26

Alosetron: 5-HT plays an important role in visceral sensitivity, gut absorption/secretion, and motility.19 The 5-HT type 3 (5-HT_3) receptor antagonists may be useful in reducing colonic transit time and in treating diarrhea.

There is convincing evidence that a drug in this class, alosetron, may delay colonic transit time.28,29 A meta-analysis of randomized trials showed that alosetron is effective against diarrhea-predominant IBS.30 However, because of its suspected side effects (eg, ischemic colitis, colonic ischemia), this drug is only approved for restricted use.31,32 Recently, a meta-analysis showed that the incidence of ischemic colitis related to use of the drug is low (1.1 per 1,000 patient-years of alosetron use) and is rarely associated with long-term sequelae or serious morbidity.33

Functional Dyspepsia

Functional dyspepsia is a clinical syndrome characterized by chronic or recurrent upper abdominal pain or discomfort having no identifiable cause.34 This disorder is not a monosymptomatic entity; several predominant symptoms may be present, including epigastric pain (22%), abdominal fullness (24%), bloating (15%), vomiting (3%), belching (8%), early satiety (12%), nausea (10%), and heartburn (6%).35 These predominant symptoms change over time, making functional dyspepsia difficult to define and classify.34

Rome III Criteria for Functional Dyspepsia

After reviewing available evidence, the Rome committee proposed updated diagnostic criteria for functional dyspepsia. As shown in Table 3,34 the Rome III criteria definition of functional dyspepsia includes at least one of the following: bothersome postprandial fullness, early satiation, epigastric pain, and epigastric burning; patients also must have no evidence of structural disease that would likely explain their symptoms, including upper endoscopic findings. The patient must meet the criteria for 3 months and must begin experiencing symptoms for at least 6 months before diagnosis.34

Functional dyspepsia may be divided into postprandial distress syndrome (characterized by postprandial fullness and/or early satiation) and epigastric pain syndrome (characterized by epigastric pain). Dyspepsia is considered to be a continuum of various gastric complaints; thus, a diagnosis of functional dyspepsia does not preclude the inclusion of gastroesophageal reflux disease (GERD) and/or IBS in its symptom complex.34

Updates in Treatment of Functional Dyspepsia

Initial management of functional dyspepsia includes reassurance, education, smoking cessation, consumption of several small and low-fat meals each day, and avoidance of coffee, alcohol, and nonsteroidal anti-inflammatory agents; however, no evidence exists that these interventions are effective.36 Other treatment modalities include psychotherapy, cognitive-behavioral therapy, and hypnotherapy; although a few studies have shown benefit of psychological therapy in functional dyspepsia,37 additional studies must be done to establish its efficacy.

Medical treatment options for functional dyspepsia remain limited, and available studies do not address the newly defined subgroups of epigastric pain syndrome and postprandial distress syndrome. In addition, up to 60% of patients respond to placebo, which further limits tests of pharmaceutical effectiveness in this patient population.38

Gastric Acid Suppression and *Helicobacter pylori* Eradication—Several lines of compelling evidence point to gastric acid suppression as the first-line drug therapy for functional dyspepsia. A Cochrane systematic review39 showed histamine-2 (H_2)-receptor

antagonists and proton-pump inhibitors to be superior to placebo in managing the disorder. Another meta-analysis of controlled, randomized trials40 both confirmed this finding and showed acid suppression to be cost-effective in treating functional dyspepsia. The benefit of acid suppression against functional dyspepsia may be related to unrecognized GERD or nonerosive reflux disease.40–42

Eradication of *H pylori* also appears to help treat functional dyspepsia. Another Cochrane systematic review43 concluded that *H pylori* eradication therapy has a statistically significant effect in treating *H pylori*-positive, nonulcer dyspepsia, and an economic model suggested that *H pylori* eradication also may be cost-effective. Thus, *H pylori* eradication is recommended for functional dyspepsia.

