

15.2 Symptoms of gastrointestinal disease 2727

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ESSENTIALS The skilful analysis of symptoms indicating disorders of the digestive system is an integral part of the practice of internal medicine. Many patients with abdominal symptoms do not have easily defined organic conditions. The traditional skills of taking a careful history and examining the patient thoroughly are invaluable in managing patients who have functional disorders such as 'irritable bowel', nonulcer dyspepsia, nonspecific diarrhoea, recurrent abdominal pain, and somatization disorder. The enormous advances in endoscopy, scanning, and other investigative techniques have not made clinical diagnosis less important. Most gastrointestinal disorders are minor self-limited conditions of uncertain cause or are 'functional' in nature, thereby often eluding definition even if extensive diagnostic procedures are (fruitlessly) employed. At the other extreme, the early suspicion of life-threatening disease and prompt referral of patients for investigation depends on clinical judgement. The nature of gastrointestinal symptoms

Symptoms arising from the gastrointestinal tract are common and the lay terminology used to describe them can be misleading. Characteristic patterns of symptoms can be associated with normal test results and classified as 'functional' rather than 'organic' in nature. Functional gastrointestinal disorders comprise more than 60% of a gastroenterologist's outpatient workload, and it is best to consider such conditions as 'disorders of brain and gut interaction' rather than originating solely in one or other of these organs. There is a risk that, with increasing reliance on diagnostic tests, the lack of positive findings can lead to a breakdown in trust and the relationship between patient and clinician. Patients' lives may be blighted by symptoms that affect their ability to socialize, sleep, leave the house, or work, and the lack of positive test results and a label (for instance) of 'irritable bowel syndrome' may lead to pejorative disbelief from relatives, friends, and

employers that confounds the situation further. It is essential that the clinician is able to recognize such symptoms and provide a diagnosis without embarking on a lengthy diagnostic trail littered with false-positive findings and negative consequences, and compassionately manage the patient's symptoms, their understanding of the condition, and their expectations. Gastrointestinal symptoms are amplified by emotional echoes linked to the primacy of food and feeding in our evolutionary past. Eating or drinking together, bringing and sharing food, and 'going out' for a meal are representative of the way in which food has long been the glue of society. Psychosocial consequences of gastrointestinal symptoms can become symptoms in their own right: the social embarrassment of refusing food that has been offered; of noises and smells generated by the intestinal tract; of vomiting at the table; of urgency to defecate after mealtimes; of faecal incontinence; of having to plan your day by the presence of toilet facilities; of having a stoma that empties unpredictably—to name just a few. Clinicians need to be aware that such symptoms may be so embarrassing that patients may be reluctant to even mention them in consultation.

Upper gastrointestinal tract symptoms

Orofacial symptoms from a gastrointestinal perspective

The gastroenterologist is rarely consulted for isolated oral symptoms. Minor aphthous ulceration is common and rarely heralds an underlying gastrointestinal disorder although it can be associated with underlying nutrient deficiency, which may relate to malabsorption. Such deficiencies may result also in fissuring and erythema of the tongue and inflammation of the corners of the mouth (angular cheilitis). The latter symptom is more commonly found in the elderly due to superficial infection of an intertriginous skin fold. Deeper fissuring ulcers of the gingival mucosa and tongue may be associated with inflammatory conditions such as Behçet's disease or Crohn's disease—in the latter case, lip swelling can be disfiguring, and can be indistinguishable from that seen with orofacial granulomatosis. Referred pain in the jaw is experienced as an unpleasant ache, for instance, as a result of myocardial ischaemia or oesophageal reflux or spasm. In the latter case, it may be associated with pain referred to the tip of the tongue and salivation. Gastro-oesophageal reflux can also lead to burning discomfort in the mouth, predominantly

15.2 Symptoms of gastrointestinal disease

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SECTION 15 Gastroenterologica I disorder 2728 in the oropharynx, a 'metallic' or 'acid' taste, and changes in the quality of the voice. It is important to distinguish the poorly understood 'burning mouth' syndrome in which the patient describes very unpleasant sensory symptoms of the tongue or entire oral cavity with no visible diagnostic features and in the absence of significant gastro-oesophageal reflux. This entity often responds to drugs used for treating neuropathic pain.

Dysphagia and odynophagia

Difficulty in swallowing (dysphagia) is considered an 'alarm' symptom and to have a good predictive value for oesophagogastric cancer. However, the overall rate of cancer diagnosis in patients referred with dysphagia is around 3 to 5%. Patients are often able to point to the level on the sternum at which they feel the food is sticking. This most accurately pinpoints the site of pathology when a lower level is identified—with higher symptoms the cause could be at that site, or be the result of proximal pooling of food and fluid from a more distal obstruction. Careful questioning can discriminate different patterns of problems with swallowing in those with high dysphagia (Table 15.2.1). High dysphagia needs to be carefully distinguished from the common feeling of a 'lump in the throat'—an awareness at the back of the throat, around the level of cricopharyngeus, that can be constantly present and lead to a perception of difficulty with swallowing. This symptom, long known as 'globus hystericus', is associated with stress and anxiety and is a classic example of a functional gastrointestinal symptom accentuated by fear of underlying disease. Simple reassurance may suffice but the symptom usually evaporates after a

negative nasendoscopic examination. The pooling of nasal secretions in the pharynx due to a postnasal drip can lead to a similar sensation. Low dysphagia, at the sternoxiphisternal level, is most frequently the result of reflux oesophagitis even in the absence of endoscopically noted stricture and can settle quickly with acid suppression using a proton pump inhibitor. Acid reflux can also lead to the appearance of a fibrostenotic 'Schatzki' ring which may result in dysphagia out of proportion to the narrowing of the lumen. Conversely, fibrotic, benign peptic strictures can produce surprisingly little dysphagia despite considerable constriction of the lumen. Similarly, oesophageal tumours may present with dysphagia at a remarkably late phase of encroachment. Obstructing lesions of the oesophagus will usually lead to better tolerance of liquid ingesta than solids. Achalasia of the cardia leading to impaired relaxation of the distal oesophagus results in prolonged dysphagia usually (but not always) without pain and predominantly for solids in the earlier stages. The gradual progression of dysphagia in achalasia can lead to significant weight loss, arousing suspicion of malignancy. In only a small number of instances is this the case, due to infiltration of the oesophageal wall with metastatic cells causing 'pseudoachalasia' or secondary to paraneoplastic antineuronal antibodies. Other manometric patterns of oesophageal dysmotility—such as hypertensive oesophagus, diffuse oesophageal spasm, and nonspecific motor disorders—tend to present with dysphagia (in about two-thirds of cases) associated with severe retrosternal pain that can be confused with myocardial ischaemia and refer in a similar pattern to the jaw, shoulder, or arm. These symptoms may last for up to an hour and respond similarly to sublingual nitrates. In addition, they tend to be intermittent and may occur weeks apart without any obvious trigger. Food bolus obstruction ('steakhouse syndrome') is the sudden, unexpected complete obstruction of the oesophagus while eating and presents a quite different pattern from progressive dysphagia—it is indeed rarely seen with conditions such as carcinoma or achalasia and patients may describe no prior symptoms of dysphagia. Patients may experience extreme distress and may be unable to swallow their own saliva, and if left obstructed in this fashion are at risk of oesophageal perforation and pulmonary aspiration of pooled ingesta and secretions. The culprit lesion may even be difficult to detect on endoscopy although about 50% of cases are associated with a Schatzki ring or peptic stricture. Poorly masticated fatty meat such as beef or pork is the usual cause but it is important to exclude inadvertent foreign object ingestion as well. The other common cause of food bolus impaction, especially in young adults, is eosinophilic oesophagitis. In this condition, most likely a manifestation of hypersensitivity to unknown food antigens, patients may experience unpredictable and intermittent symptoms of dysphagia. Patients may describe food sticking at different levels in the oesophagus, including the mid sternum, rather than the cricopharyngeal or lower sternal edge as for most other causes. Dysphagia needs to be discriminated from pain on swallowing (odynophagia). In the oropharynx, odynophagia may be related to