Antidepressants—Few studies have investigated the effectiveness of antidepressants against functional dyspepsia. A small, randomized, crossover trial of seven patients showed that 50 mg of amitriptyline increased tolerance to aversive visceral sensations when compared with placebo administration.44

Despite the limited data, treatment with tricyclic antidepressants (eg,10–25 mg of imipramine or desipramine at night) or a selective serotonin reuptake inhibitor (eg, 10 mg of escitalopram or 20 mg of sertraline in the morning) is recommended after standard therapy with a proton-pump inhibitor or *H pylori* eradication fails.45

Prokinetic Agents—Prokinetic agents, such as metoclopramide, domperidone, and cisapride, reduce symptoms of functional dyspepsia. These drugs have proven more effective than has placebo against the disorder, especially in patients presenting with predominant symptoms of fullness, bloating, or nausea.39,46

However, patients must be monitored closely when using these agents. For example, metoclopramide must be used cautiously in the elderly because of undesired side effects (eg, tardive dyskinesia).47 Domperidone is not available in the United States because of its risk of cardiotoxicity48; cisapride was withdrawn from the US market because it caused QT-interval prolongation and rare cases of fatal arrhythmia.49

5-HT Agonists/Antagonists—The 5-HT pathway is important to visceral sensitivity, so its alteration may treat functional dyspepsia effectively.27 One randomized, controlled trial showed that alosetron caused significantly greater reductions in the severity and frequency of functional dyspepsia symptoms than did placebo ($P < 0.001$).50

Because a link between alosetron and ischemic colitis was suggested, the drug is not used for functional dyspepsia; however, no causal relationship between the two has been established.51 However, a recent meta-analysis concluded that alosetron use is rarely associated with serious morbidity and is associated with a low incidence of ischemic colitis (1.1 cases per 1,000 patient-years of alosetron use).33

Chronic Constipation and Functional Anorectal Disorders

There have been advances in the understanding of symptom patterns of chronic constipation and functional anorectal disorders, and updated criteria have been based on new scientific evidence. These criteria are designed to enhance clinical recognition and develop better scientific understanding of these disorders and to standardize their management.

Rome III Criteria for Functional Constipation

Functional constipation presents as persistently difficult, infrequent, and seemingly incomplete defecation that does not fulfill the IBS criteria. The subjective and objective criteria for functional constipation are summarized in Table 4.8

A comparison of the Rome II criteria and the Rome III criteria shows two main changes. First, the more recent criteria are consistent with those for other functional bowel disorders, as the frequency of bowel movements now is > 25% instead of ≥ 25%. Second, the newer criteria permit laxative-induced loose stools when treating functional constipation, because studies using Rome II criteria yielded a lower prevalence of the disorder than did those using Rome I.52

Functional Anorectal Disorders

Functional anorectal disorders include fecal incontinence, anorectal pain, and disorders of defecation; they are defined by specific symptoms, as shown in Table 5.53

Rome III Criteria for Functional Fecal Incontinence—Functional fecal incontinence is defined as uncontrolled leakage of fecal material for at least 3 months in an individual over 4 years of age (Table 6).53 Because it is unclear when the passage of flatus is abnormal, leakage of flatus alone should not be considered a symptom of fecal incontinence.

In general, the spectrum of functional incontinence is broader than that of fecal incontinence. According to the Rome III criteria, functional fecal incontinence may be associated with such organic disorders as dementia, multiple sclerosis, and Crohn's disease, because the relationship between structural disturbances and the disorder is unclear and asymptomatic patients may have small anal sphincter defects. However, if anal sphincter electromyography demonstrates abnormal innervation (eg, pudendal neuropathy), organic diseases that may lead to denervation/ reinnervation changes (eg, dementia, multiple sclerosis, and diabetes) are excluded as the cause of incontinence.53

Chronic Proctalgia—Chronic proctalgia and proctalgia fugax are the two functional anorectal pain disorders. Chronic proctalgia may be broken down into levator ani syndrome (tenderness during posterior traction on the puborectalis muscle) or unspecified anorectal pain, depending upon the presence or absence of puborectalis tenderness during digital rectal examination (Table 7).53 The distinction between these two types is emphasized by modified nomenclature featured in the Rome III criteria.

Proctalgia Fugax—Proctalgia fugax is distinguished from chronic proctalgia by the duration, frequency, and character of the pain. This disorder is manifested as a sudden, severe pain in the anal area that lasts several seconds to minutes and then disappears completely (Table 8).8

For research purposes, the diagnostic criteria for proctalgia fugax must be fulfilled for 3 months or more. However, in clinical practice, its diagnosis, evaluation, and treatment may take place before 3 months pass.53

Functional Defecation Disorders

Functional defecation disorders are defined as paradoxical contraction or inadequate relaxation of the pelvic floor muscles along with fulfillment of the Rome III criteria for functional constipation (Table 9).53 This anorectal disorder may be broken down further as either dyssynergic defecation (inappropriate pelvic floor contraction or sphincter relaxation with adequate propulsive forces during attempted defecation) or inadequate defecatory

propulsion (inadequate propulsive forces during attempted defecation with or without dyssynergic defecation).

According to both the Rome II and Rome III criteria, the diagnosis for functional defecation disorders requires both abnormal diagnostic test results and the presence of defecation symptoms53; a list of symptoms alone does not distinguish patients with functional defecatory disorders from those with functional constipation. Further, the diagnostic criteria for dyssynergia also are the same for both maladies. However, studies using balloon expulsion and rectal barium evacuation showed that inadequate rectal propulsive force causes impaired evacuation54,55; thus, the revised criteria highlight the possibility that functional defecation disorders may be caused by inadequate propulsive forces.

Updates in Treatment of Chronic Constipation and Functional Anorectal Disorders

As illustrated in Figure 3,56 a pathophysiologic approach to managing chronic constipation is recommended. Classes of medications marketed to manage chronic constipation include bulking agents, osmotic and stimulatory laxatives, stool softeners, lubricants, and newer receptor-based therapies (eg, tegaserod, lubiprostone, and alvimopan). However, patients with dyssynergic defecation may benefit more from biophysical therapies than from pharmacotherapy.

The literature shows no clear benefit when chronically constipated patients used bulking agents (eg, psyllium, calcium polycarbophil, methylcellulose, bran, aloe vera),24 stimulant laxatives (eg, bisacodyl, senna),57 or stool softeners (eg, docusate sodium, docusate calcium).57,58 However, several high-quality, placebo-controlled, randomized trials have shown osmotic laxatives (eg, polyethylene glycol solutions) and lactulose to be effective in this population.57,59,60

Serotonin-Receptor Agonists/Antagonists—Approximately 90% of serotonin is found within the enterochromaffin cells of the GI tract.61 When 5-HT interacts with 5-HT_3 and 5-HT type 4 (5-HT_4) receptors, it promotes peristalsis and modulates fluid content of the stool and visceral sensation via 5-HT_4 receptors.62 Such findings resulted in the development of 5-HT_4 receptor agonists.

Tegaserod, a partial 5-HT_4 receptor agonist, was approved by the US Food and Drug Administration (FDA) for long-term treatment of chronic idiopathic constipation in men and women younger than 65 years of age. Kamm and others63 performed a placebo-controlled, randomized clinical trial that compared placebo with 2 mg and 6 mg of tegaserod given twice daily to patients with chronic constipation (defined as fewer than three complete spontaneous bowel movements per week). At 4 weeks, a significantly greater number of spontaneous bowel movements were noted among patients receiving 2 mg (41.4%) or 6 mg (43.2%) of tegaserod twice daily than among the placebo group (25.1%; $P < 0.0001$). These results were confirmed by a similarly designed, international, multi-center study.63 The high-quality designs of such trials and reproducibility of the findings led to tegaserod receiving a “grade A” recommendation for treatment of chronic constipation from two systematic reviews.57,58

Chloride-Channel Activators—Chloride channels are located on the apical surfaces of the gut mucosal epithelial cells. As negatively charged chloride ions enter the intestinal lumen through these channels, so do sodium ions and water passively to maintain isoelectric neutrality.64

Lubiprostone, a bicyclic fatty acid, is the first chloride-channel activator to be approved by the FDA. In a randomized clinical trial that compared placebo with 24 μg of lubiprostone

given twice daily to patients with chronic constipation for up to 4 weeks, Johanson's team65 found the drug to effectively increase the number of spontaneous bowel movements, decrease straining, and improve stool consistency when compared with placebo ($P < 0.002$ for all outcome measurements). Johanson et al66 also showed that lubiprostone improved abdominal bloating, discomfort, and severity of constipation when compared with baseline examination for up to 24 weeks ($P < 0.001$).

Adverse effects associated with lubiprostone therapy included nausea, diarrhea, headache, and abdominal pain.