Table 15.2.1 High dysphagia Symptom Associations
Sensation of awareness at the back of the throat worse with swallowing liquids or solids and not obstructing the passage of food
Common. 'Globus' symptom may be associated with anxiety and stress
Swallowing difficulty associated with pain
Local infective or inflammatory causes such as tonsillitis, pharyngitis, oropharyngeal candidiasis, mucosal ulceration, or tumour
Difficulty forming a food bolus and moving it around the mouth
Dry mouth—anxiety, medications (i.e. antihistamines, tricyclic antidepressants, diuretics, antihypertensives), Sjögren's syndrome, IgG4 disease
Neurological impairment—i.e. bulbar/pseudobulbar palsy, Parkinson's disease, motor neuron disease, multiple sclerosis
Advanced dementia
Choking, coughing, and/or nasal regurgitation, worse with fluids than solids while swallowing
Neurological dysphagia—as above including small vessel ischaemia, large territory cortical infarcts, and brainstem damage
Choking, coughing after swallowing with inability

to clear bolus Local high obstructive causes including pharyngeal pouch, cricopharyngeal spasm, postcricoid web, cervical spine osteophytes, tumour, dysphagia lusorum (aberrant aortic origin of right subclavian artery)

15.2 Symptoms of gastrointestinal disease 2729 local causes such as oral candidal infection, tonsillitis, viral pharyngitis, or ulceration. Painful sensations associated with the passage of food in the lower oesophagus experienced behind the sternum should be distinguished from a heightened awareness of the passage of food and fluids that is a common manifestation of visceral hypersensitivity, especially with hot or cold fluids. Odynophagia in this location is most commonly related to reflux oesophagitis, but may occur along with dysphagia in mechanical obstruction due to oesophageal dysmotility or strictures. Isolated odynophagia is rarely reported in malignancy without associated dysphagia, possibly as a result of patients changing the consistency of their dietary intake to avoid obstruction. Heartburn A distinctive and familiar symptom, heartburn can be difficult for patients to describe. However, the 'burning' nature of the discomfort and the central location with upwards radiation are characteristic of a symptom that is almost always associated with gastro-oesophageal reflux. With such a common symptom it is important to be aware of variants, which include radiation across the chest or into the jaw and tongue, left scapula, or centre of the back. Patients may describe a bad taste in their mouth or the presence of clear fluid. Heartburn and reflux may be associated with eating, or just experienced at night or on bending or lying down. It may be relieved by drinking water, milk, or antacids but not invariably so. It is important for patients and clinicians not to confuse heartburn with myocardial ischaemia. It is also important for clinicians to realize that acid reflux does not always cause heartburn. It is frequently asymptomatic and in over half of those experiencing symptoms, the only feature is a localizing pain high in the epigastrium behind the xiphisternum. 'Dyspepsia' and 'indigestion' These terms are synonymous and relate to a lay perception of upper abdominal symptoms being related to the inability to digest food. 'Indigestion' is used to describe a variety of different postprandial symptoms that have little to do with digestion but may include postprandial fullness, heartburn, dysphagia, upper abdominal pain, bloating, distension, belching, and nausea. 'Dyspepsia' has been used in criteria to try to define a stomach origin of upper gastrointestinal symptoms as differentiated from 'heartburn' arising from the oesophagus. While dyspepsia can be due to significant underlying pathology such as gastroduodenal ulceration or malignancy, the majority of patients with 'dyspepsia' have normal gastroscopic appearances such that endoscopy is not recommended in the United Kingdom for those under 55 years without the 'alarm' features of weight loss, persistent vomiting, or anaemia. Minor endoscopic appearances of erythema or gastritis are rarely responsible for 'dyspeptic' symptoms. However, when *Helicobacter pylori* is present, its eradication leads to resolution of symptoms in up to 40% of cases of dyspepsia without ulceration. Hiccups Hiccups are caused by a sudden reflex contraction of the diaphragm, usually unilateral, with the characteristic sound produced by glottic closure against the inflow of air. Normally self-limiting and with no known purpose, hiccups can be caused by gastric distension, gastro-oesophageal reflux, pericarditis, subphrenic collections, intrathoracic tumours, or metabolic derangements (uraemia, alcohol excess). A significant underlying cause is more likely if present for more than 48 h. Stimulation of the nasopharynx with iced water or lemon juice, or altering respiratory function by breath holding or the Valsalva technique will resolve the symptom in the majority of cases. Belching Eructation, persistent burping or belching, can be associated with anxiety and is a common accompaniment of gastro-oesophageal reflux. The excess gas is usually due to air swallowed by an increased frequency of dry swallowing as a learnt response to reflux