Peripheral μ-Opioid Antagonists—Another class of medications being tested against chronic constipation, peripheral μ-opioid antagonists may be particularly useful in managing opioid-induced constipation. In initial physiologic studies, a drug in this class, alvimopan, accelerated whole gut transit time and reversed the constipating and gut-slowing effects of codeine phosphate when compared with placebo at 24 and 48 hours ($P < 0.05$).67

Biofeedback Therapies—Functional defecation disorders also may be managed by pelvic floor training or biofeedback therapies.68–71 Chiarioni and others72 recently reported that biofeedback sessions were more effective than were continuous polyethylene glycol for treating pelvic floor dyssynergia (80% vs 22% improvement, respectively; $P < 0.001$), noting that the benefits of biofeedback persisted for at least 2 years. Furthermore, this method also produced greater reductions in straining, sensations of incomplete evacuation and anorectal blockage, use of enemas and suppositories, and abdominal pain ($P < 0.01$ for all parameters). The superiority of biofeedback therapy to alternative treatments for patients with pelvic floor dyssynergia-type constipation also was confirmed by two other randomized controlled trials.73,74

Conclusion

The symptoms of functional bowel disorders vary and may include both GI and extraintestinal complaints. Alarm symptoms suggest the possibility of structural disease, but they do not exclude a diagnosis of functional bowel disorder. Thus, the patient's diagnosis should be based on a symptom complex that fulfills the accepted Rome criteria.

Over the past 15 years, the definition of functional bowel disorder has evolved with advances in the understanding of symptom patterns; currently, this knowledge is reflected in the Rome III criteria. Historically, management of functional bowel disorders has been frustrating; today, however, it is possible to design effective therapies using a pathophysiologic approach.

The cure of functional bowel disorders ultimately requires scrutiny of basic research results to better understand the underlying pathogenesis. With time, evidence-based therapy will help us to specifically target these physical conditions—and not just their symptoms.

Biographies

Dr. Shih is a Gastroenterology Fellow at Cedars-Sinai Inflammatory Bowel Disease Center and the University of California, Los Angeles.

Dr. Kwan is a Resident in Internal Medicine at Cedars-Sinai Medical Center, Los Angeles, California.

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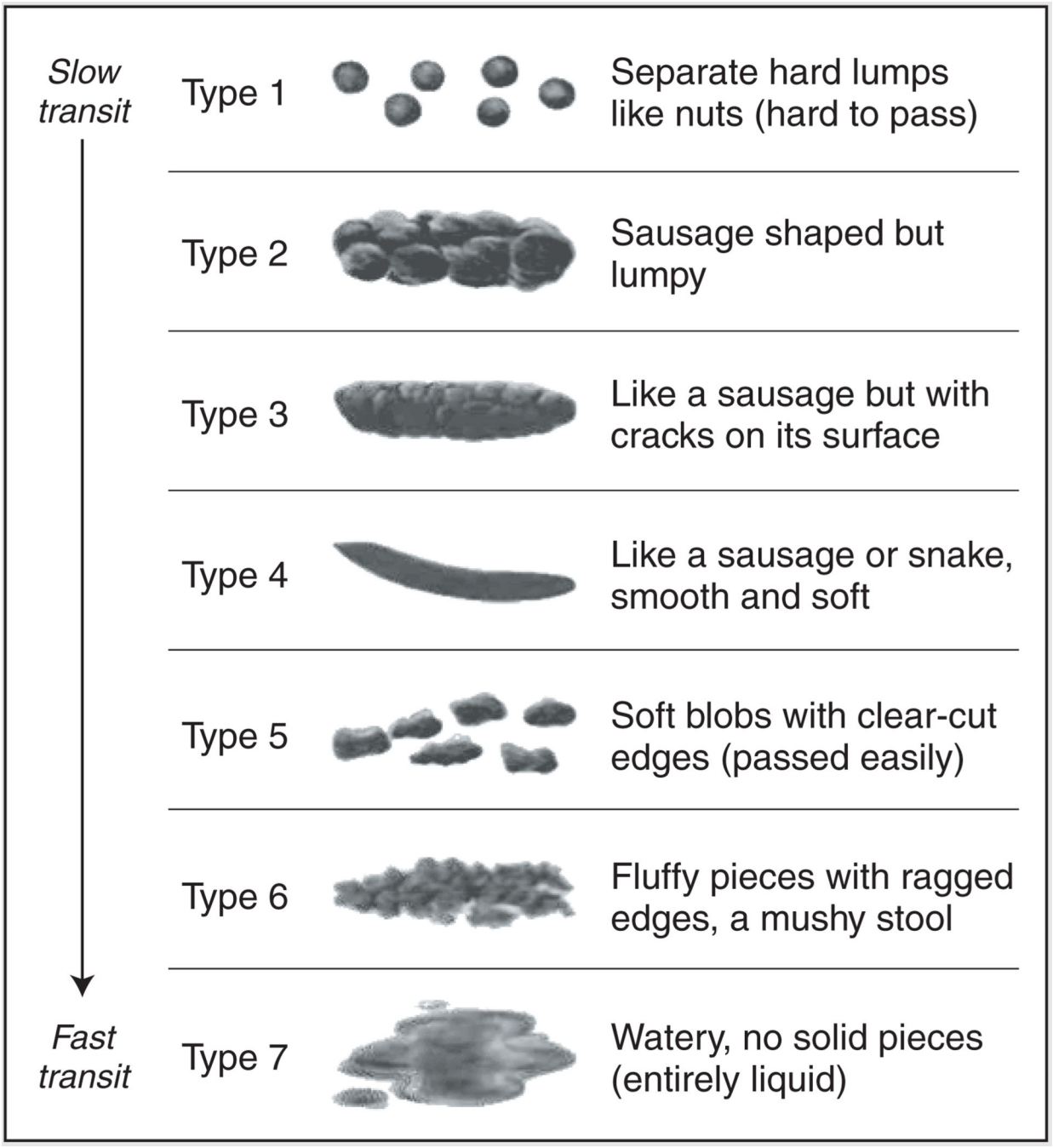