episodes. Acid suppression with proton pump inhibitors significantly reduces the symptom (and swallowing frequency) when associated with acid reflux, but it may require breathing exercises or psychological approaches in other cases. Halitosis or foul smells associated with burping may be due to gastric stasis or food fermenting in a gastric or oesophageal diverticulum. Nausea is perhaps the most diffuse and difficult gastrointestinal symptom to evaluate. Derived from a Greek word for 'ship', it is most easily associated with motion (or sea-) sickness and is induced centrally by a wide variety of different triggers with vomiting as the linked effector component. It probably evolved as a reflex means of voiding potentially toxic food substances. The afferent limb arises from chemoreceptors in the stomach and upper intestine as well as visceral stretch receptors predominantly in the stomach, and chemoreceptors outside the blood-brain barrier in the medulla oblongata. Nausea has a strong learned component in order to lead to conditioned food aversion and prevent repeated ingestion of toxins. This memory for chemical stimuli leads to subjective nausea with sensory triggers such as sight, sound, or smell. An evolutionary advantage for motion sickness is unclear unless to expel neurotoxins leading to disequilibrium, such as natural alcohols. Nausea arising from stomach mechanoreceptor activation can be relieved by emptying the stomach and explains the relief that can arise with vomiting, but also the significant benefit achieved in nausea associated with (e.g. malignant bowel obstruction) using a Ryle's tube or venting gastrostomy. Vomiting is the forceful ejection of gastric contents by sudden contraction of the abdominal and gastric musculature with relaxation of the gastro-oesophageal junction, usually preceded by nausea. Different patterns of vomiting can be discerned relating to the underlying pathology—patients with self-limiting acute, repetitive vomiting usually have a toxic or viral gastroenteritic cause. Patients with mechanical obstruction of the pylorus or proximal duodenum due to tumour, scarring, or oedema associated with peptic ulceration, or 'groove' pancreatitis, may eat normally and then experience large-volume vomits without warning including evidence of meals eaten much earlier. Bile-stained or feculent vomits may provide a clue to the level of obstruction. Some patients vomit after eating or drinking very small quantities such that the gastric sensation or chemoreception may be sufficient to trigger a learnt vomiting reflex. In around 40% of cases, this reflex can be suppressed using psychological approaches. This symptom is prominent in patients with gastric dysmotility, often young females.

SECTION 15 Gastroenterologica I disorder 2730 Frequently the patient describes vomiting everything they eat or drink shortly afterwards, yet maintain their weight and hydration, presumably only vomiting a small proportion of their intake. This pattern of vomiting may correspond to a reflex contraction of abdominal wall muscles known as 'rumination' syndrome. There is a strong psychogenic component to nausea and vomiting, including a response to altered eating behaviour such as anorexia or bulimia nervosa where patients may self-induce vomiting by pharyngeal stimulation prior to learning to vomit spontaneously. A condition previously only recognized in children of 'cyclical vomiting syndrome' is poorly understood but results in repeated stereotypical episodes of vomiting with a prodrome reminiscent of migraine. Early satiety and postprandial fullness Patients with gastric outlet obstruction or gastroparesis may complain of a feeling of fullness when eating and may only be able to manage small meals. However, gastric emptying can be normal or fast with symptoms of early satiety which may arise from impaired fundic relaxation after eating—a 'functional' dyspepsia. Rapid gastric emptying, due to neuromuscular discoordination or surgically altered gastroduodenal anatomy, can lead to postprandial fullness and bloating, associated with sweating or fainting. This 'dumping' syndrome is due to both delivery of a high osmolar load into the intestine and a lack of integrated neurohormonal

feedback. The latter can also result in a delayed postprandial hypoglycaemia or 'late dumping'. Avoiding taking liquid and solids within half an hour of each other and regular eating of small quantities ('grazing') can help to manage dumping symptoms.

Abdominal pain The acute abdomen Sudden onset of pain, often well localized and associated with features of peritonism, such as exacerbation with breathing or movement, guarding, and rebound or percussion tenderness, signifies an acute abdomen and warrants urgent cross-sectional imaging and surgical review (see Chapter 15.4.1 for further discussion). Metabolic disorders that can masquerade as a surgical acute abdomen include diabetic ketoacidosis and lactic acidosis. The latter should raise concern about the possibility of underlying visceral ischaemia regardless of risk factors for arterial vascular disease as it can occur at any age as a result of prothrombotic disorders (arterial or venous thromboses) or congenital malrotation of the gut. First presentation of a familial periodic fever or acute porphyria may rarely present with an acute abdomen. Approach to chronic or recurrent abdominal pain

Chronic abdominal pain (Table 15.2.2, Fig. 15.2.1) can present a significant challenge. Often the duration of the symptoms indicate a benign condition and there may be no obvious abnormality on initial investigations, leading to a functional diagnosis. Chronic abdominal pain due to 'visceral hypersensitivity' may be initiated by an acute initial pathology which it might mimic in nature and location. Such pain can usually be identified as functional if the underlying cause is removed—after appendicectomy or cholecystectomy, for instance. An understanding of such secondary functional visceral hypersensitivity aids the clinician greatly

Table 15.2.2 Considerations in diagnosis of chronic abdominal pain

Feature	Considerations
Duration	<ul style="list-style-type: none"> • Long-standing symptoms without progression are unlikely to signify serious underlying pathology • It may be possible to identify a change in diet or lifestyle at the time the pain started
Temporal pattern	<ul style="list-style-type: none"> • Frequent intermittent pain occurring only some days a week with long periods of respite is common with functional disorders • Rare, infrequent episodes may be due to functional disorders but consideration of other pathologies such as biliary colic, recurrent acute pancreatitis, intermittent volvulus, or acute diverticulitis should be considered • Spasmodic pain in the early morning may arise from the colon due to the diurnal pattern of migrating motor complexes • Pain associated with fermentative gas bloating may get worse during the day and ease overnight
Functional colonic pain may be affected by the menstrual cycle	<ul style="list-style-type: none"> • Pain that appears in different places around the abdomen is most likely to be of functional colonic origin rather than a fixed pain of localized pathology • Pain localized (with one finger) to a specific point is more likely of abdominal wall (musculoskeletal) origin than visceral
See Fig. 15.2.1 for chronic pain localizations	<ul style="list-style-type: none"> • Spasmodic (cramping) pain may be described as 'twisting' or 'vice like' and usually signifies hollow organ origin • Constant deep aching pain should lead to exclusion of localized inflammation—for instance, associated with diverticulitis or ileocaecal Crohn's disease but may still be functional in origin • 'Knife like' through to back is a common description of pancreatic pain (rather than radiating around to the back)
Exacerbating and relieving factors	<ul style="list-style-type: none"> • Position—pain triggered by movement is most likely of musculoskeletal origin but changes in position can exacerbate or relieve functional pains, particularly associated with bloating (right iliac fossa pain due to bloating is often worse when sitting or driving). Movement of the right hip can be painful with transmural ileocaecal inflammation associated with Crohn's disease. Pain due to gastro-oesophageal reflux can be worse when lying on one side (usually the left) • Eating—exacerbation of a severe upper abdominal pain with eating may indicate gastroduodenal ulceration or a pancreaticobiliary origin. However, pain arising from anywhere in the gastrointestinal tract can be triggered by eating due to the stimulation of motility • Defecation—pain eased or exacerbated by opening the bowels usually implicates a colonic origin