Figure 1.
Bristol Stool Form Scale. Adapted from Lewis and Heaton.10

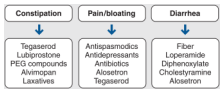

Figure 2.
Pharmacologic therapy for irritable bowel syndrome. PEG = polyethylene glycol. Adapted from Prather.23

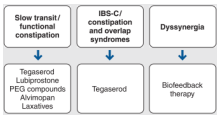

Figure 3.
Pathophysiology-based treatment for chronic constipation. IBS-C = constipation-predominant irritable bowel syndrome; PEG = polyethylene glycol. Adapted from Rao.56

Table 1

Rome III Diagnostic Criteria* for Irritable Bowel Syndrome

Recurrent abdominal pain or discomfort† more than 3 days per month over the previous 3 months associated with two or more of the following: • Improvement with defecation • Onset associated with a change in frequency of stool • Onset associated with a change in form or appearance of stool

*Symptom onset greater than 6 months prior to the diagnosis, with the above criteria fulfilled for the past 3 months

†Discomfort means an uncomfortable sensation not described as pain

Adapted from Longstreth et al8

Table 2

Irritable Bowel Syndrome (IBS) Subtypes by Predominant Stool Pattern

1	IBS with constipation (IBS-C): hard or lumpy stool* with at least 25%, and loose or watery stool† with less than 25%, of bowel movements‡
2	IBS with diarrhea (IBS-D): loose or watery stool† with at least 25%,and hard or lumpy stool* with less than 25%, of bowel movements‡
3	Mixed IBS (IBS-M): hard or lumpy stool* with at least 25%, and loose or watery stool† with at least 25%, of bowel movements‡
4	Unsubtyped IBS: insufficient abnormality of stool consistency to meet criteria for IBS-C, -D, or -M‡

*Bristol Stool Form Scale 1–2

†Bristol Stool Form Scale 6–7

‡In the absence of antidiarrheals or laxatives

Adapted from Longstreth et al8

Table 3

Rome III Diagnostic Criteria for Functional Dyspepsia

Symptom onset greater than 6 months prior to the diagnosis, with the following criteria fulfilled for the past 3 months:

- No structural disease by upper endoscopy to explain the symptoms
- At least one of the following symptoms:
    - **a.** Bothersome postprandial fullness
    - **b.** Early satiation
    - **c.** Epigastric pain
    - **d.** Epigastric burning

Postprandial Distress Syndrome

At least one of the following:

- Bothersome postprandial fullness after ordinary sized meals occurring at least several times a week
- Early satiation that prevents finishing a regular meal at least several times a week

Epigastric Pain Syndrome

Must include all of the following:

- Epigastric pain or burning at least once a week
- Intermittent pain
- Not generalized or localized to other abdominal or chest regions
- Not relieved by defecation or passage of flatus
- Not related to gallbladder or sphincter of Oddi disorders

Adapted from Tack et al34

Table 4

Rome III Diagnostic Criteria for Functional Constipation

Symptom onset more than 6 months prior to the diagnosis, with the following criteria fulfilled for the past 3 months:

- Loose stools rarely present without the use of laxatives
- Insufficient criteria met to establish a diagnosis of irritable bowel syndrome
- Two or more of the following criteria must be met:
  - **a.** Less than three bowel movements per week
  - **b.** Manual maneuvers necessary to facilitate defecation more than 25% of the time.
  - **c.** Hard or lumpy stools more than 25% of the time
  - **d.** Sensation of incomplete evacuation more than 25% of the time
  - **e.** Sensation of anorectal obstruction more than 25%of the time
  - **f.** Straining with defecation more than 25% of the time.

Adapted from Longstreth et al8

Table 5

Functional Anorectal Disorders

Functional fecal incontinence

Functional anorectal pain

- Chronic proctalgia
  - **a.** Levator ani syndrome
  - **b.** Unspecified functional anorectal pain
- Proctalgia fugax

Functional defecation disorders

- Dyssynergic defecation
- Inadequate defecatory propulsion

Adapted from Bharucha et al53

Table 6

Rome III Diagnostic Criteria for Functional Fecal Incontinence

Recurrent uncontrolled passage of fecal material in a patient at least 4 years of age and more than one of the following:

- Abnormal functioning of normally innervated and structurally intact muscles
- Minor abnormalities of sphincter structure and/or innervation
- Normal or disordered bowel habits: fecal retention or diarrhea
- Psychological causes

Symptoms must persist for over 3 months

Exclusion of all of the following:

- Abnormal innervation caused by lesion(s) within the brain (eg, dementia), spinal cord or sacral nerve roots or mixed lesions (eg, multiple sclerosis), or generalized peripheral or autonomic neuropathy (eg, diabetes)
- Anal sphincter abnormalities associated with a multisystem disease (eg, scleroderma)
- Structural or neurogenic abnormalities believed to be the primary cause of fecal incontinence

Adapted from Bharucha et al53

Table 7

Rome III Diagnostic Criteria for Chronic Proctalgia

Symptom onset more than 6 months prior to the diagnosis, with all of the following criteria fulfilled for the past 3 months: • Chronic or recurrent rectal pain or aching • Episode lasting for longer than 20 minutes • Exclusion of other causes of rectal pain, including ischemia, inflammatory bowel disease, cryptitis, intramuscular abscess and fissure, hemorrhoids, prostatitis, and coccygodynia
Levator Ani Syndrome
Fulfilled criteria for chronic proctalgia and tenderness during posterior traction on the puborectalis
Unspecified Functional Anorectal Pain
Fulfilled criteria for chronic proctalgia but no tenderness during posterior traction on the puborectalis

Adapted from Bharucha et al53

Table 8

Rome III Diagnostic Criteria for Proctalgia Fugax

Must include all of the following criteria:
• Recurrent anal or lower rectum pain episodes
• Episodes last from seconds to minutes
• No anorectal pain between episodes
For research purposes: criteria must be fulfilled for 3 months
Clinical practice: diagnosis and evaluation may be made before 3 months

Adapted from Longstreth et al8

Table 9

Rome III Diagnostic Criteria for Functional Defecation Disorders

Symptom onset more than 6 months prior to the diagnosis, with all of the following criteria fulfilled for the past 3 months:

- Diagnostic criteria for functional constipation fulfilled
- Must have at least two of the following:
  - **a.** Evidence of impaired evacuation based on balloon expulsion test or imaging
  - **b.** Inappropriate contraction of pelvic floor muscles or < 20% relaxation of basal resting sphincter pressure by manometry, imaging, or electromyography
  - **c.** Inadequate propulsive forces assessed by manometry or imaging

Dyssynergic Defecation

Inappropriate contraction of the pelvic floor or less than 20% relaxation of basal resting sphincter pressure with adequate propulsive forces during attempted defecation

Inadequate Defecatory Propulsion

Inadequate propulsive forces or less than 20% relaxation of the anal sphincter during attempted defecation.

Adapted from Bharucha et al53