Breathing—may indicate a musculoskeletal origin, but can exacerbate capsular pain from the liver or spleen

Associated symptoms

- Bloating and loud borborygmi associated with chronic pain may indicate a stricture, but more commonly represent colonic fermentation of dietary substrate or as a result of malabsorption (i.e. bacterial overgrowth, coeliac disease)
- Nausea may be a secondary response to pain, but vomiting could implicate a gastroduodenal or pancreaticobiliary origin
- Altered bowel function at the time of the pain or an alternating bowel habit would usually indicate a functional colonic origin

15.2 Symptoms of gastrointestinal disease 2731 in the management of conditions such as chronic pancreatitis where ongoing pain may not arise from the pancreas itself and may be exacerbated by long-term use of opiate analgesia.

Altered bowel function

Normal bowel function varies significantly and although the bowel operates to a diurnal motility rhythm, a daily bowel motion is passed by only around a third of the population (Fig. 15.2.2). It is essential to clarify the patient's description of their bowel habit in terms of frequency and consistency—some patients describe faecal incontinence or the passage of mucus as 'diarrhoea' and 'constipation' may be used variably to describe the passage of hard stools, infrequent passage of stools (of any consistency), or having to strain to pass a motion regardless of frequency or form. The Bristol stool chart can be helpful for patients to describe the form of their stool (Fig. 15.2.3). A persistent change in bowel habit (>6 weeks) in patients over 60 years is considered an indication for urgent referral in the United Kingdom to exclude colorectal cancer.

Diarrhoea

Diarrhoea is best considered as the more frequent passage of looser motions than usual, rather than an objective definition of more than 200 g of stool per day. Paradoxical 'overflow' diarrhoea due to faecal impaction should be excluded in very elderly or infirm patients by rectal examination. A variety of medications—including nonsteroidal anti-inflammatory drugs, selective serotonin reuptake inhibitors, proton pump inhibitors, and metformin—can cause diarrhoea. The duration of a diarrhoeal episode and the appearance of the stools often help to define the cause. Acute self-limiting diarrhoea is usually of infectious origin and may be bloody (dysenteric) in the case of ischaemic colitis, cytomegaloviral, bacterial, or amoebic infection, and can be prolonged (up to 3–4 weeks). Diarrhoea due to idiopathic inflammatory bowel disease (most commonly ulcerative or Crohn's colitis) is usually frequent, bloody, and small volume and may be associated with fever, tenesmus, and systemic symptoms. Fatty diarrhoea (steatorrhoea) can be detected by the pale colour, unpleasant odour, and fatty globules and is a specific sign of fat malabsorption but is only present in about one-third of cases. Common causes include pancreatic exocrine deficiency, giardiasis, small intestinal bacterial overgrowth, and coeliac disease—the lack of obvious fatty change in the stools should not exclude these diagnoses. Chronic watery diarrhoea may be due to unabsorbed osmoles, for instance, sugar alcohols such as xylitol, or magnesium, phosphate, or sulphate ions present in laxatives.

Nonabsorbed Gastro-oesophageal reflux

Isolated epigastric or radiating upwards

Functional colonic pain (locations may change)

Chronic cholecystitis (to right scapula)

Biliary colic (to back and epigastrium)

Pancreatic pain

Pain from small intestine, i.e. mesenteric angina

Ileocaecal inflammation

Crohn's, tuberculosis

Liver capsule pain

Fig. 15.2.1 Anatomical considerations of chronic abdominal pain.

0 5 10 15 20 25 30 35 40 <1 2 3 4 5 6 7 8 10 11 12 13 14 15 16 17 18 19 20 21>21

Percentage of population

Number of bowel motions per week

Men

Women

Fig. 15.2.2 Normal bowel frequency. Data from Heaton KW, et al. (1992). Defecation frequency and timing, and stool form in the general population: a prospective study. *Gut*, 33, 818–24.

SECTION 15 Gastroenterologica I disorder 2732 carbohydrates, such as lactose or fructose, or complex polysaccharides as excess dietary fibre cause osmotic diarrhoea due to bacterial fermentation to short-chain fatty acids and an acidic stool (pH <6) often associated with bloating or passage of wind. Secretory causes can be differentiated from osmotic causes of profuse watery diarrhoea by persistence during fasting, and commonly signifies bile salt malabsorption or forms of microscopic colitis. Gastrointestinal neuroendocrine tumours (carcinoid, vasoactive intestinal peptide-producing tumour) account for less than 1% of cases, and classically gastrinoma results in steatorrhoea due to acid inactivation of pancreatic enzymes. Constipation A common problem that increases with age and immobility, constipation affects up to 70% of elderly patients in nursing or residential care homes. Careful questioning can distinguish patients who have obstructed defecation as a result of pelvic floor dysfunction from patients with slow-transit constipation. The latter is frequently related to voluntary withholding of stool, often dating from adverse experiences in childhood or adolescence, or due to painful defecation due to anal causes such as fissure. Insufficient fluid or dietary fibre intake may be causative. Many medications lead to constipation, including opioids, antidepressants, serotonin receptor blockers, diuretics, and calcium channel blockers. Significant underlying causes are uncommon but include hypothyroidism, coeliac disease, Parkinson's disease, and hypercalcaemia. Motor dysfunction of the gut due to neuromuscular disorders can result in a variety of presentations—Hirschsprung's disease can result in severe constipation in neonates and young infants but in its short segment form may not be diagnosed until adulthood with a 'mega rectum'. Chronic idiopathic intestinal pseudo-obstruction is the term given to the clinical presentation of various smooth muscle and enteric neuronal pathologies and may be heralded by profound constipation and abdominal distension. Alternating pattern Alternating diarrhoea and constipation should be clearly distinguished from either symptom in isolation and usually signifies a benign functional disturbance that frequently responds to dietary fibre supplementation. Patients may only report the symptom (either constipation or diarrhoea) that is most troublesome and it can require careful questioning to uncover the alternating pattern. 'Irritable bowel syndrome' This term implies abdominal pain or discomfort, bloating, and a change in bowel habit and has been defined by a number of different criteria (Table 15.2.3). It is often separated as diarrhoea, constipation, or alternating pattern predominant. Specified criteria should be seen as tools for research and not diagnostic tests that require strict fulfilment as a prerequisite for the diagnosis of a functional disorder. While it can be reassuring, the use of the term 'irritable bowel syndrome' can have detrimental consequences. Clinicians may consider this to be the end of their engagement with the patient (having ruled out 'serious' pathology), whereas it should instead be the start of managing and treating their symptoms. Possibly as a result of such attitudes, the term carries lay undertones of trivialization which some patients may find difficult to accept. Indeed, it may be that (in common with many gastrointestinal symptoms) the overuse of the term 'irritable bowel syndrome' has led to such misunderstanding and decrease in its utility that it should be used sparingly or even replaced by a narrative diagnosis of a 'disorder of brain-gut interaction'. Bristol Stool Chart Type 1 Separate hard lumps, like nuts (hard to pass) Sausage-shaped but lumpy Like a sausage but with cracks on the surface Like a sausage or snake, smooth and soft Soft blobs with clear-cut edges Fluffy pieces with ragged edges, a mushy stool Watery, no solid pieces. Entirely Liquid Type 2 Type 3 Type 4 Type 5 Type 6 Type 7 Fig. 15.2.3 Bristol Stool Chart. Copyright 2000 Rome Foundation, Inc. All rights reserved. Table 15.2.3 Definitions for irritable bowel syndrome 'Rome IV' criteria, 2016 Recurrent abdominal pain or discomfort experienced on average at least one day a week over the last 3 months associated with two or more of the following (with onset at least 6 months prior to

diagnosis): • Association with defecation • Onset associated with a change in bowel frequency • Onset associated with a change in stool appearance 'Manning' criteria, 1978 (the more positive criteria, the greater likelihood of irritable bowel syndrome) • Onset of pain linked to more frequent bowel movements • Looser stools associated with onset of pain • Pain relieved by passage of stool • Noticeable abdominal bloating • Sensation of incomplete evacuation >5% of the time • Diarrhoea with mucus >25% of the time

15.2 Symptoms of gastrointestinal disease 2733 Anorectal symptoms Bright red bleeding arising from the anus is a frequent accom- paniment of hard stools and may be noticed only on wiping. Such bleeding can arise from the distal rectum in proctitis, without asso- ciated diarrhoea. While patients may report profuse bleeding from haemorrhoids, this is usually more worrying in its appearance and rarely sufficient to cause significant blood loss or iron deficiency. Rectal bleeding as a result of pelvic radiotherapy can be profuse and troublesome, and associated with reduced rectal compliance leading to urgency and frequent small stools. The latter may equally accom- pany chronic rectal involvement with inflammatory bowel disease. Prolapse of haemorrhoidal tissue ('piles') can be associated with straining at stool and is usually only painful when associated with thrombosis. More significant prolapse of the anterior rectal wall can be due to pelvic floor weakness (usually in females and often relating to childbirth), and can result in traumatic 'solitary rectal ulcer syndrome' that can be confused with proctitis but shows a distinctive histology. Rectal pain on defecation is usually due to an anal fissure and can be accompanied with bleeding and lead to secondary constipation by voluntary avoidance of passing stool. Superficial perianal fistulae may be isolated but when complex or deep are usually associated with Crohn's disease and may not always be associated with symp- toms of colonic involvement. A symptom of intense, deep rectal pain—'proctalgia fugax'— is common, fleeting, and of unknown origin and rarely signifies underlying pathology. A more chronic form of similar rectal pain— 'levator ani syndrome'—can be identified by pain felt on the left side approximately 5 cm inside the rectum. FURTHER READING Abdel Jalil AA, Katzka DA, Castell DO (2015). Approach to the pa- tient with dysphagia. *Am J Med*, 128, 1138.e17-23. Bellini M, et al. (2015). Irritable bowel syndrome and chronic consti- pation: fact and fiction. *World J Gastroenterol*, 21, 11362-70. Drossman DA, Hasler WL (2016). Rome IV - Functional GI dis- orders: Disorders of Gut-Brain Interaction. *Gastroenterology*, 150(6), 1257-61. Gunnarsson J, Simren M (2008). Efficient diagnosis of suspected func- tional bowel disorders. *Nat Clin Pract Gastroenterol Hepatol*, 5, 498-507. Heaton KW, et al. (1992). Defecation frequency and timing, and stool form in the general population: a prospective study. *Gut*, 33, 818-24. Levy RL, et al. (2006). Psychosocial aspects of the functional gastro- intestinal disorders. *Gastroenterology*, 130, 1447-68. Longstreth GF, et al. (2006). Functional bowel disorders. *Gastroenterology*, 130, 1480-91. National Institute for Health and Care Excellence (2014). Gastro- oesophageal reflux disease and dyspepsia in adults: investigation and management. Clinical guideline. <http://www.nice.org.uk/guidance/cg184> National Institute for Health and Care Excellence (2017). Clinical knowledge summaries: hiccups. <http://cks.nice.org.uk/hiccups> Sweetser S (2012). Evaluating the patient with diarrhea: a case-based approach. *Mayo Clin Proc*, 87, 596-602. Talley NJ, Ford AC (2015). Functional dyspepsia. *N Engl J Med*, 373, 1853-63. Talley NJ, et al. (1990). Diagnostic value of the Manning criteria in ir- ritable bowel syndrome. *Gut*, 31, 77-81.

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